HYPERURICEMIA AS AN EARLY MARKER IN PREDICTING MORTALITY AND MORBIDITY IN PATIENTS WITH SEPSIS

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Dr. Sreekanth Chowdary…

ABSTRACT

Background and Objectives

Sepsis continues to be a medical and financial burden for both developed and developing countries, and affects all age groups. The normal value of uric acid varies with age, gender, ethnicity and physiologic status of the population. Uric acid levels in septicemic patients and their correlation with increased global prevalence along with septic shock depends on its severity. The use of uric acid levels for grading the severity and its outcome in these patients is widely advocated despite caveats to its use with septicemia and conditions like shock being cited as a major confounding factor. The value of serum uric acid may be erroneous in those with septicemia and its sequelae, thus mandating its evaluation. The objective of this study was to identify a correlation between severity of sepsis & outcome of patients and the derangement of uric acid levels in an adult population.

Materials and Methods

The present study was conducted on patients with a diagnosis of sepsis admitted in the Department of General Medicine of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre from January 2019 to December 2020. Relevant data was collected by a detailed interview with the patient, clinical examination and blood investigations. The patients were categorized into the 3 categories of severity of sepsis, as per the patient's procalcitonin values. Serum uric acid levels were measured and recorded and a correlation of severity of different types of sepsis with the outcome was studied. Statistical tests such as Chi Square test and ANOVA were used for analysis.

Results

In the 79 septicemic patients, age ranged from 18-88 years. The number of male patients was slightly more than females. The commonest symptom of patient presentation was generalized weakness, and the most common sign was pallor. Majority of our patients were found to have severe septicemia with shock. We observed higher levels of serum uric acid levels in patients with severe sepsis. There was higher mortality observed in patients with severe sepsis. There was an observed correlation between consumption of alcohol and the outcome of patient, mostly leading to mortrality.

Conclusion

Uric acid levels in patients with septicemia may be affected by variables such as age, sex, chronic illnesses or drugs. We feel it is worthwhile to study these confounding factors with large sample sizes for ascertaining a correlation of uric acid levels with sepsis and the outcome of patients considering these variables.

Keywords: SERUM URIC ACID, SEPTICEMIA.

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INTRODUCTION

Sepsis, a major medical condition within the body associated with inflammatory state (called SIRS) along with the presence or absence of a known infection having a serious consequence to the body. During the course of hospitalization of the patient, most of the them undergo reperfusion. Injury and severe inflammatory response to the ischemic injury.

In the recent past about in the last two decades it has been proven that occurrence of MODS is a possibility without actual SIRS in the patients. This is a possibility because there are both pro and anti-inflammatory cytokines present in the body at at the same time. According to, "The Sepsis Definitions Task Force in 2016 proposed the Third International Consensus Definitions specifying that sepsis is a dysregulated host response to infection that leads to acute organ dysfunction and Septic shock is defined as a complication of sepsis resulting in derangements in circulatory and metabolic pathways in the body".

The nitrogenous compounds purines, have two sources endogenous (within the body) exogenous (via food)

The final end product of purine metabolism in animals is allantoin whereas in human beings it is uric acid, which passes through the liver, reaches the blood stream and most of the uric acid reaching the blood stream is finally excreted via urine [1,2]. A small quantity of uric acid in the body is degraded after reaction with oxidants like free radicals [1]. Over the past decade or so, there have been major studies suggesting a strong association between atherosclerosis [3-7], systemic hypertension, hyperinsulinemia [8,9] leading to diabetes mellitus and chronic kidney disease [10] and levels of serum uric acid. In patients with sepsis associated with MODS due to presence of high levels of oxygen free radicals, elevated levels of serum uric acid

levels are observed indicating raised oxidative stress levels in the body. In patients with acute severe infections and/or chronic inflammation, the levels of serum uric acid is elevated leading to activation of transcriptional factors. These factors indicate a poor prognosis in the outcome of the patient.

This study was conducted at Dr Prabhakar Kore Hospital and MRC, Belagavi, a tertiary care center, to bring out the correlation between hyperuricemia in patients diagnosed with sepsis with morbidity & mortality. This study is also aimed at finding a correlation between hyperuricemia in sepsis patients and acute kidney injury, ARDS & duration of stay in the hospital. Serum uric acid levels of >7 mg/dl was defined as hyperuricemia (in males and females) [13]. AKI was defined as, "An absolute increase >0.3 mg/dl increase in creatinine above the baseline"

OBJECTIVE

The objective of the present study was

- To study hyperuricemia as an early marker in predicting mortality and morbidity in patients with sepsis.

REVIEW OF LITERATURE

The word sepsis has its origin from a Greek word “sepo”, which means “putrefaction” and the word Septic shock from the French word “choquer” which means “to collide with”.[14] Millions of people, every year get affected by sepsis throughout the world having high chances of mortality.

Definition of systemic inflammatory response syndrome (SIRS)	
1.	temperature> 38 °C or, < 36 °C
2.	heart rate > 90 beats/min
3.	respiratory rate >20/min or (PaCO2) < 32 mmHg
4.	white blood cell count >12.0 ×109/L, < 4:0 × 109/L, or > 10% immature (band) forms
*2 or more of the following conditions	

AS PER THE NEW GUIDELINES	
1.	sepsis = infection + systemic manifestations of infection.
2.	Severe sepsis = sepsis + sepsis-induced organ dysfunction or tissue hypoperfusion.
3.	Septic shock = sepsis-induced hypotension persisting despite adequate fluid resuscitation.

HISTORICAL REVIEW:

1.	The first mentions of sepsis is an ancient literature work by Hippocrates, a philosopher and a physician during 400 BCE. He believed the decaying process of the body releases many substances that can cause "auto-intoxication". He also observed a few antiseptic properties due to presence of ethanol tried to discover pharmacological response based on these this finding.
2.	Galen (129-199 AD), a Roman physician and philosopher, worked on sepsis and proposed theories about pus and wound healing. His theories had lasted for over 1500 years. Romans believed that sepsis was caused by some invisible creatures that secreted fumes and laid the foundation for the Roman public health system. They emphasized on the hygiene practices.
3.	In early 1800's Joseph Lister, Louis Pasteur and Robert Koch were the pioneers in the field of microbiology and infectious disease.
4.	Pasteur's experiments disproved the theory of spontaneous development of diseases.
5.	Lister also formulated his theories on wound sepsis. Lister postulated that wound sepsis occurs by entry of pathogens through breaks in the skin and hence he developed carbolic acid dressings. This led to a tremendous decrease in wound sepsis and sepsis related deaths.
6.	Dr. Edward Frank (In 1964) a Boston surgeon, framed a management strategy for septic shock. The strategy included continuous monitoring of cardiac output, urine output, systemic pressure, blood volume, blood chemistries, pH, electrolytes and central venous pressure.
7.	In 2003, an international committee published latest guidelines for severe sepsis and septic shock

AETIOLOGY:

Majority of the septicemia cases can be classified as

1) Community acquired
2) Hospital acquired

Pneumonia numbers to half of all cases of septicemia, followed by intra-abdominal and GU infections [15]. Only 1/3 rd of patients have positive blood culture reports while remaining 2/3 rd are culture negative.

Most commonly isolated gram-positive bacteria are:

1) Staphylococcus aureus.
2) Streptococcus pneumoniae.

Most commonly isolated gram-negative bacteria are:

1) Escherichia coli.
2) Klebsiella species.
3) Pseudomonas aeruginosa.

The risk/predisposing factors for sepsis are:

1) chronic diseases like HIV infection.
2) chronic obstructive pulmonary disease.
3) cancers and immunosuppression.

Risk factors of multiorgan dysfunction are not well studied, but may include:

1) Underlying health status of the individual patient.
2) Baseline organ function before the onset of infection.
3) Start of treatment from the time of diagnosis.
4) Age, sex, and ethnicity can influence the incidence of sepsis.
5) Genetic factors.

GENERAL FACT FIND
In India, large studies not sufficient enough to get an overview of the incidence and prevalence of sepsis. In a recent study involving 5 years duration in a hospital, it was found that the mortality among sepsis patients was 63.6% out of which 56% died in the ICU [15].

PATHOGENESIS:

The older understanding of sepsis was considered as a result of overactivation of inflammatory response in the patient leading to SIRS [16]. The newer studies have shown that infection actvates a cascade of events which are complex and may be responsible for pathogenesis of sepsis. The host response to sepsis is an individual response and is dependent on various factors like genetic predisposition and associated comorbidities. The response also varies at the local and the systemic levels. The systemic effects of sepsis is an effect of the pro-inflammatory factors leading to tissue damage. The anti-inflammatory responses in the body protects from the tissue damage. As the course of the disease progresses there is an increased chances of acquiring secondary infections. The overall capacity of the host to resist these pro inflammatory reactions would decide the clinical outcome of the patients i.e, whether the patient survives or succumbs to the disease process.

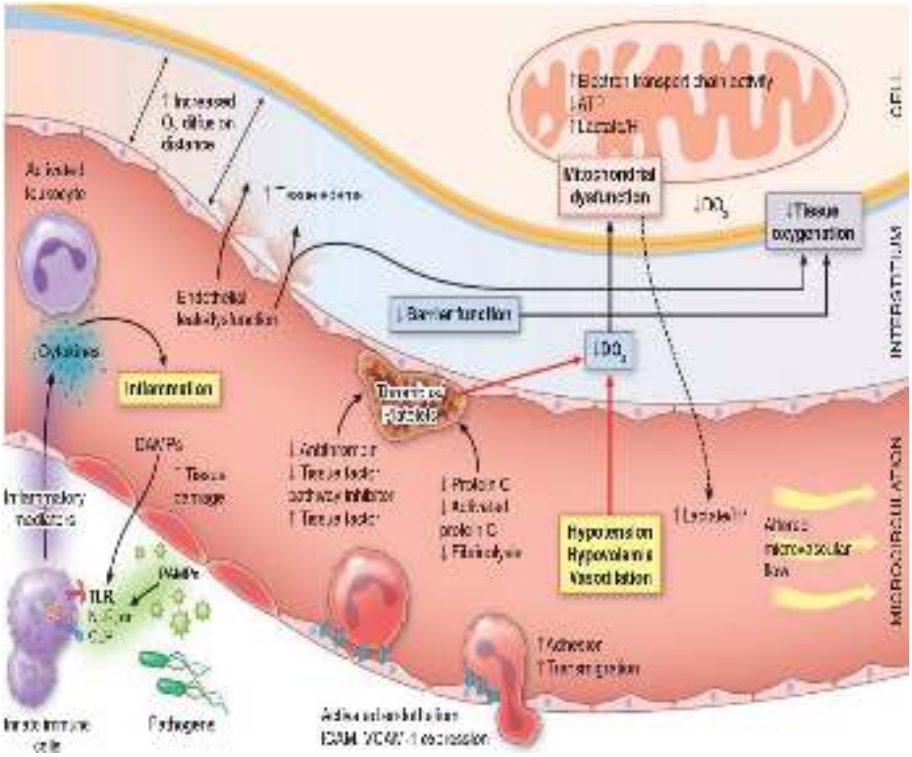


Figure-1 showing the pathogenesis of sepsis

INFLAMMATORY RESPONSE

The 4 main classes of pattern recognition receptors (PRR) for activating immune cells are:	
1.	TLRs
2.	RIG-I-like receptors
3.	C-type lectin receptors
4.	NOD-like receptors. The activity of the NOD like receptors occurs partially in protein complexes called *inflammasomes*.

The upregulation of expression of the pro-inflammatory genes and their translation into the pro inflammatory protiens are responsible for recognizing the microbial specific molecular patterns called as 'pathogen associated molecular patterns (PAMPS)'. These PAMPS contain a lipid A moiety of lipopolysaccharide (LPS & endotoxin). These moieties get attached to the LPS binding protein which is usually seen on the surfaces of monocytes, macrophages and polymorphonuclear cells a.k.a. neutrophils. The signals which are generated are then transmitted to the TLR4 receptor for production and release of cytokines like TNF alpha whose main function is to alert the adjoining normal cells and tissues. There are about 10 types of TLR's which are identified in the humans.

An additional feature of these receptors is the ability to sense the endogenous molecules that are released by the injured cells – Damage associated molecular patterns (DAMPs). These DAMPs recognize high-mobility protein B1, S100 protiens, extracellular RNA and DNA, and histone molecules. Newer studies have shown that production of DAMPs is associated with the pathogenesis of MODS which is similar in both sepsis and non-infectious critically ill patients. These inflammatory processes

are also responsible for activation of the complement system, PAF, arachidonic acid and its metabolites like prostaglandins and nitric oxide. The production of nitric acid is associated with vasodilatation and may lead to MODS eventually.

COAGULATION DISORDERS:

The most common coagulation abnormality in patients with sepsis is disseminated intravascular coagulation. These abnormal coagulation processes were initially thought to be the effect of the invading micro-organisms. Fibrin, a transmembrane glycoprotein that is produced by many cells in our body when produced in excess is responsible for DIC. These associated with impaired anticoagulant mechanisms like protein C & S, antithrombin and down regulation of the fibrinolytic systems also play a vital role in the pathogenesis of DIC. A few micro-organisms like the meningococcus has a lethal and unique mechanism of inducing DIC. It produces inflammation associated proteases, which leads to the activation of endothelial cells and its damage. This mechanism of meningococcal makes it more lethal.

ORGAN FAILURE:

Tissue hypoperfusion with reduction in the availability of oxygen leads to cellular dysfunction and tissue damage. The most common cause of reduction in oxygenation the tissues is hypotension (hypotensive shock). This reduction in the effective blood volume may lead to the reduction in red cell membrane stability, eventually leading to microvascular thrombosis. The ongoing inflammation may lead to vascular endothelial dysfunction and vascular leak. Cellular death and loss of cellular barrier integrity leads to anasarca (generalized edema of the body). The cascade of inflammatory processes are involved in the overproduction of nitric oxide resulting in vasomotor collapse. The opening of AV fistulas may lead to shunting of

oxygenated blood into the venous pool. These results in reduced oxygenation of the more susceptible tissues. Production of free oxygen radicals may interfere with the ability of mitochondria to produce ATP via oxidative phosphorylation and oxygen delivery to the cells. ATP is needed for the tissued to perform their basal metabolic functions and vital cellular functions. These basic energy requirements are usually matched via glycolysis and fermentation. The basal production of ATPs when falls below the critical values required for cell maintenance, “bioenergentic failure” occurs. This leads to production of oxygen free radicals and the apoptotic pathway is activated leading to cellular death.

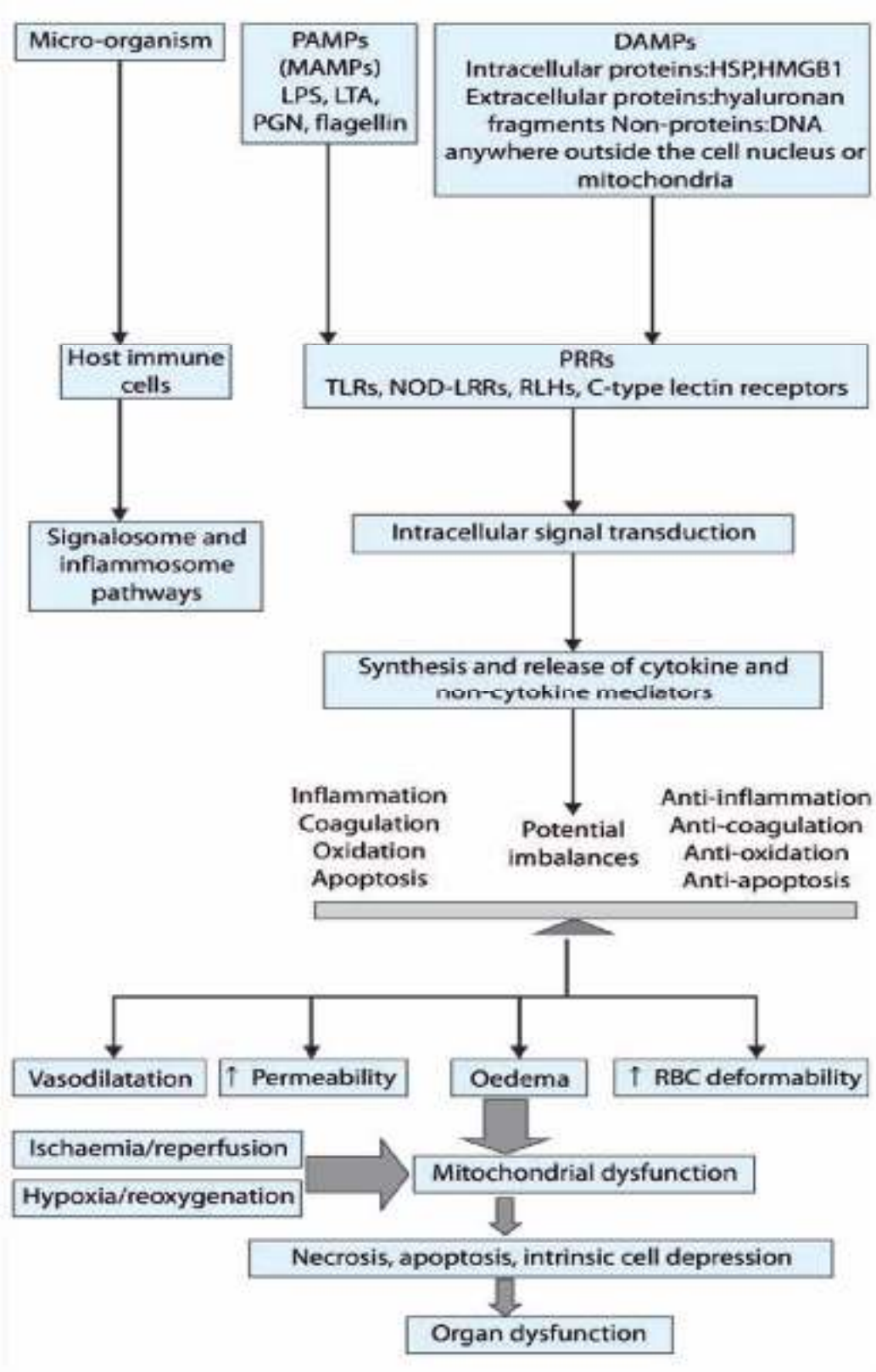


Figure-2 An overview of the pathogenesis of sepsis

ANTI-INFLAMMATORY MECHANISMS:

The main mechanisms by which the immune system can manifests as harmful effects are mainly by:

1) Humoral immune response
2) Cell mediated immune response
3) Neural mechanism

These mechanisms are well known to damage the tissues.

The host phagocytes can promote anti-inflammatory response and with the help of regulatory T lymphocytes and myeloid-derived suppressor T cells can further reduce the inflammatory process.

The neuroinflammatory reflex:

Vagus nerve acts as afferent system takes the impulse to the brain stem → efferent via vagus nerve activates splenic nerve (of celiac plexus) → norepinephrine released in the spleen and acetylcholine is released from a subset of CD4+ T cells. This released acetylcholine targets alpha 7 cholinergic receptors on macrophages, thus reducing cytokine release which reduces the proinflammatory activity.

Few experimental studies have shown that vagotomy in animals make them more susceptible to endotoxin shock and by stimulating the efferent vagus nerve or the alpha 7 cholinergic receptors, systemic inflammation is attenuated.

IMMUNE SUPPRESSION – ITS ROLE:

In the patients who are dependent on intensive care units after surviving the early sepsis have evidence of suppressed immune system. This may manifest due to inadequate anti microbial therapy and activation of latent viruses. There is documented evidence of reduced response of the WBC's to these pathogens. These findings have been confirmed via post-mortem findings of the patients who died in

ICU which reveal reduced splenocyte function. The two main organs which are immune suppressed are spleen and lungs where there is increased expression of T cell inhibitory receptors on parenchymal cells. Increased apoptotic cell death of CD4+ cells, dendritic cells and B cells is associated with immune suppression due to sepsis. Similarly immune system dysfunction is leading to multi system failure.

CLINICAL MANIFESTATIONS:

In response to severe infection, the patient can clinically manifest as coagulation and neuroendocrine symptoms which are due to complex mechanisms. [15]. In response to severe sepsis, if the patient develops hypothermia, tachycardia and tachypnoea it is termed as septic shock. This leads to decreased organ perfusion and hypotension. The main predisposing factors for sepsis are diseases like diabetes mellitus, liver cirrhosis and CKD. Diseases like cellulitis, pustules, bullae or hemorrhagic lesions occur due to spread through the underlying soft tissue infections mainly via blood. Additional manifestations like reduced organ perfusion, encephalopathy, jaundice, GI bleeding, DIC and ARDS can be seen in patients with septic shock.

FACTORS INDICATING POOR PROGNOSIS IN SEPTIC SHOCK:

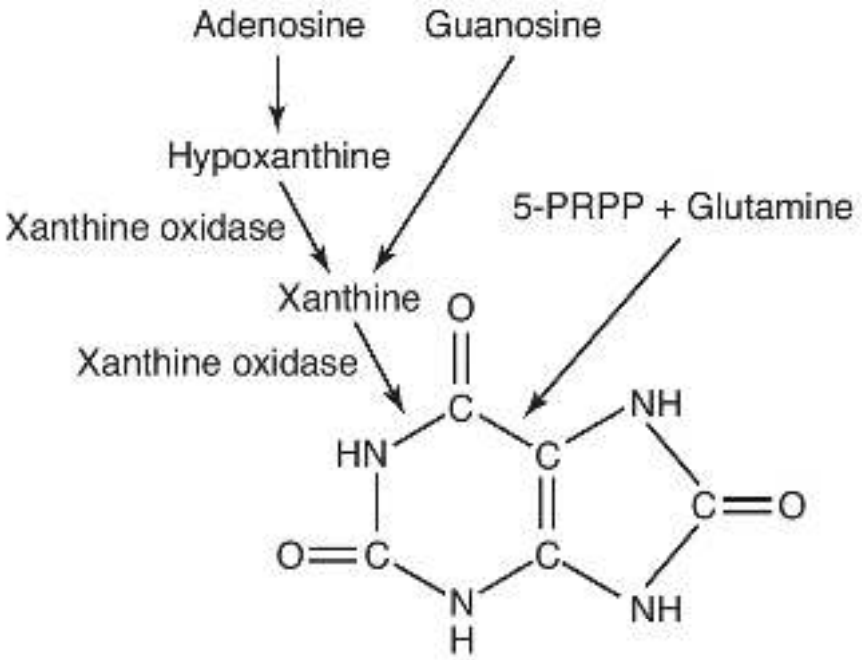
PRE-EXISTING FACTORS
Old age
Nature of co-existing illness
Number of failed organs
CARDIOVASCULAR
Lack of ventricular dilatation
Persistence of tachycardia and raised cardiac output
OTHERS
Delay in institution of treatment
Acute respiratory distress syndrome
Disseminated intravascular coagulation
Hypothermia
Leucopenia
Hyperglycemia
Hyperuricemia
Metabolic acidosis
Renal failure
Polymicrobial infection

DIAGNOSTIC CRITERIA FOR SEPSIS:		
A. GENERAL VARIABLES		
Fever (core temperature >38.3^0 Celsius)	Tachypnoea	Hyperglycemia (plasma glucose >120 mg/dl) in the absence of diabetes
Hypothermia (core temperature <36^0 Celsius)	Altered mental status	
Tachycardia	Significant oedema	
INFLAMMATORY VARIABLES		
Leucocytosis (>12000/ml)	Plasma C- reactive protein > 2 SD above the normal value	Plasma procalcitonin>2 SD above the normal value
Leucopenia (<4000/ml)	Normal WBC count with >10% immature forms	
HEMODYNAMIC VARIABLES		
Arterial hypotension (SBP <90 mmHg, MAP<70, or SBP decrease >40 mmHg)	Cardiac index > 3.5 L/min/m2	
ORGAN DYSFUNCTION VARIABLES		
Arterial hypoxaemia	Creatinine increase >0.5 mg/dl	Thrombocytopenia (platelet count < 100000/ microliter)
Acute oliguria (urine output <0.5 ml/kg/h for at least 2 hrs)	Coagulation abnormalities(INR> 1.5 or APTT >60 sec)	Hyperbilirubinemia
TISSUE PERFUSION VARIABLES		
Hyperlactatemia (>1 mmol/L)	Decreased capillary refill or mottling	

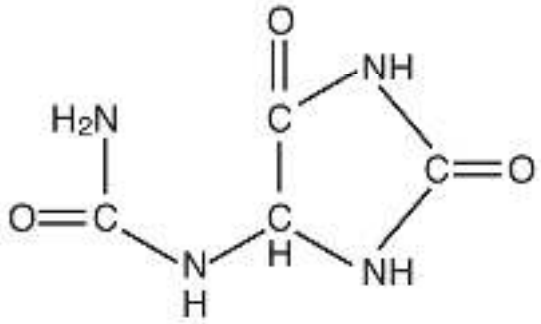
qSOFA SCORE: quick Sequential Organ Failure Assessment score		
Glasgow coma scale or altered sensorium	respiratory rate > 22	systolic BP < 100 mm Hg
one point each for altered mental status; GCS < 15, RR>=22, SBP <=100. A score of 2-3 prompts a high risk for sepsis		
qSOFA score of 2 or higher, there was a 3-14 fold increase in the rate of in hospital mortality [17].		

URIC ACID:

In humans, adenosine and guanosine is converted into uric acid. At first the adenosine molecule is metabolized into inosine by the enzyme adenosine deaminase. Uric acid is the end product of purine metabolism. The enzyme involved is xanthine oxidase.



Uric acid (excreted in humans)



Allantoin (excreted in other mammals)

The major source of excretion of uric acid is via kidney (70%) and tge remaining is via the GIT.inside the body with normal pH, uric acid is mostly found in the form of peroxynitrate.

The reference range of uric acid in humans is

1) In males – 3.4 to 7.2 mg/dl
2) In females – 2.4 to 6.1 mg/dl

Hyperuricemia is can be due to overproduction, undersecretion or both [13]. Patients suffering from CKD have elevated uric acid levels [10]. Other causes of raised uric acid levels are hypertension, hyperinsulinemia[8,9], atherosclerosis [3-7], COPD [18,19] and CHF [1,11,12].

When the uric acid levels in the body is too high, it leads to crystallization in uric acid crystals. This further lead to acute inflammation of kidney and other organs. This leads to activation of RAAS system in the body [20]. This further leads to activation of inflammatory factors and cytokine production [22].

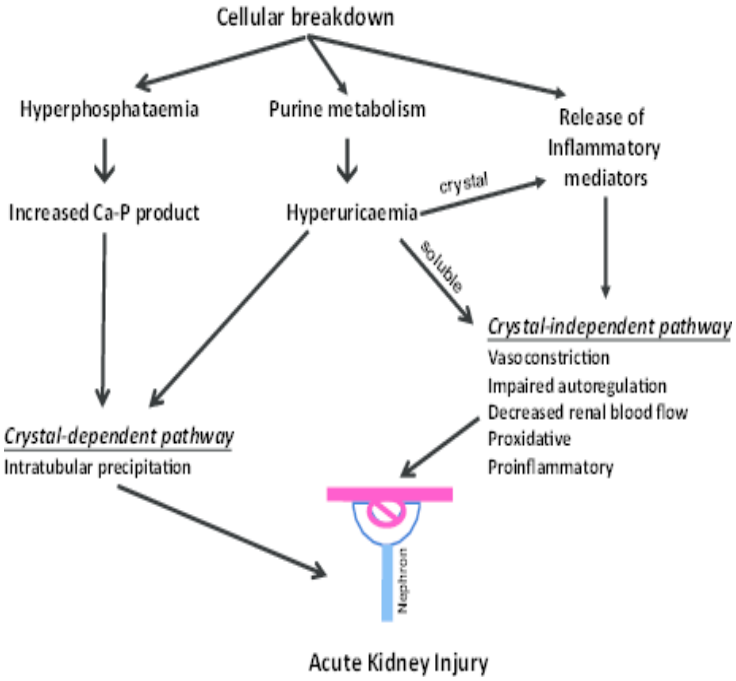
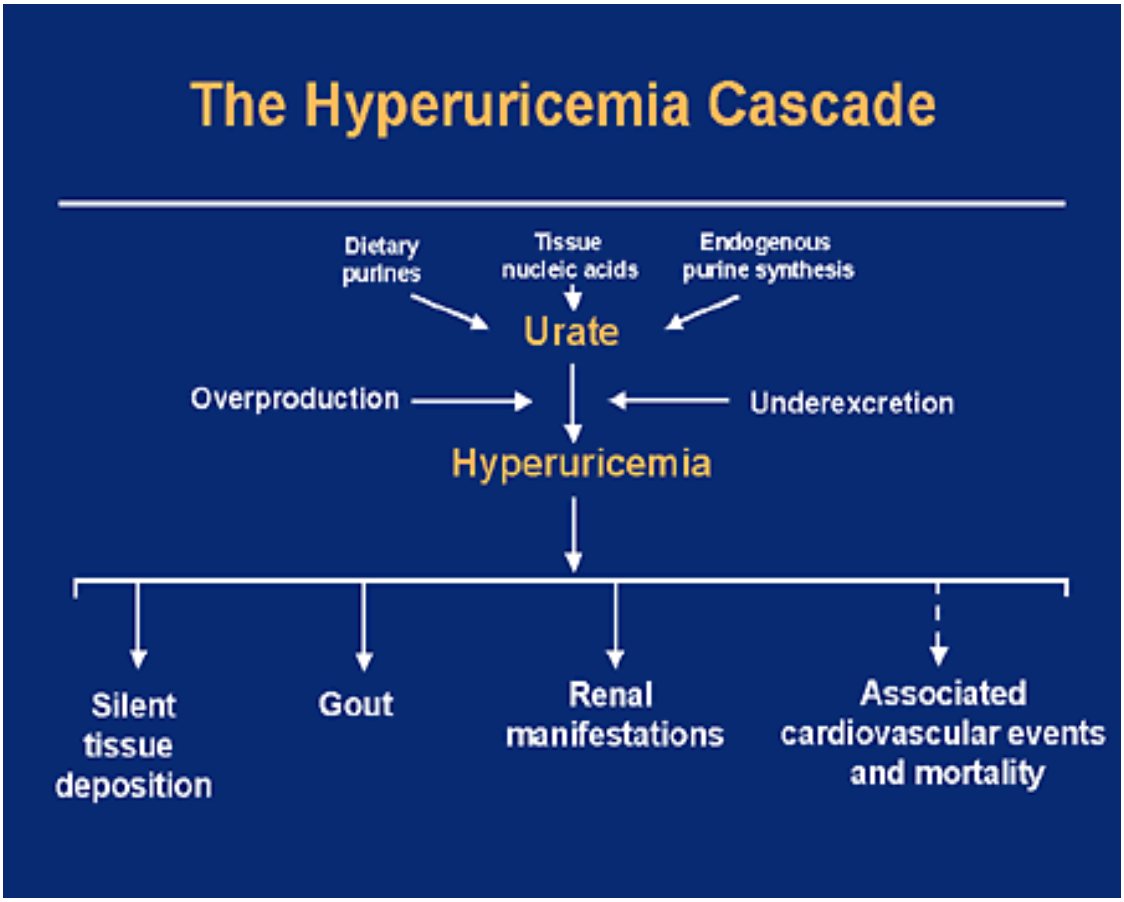


Figure 3 - pathophysiology of AKI in hyperuricemia

When the uric acid levels are elevated, it might cause AKI due to crystal deposition. These crystals, when precipitate, tend to block the renal tubules. AKI secondary to uric acid is due to:

1) Vasoconstriction
2) Reduced blood supply to kidney
3) Dysfunctional autoregulation
4) Increased inflammatory response [23,29,30]

Uric acid acts as both oxidant and antioxidant. Therefore, it can be used in the prediction of sepsis and its outcome [24].



MAJOR COMPLICATIONS OF SEPSIS:
1) Cardiopulmonary – ARDS, hypotension, shock etc
2) Renal - AKI
3) Neurological
4) DIC
5) MODS etc

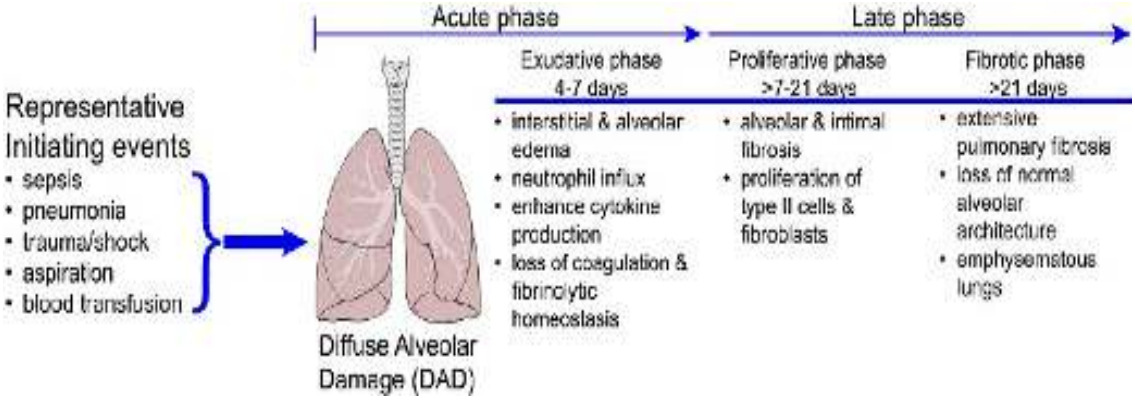


Figure 4- Pathophysiology of ARDS

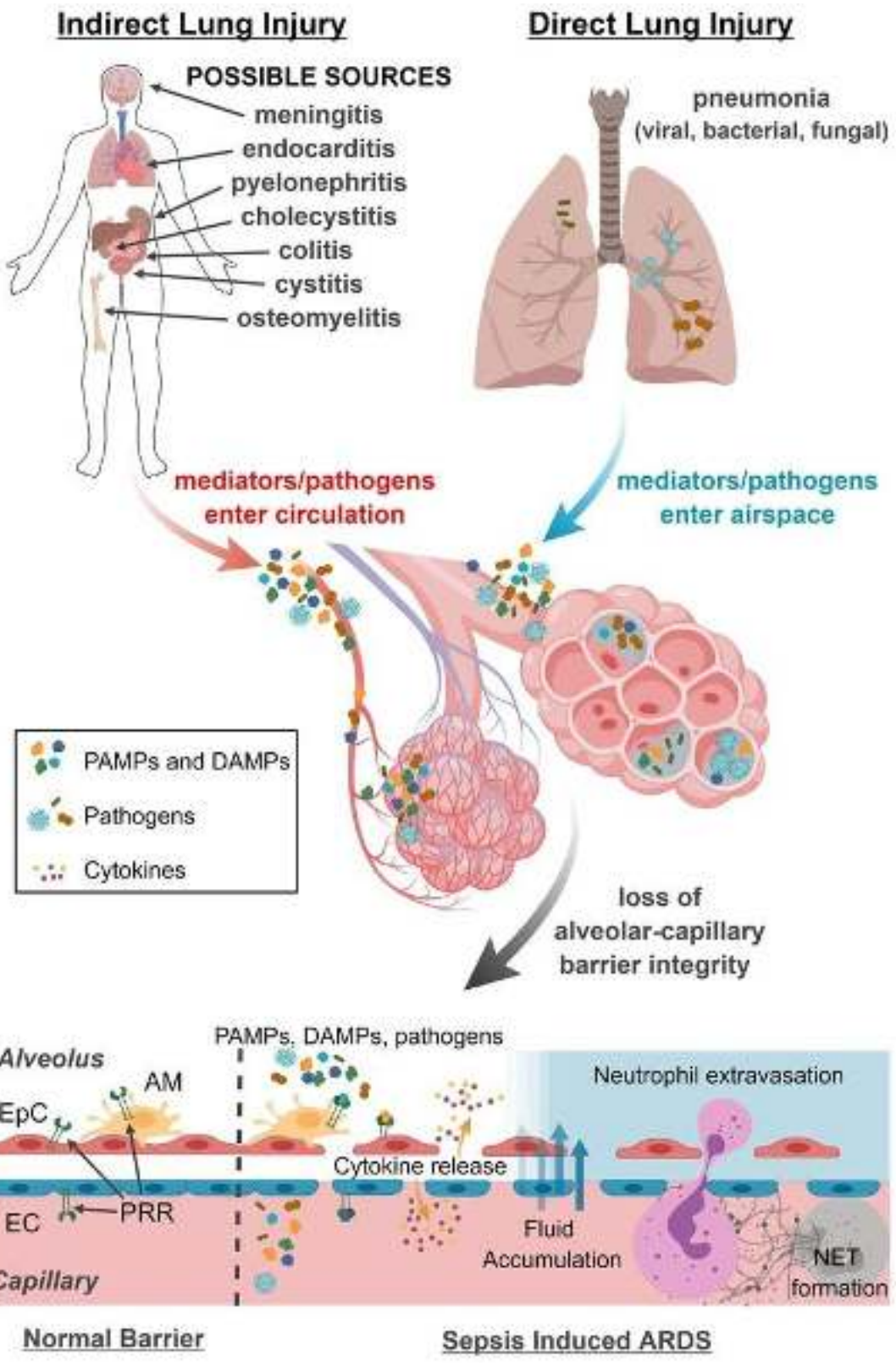


Figure 5- pathophysiology of sepsis induced ARDS

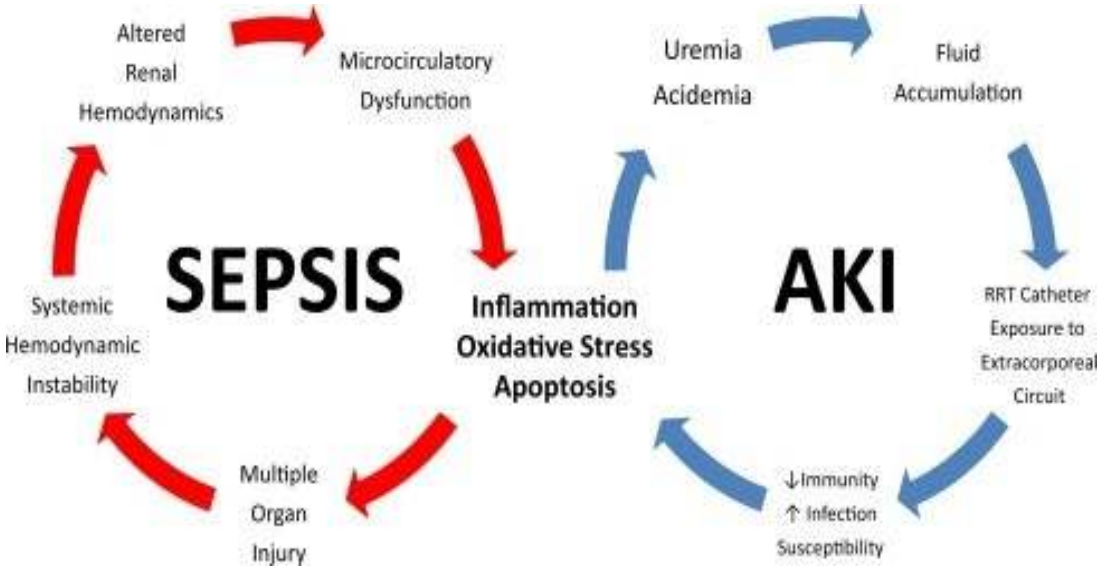


Figure 6- shows how inflammation and oxidative stress both cause the progression of sepsis and AKI

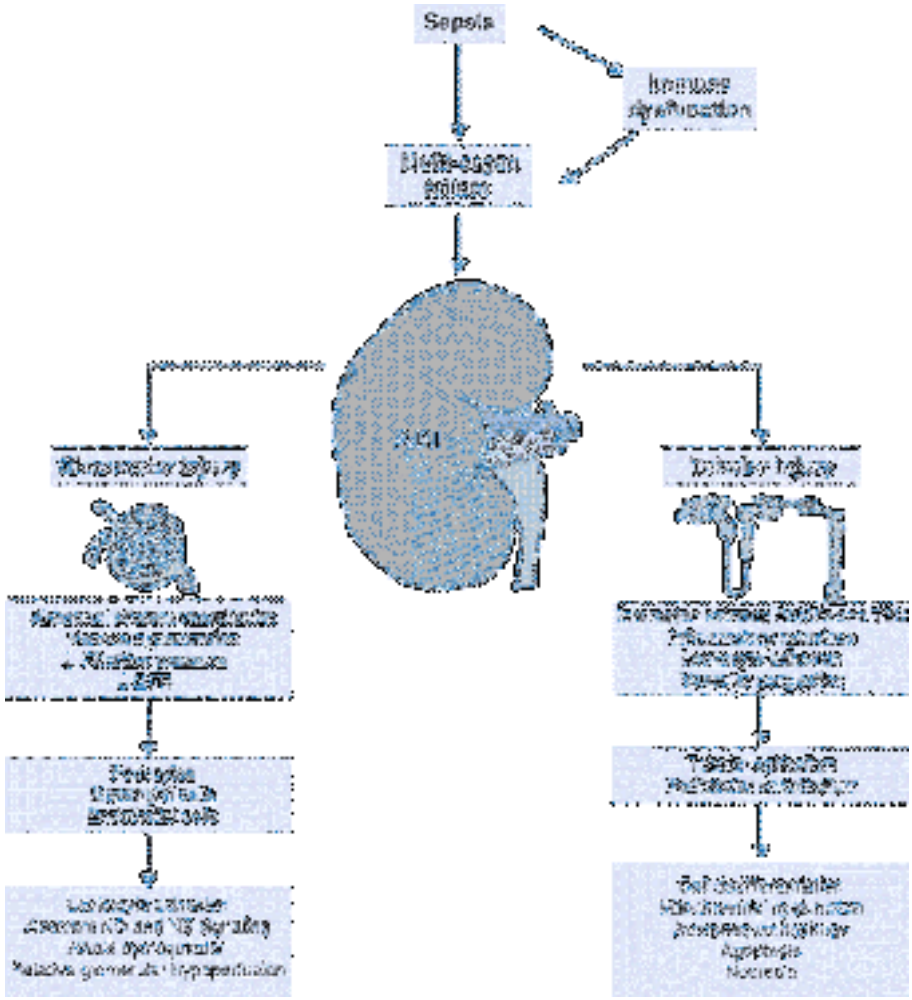


Figure 7 - pathophysiology of sepsis induced AKI

AKI is defined as any one of the following: (KDIGO DEFINITION)
1) Increase in serum creatinine by >=0.3 mg/dl within 48 hours
2) Increase in serum creatinine to >=1.5 times baseline, which is known or presumed to have occurred within the prior 7 days
3) Decrease in urine output <0.5 ml/kg/h for 6 hours

The other definitions are:
• RIFLE CRITERIA
1) Increase in serum creatinine by 1.5 times or GFR decrease by more than 25%
• AKIN CLASSIFICATION:
1) Increase in SCr>=0.3 mg/dl or
2) Increase of >= 150% to 200% (1.5 to 2 fold increase) from baseline within 48 hours

MATERIALS AND METHODS

The present study was conducted in the Department of General Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Study Design

This study was a hospital based cross-sectional study.

Study Period

It was conducted over a period of one year from January 2020 to December 2021.

Study Site

The present study was carried out at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. A tertiary care teaching hospital attached to Jawaharlal Nehru Medical College, Belagavi.

Study Population

All patients admitted to the wards of Department of General Medicine at KLES Dr. Prabhakar Kore Hospital, Belagavi fulfilling the inclusion criteria.

Sample Size

A total of 79 patients with anemia were studied.

Sampling Method

The following formula was used for calculation of the sample size

$$n = \frac{z_\alpha{}^2 P(1-P)}{d^2}$$

Where:

z_α = 1.96 (at 95% confidence interval)

P = percentage of prevalence

d = absolute error

INCLUSION CRITERIA:

- All patients admitted to Medical Intensive Care unit with a clinical diagnosis of sepsis, age more than 18 years based on the q SOFA criteria

EXCLUSION CRITERIA:

- Drugs known to cause hyperuricemia.
- Pregnant females.
- Patients with chronic kidney disease.
- Patients with gout.

Ethical Clearance

Prior to the commencement, the study was cleared by the Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belagavi.

Informed Consent

Informed consent was obtained from all the study participants and only those participants who willingly signed the informed consent were included in the study. The risks and benefits involved in the study, and the voluntary nature of participation were explained to the participants before obtaining consent. Confidentiality of the study participants was maintained.

Data Collection

All relevant parameters were documented in a structured Study Proforma

Methodology

- It uses three criteria, assigning one point for low blood pressure (SBP≤100 mmHg), high respiratory rate (≥22 breaths per min), or altered mentation (Glasgow coma scale<15). 2 or 3 points indicate high risk of poor outcome in patients with clinically suspected sepsis.
- Once the patient met the inclusion criteria blood samples were obtained for uric acid, urea, creatinine, complete blood count, arterial blood gas analysis, serum electrolytes and chest x ray was done.
- Patients data such as age, gender, comorbidities, ventilation status, need for renal replacement therapy, duration of stay in the hospital will be collected. We will use the baseline creatinine value the patient's creatinine value at the time of initial presentation to medical intensive care unit.
- The primary end point will be correlation between hyperuricemia in clinically suspected sepsis patients and morbidity and mortality rate.

Investigations

Venous blood samples were collected and subjected to the following investigations

- Complete blood count.
- Liver function test.
- HBA1C.
- USG abdomen.
- Urine routine and microscopy.
- Chest x-ray
- Wound culture/ Urine culture/ blood culture/ CT-abdomen, CT-Thorax (if needed) .

Statistical Methods

- Since the study is of prospective cohort study the plan of analysis will be as follows.
- For the continuous quantitative variables mean and standard deviation will be calculated. For the purpose of comparison if the data is divided into two groups with respect to certain qualitative characteristic, the continuous variables will be compared using suitable tools of statistics like student's unpaired t test. The pre and post treatment measures will be compared using student's paired t test
- Discrete variables will be represented by median.
- The categorical data will be expressed in terms of rates, ratios and percentages. The association between the outcome, clinical and demographic

characteristics will be tested using Chi-square test, test of proportion or Fisher's exact test.

- For discrete variables nonparametric tests will be used.
- Apart from the above suitable tools like ANOVA, correlation, regression etc., will be used according to the need.
- Suitable graphs will be used to depict the comparison.
- For all the tests the value of p less than 5% (0.05) will be considered significant.

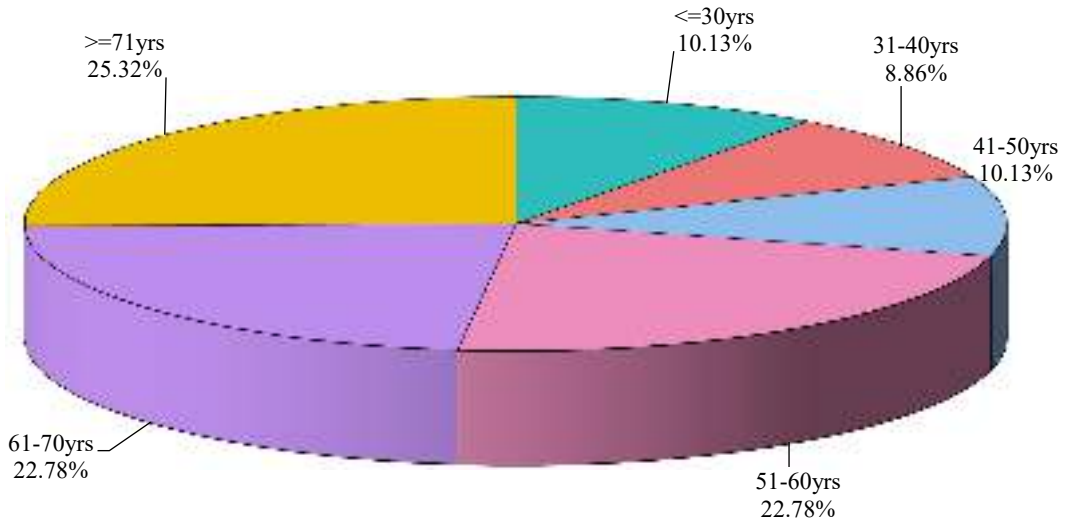
RESULTS

The present cross-sectional study titled “Hyperuricemia As An Early Marker In Predicting The Mortality And Morbidity In Patients With Sepsis” - A One Year Study In KLE’s Dr. Prabhakar Kore Hospital & MRC was carried out in the department of General Medicine. During the study period from January 2020 to December 2021, a total of 79 patients were studied. The findings / observations and final results are as tabulated below.

Table 1: Age wise distribution of patients

Age groups	No of patients	% of patients
<=30yrs	8	10.13
31-40yrs	7	8.86
41-50yrs	8	10.13
51-60yrs	18	22.78
61-70yrs	18	22.78
>=71yrs	20	25.32
Total	79	100.00
Mean age	57.77	
SD age	17.56	

Graph 1: Age wise distribution of patients



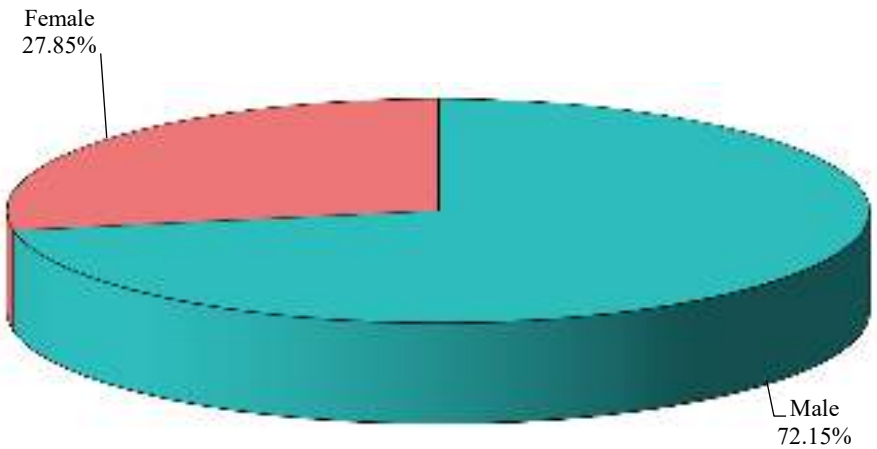
In the present study of 79 patients, ages ranged from 18-88 years. There were 20 patients (25.32%) above the age of 70 years, 18 patients (22.78%) each between 51-60 years and 61-70, 8 patients (10.13%) each in between 41-50 years and below 30 years. In between the age of 31-40 years and only 7 patients (8.86%) were present. The mean age was 57.77 with a standard deviation of 17.56 years.

Inference: maximum number cases were in age group of above 50 years and below 90 years.

Table 2: Gender wise distribution of patients

Gender	No of patients	% of patients
Male	57	72.15
Female	22	27.85
Total	79	100.00

Graph 2: Gender wise distribution of patients



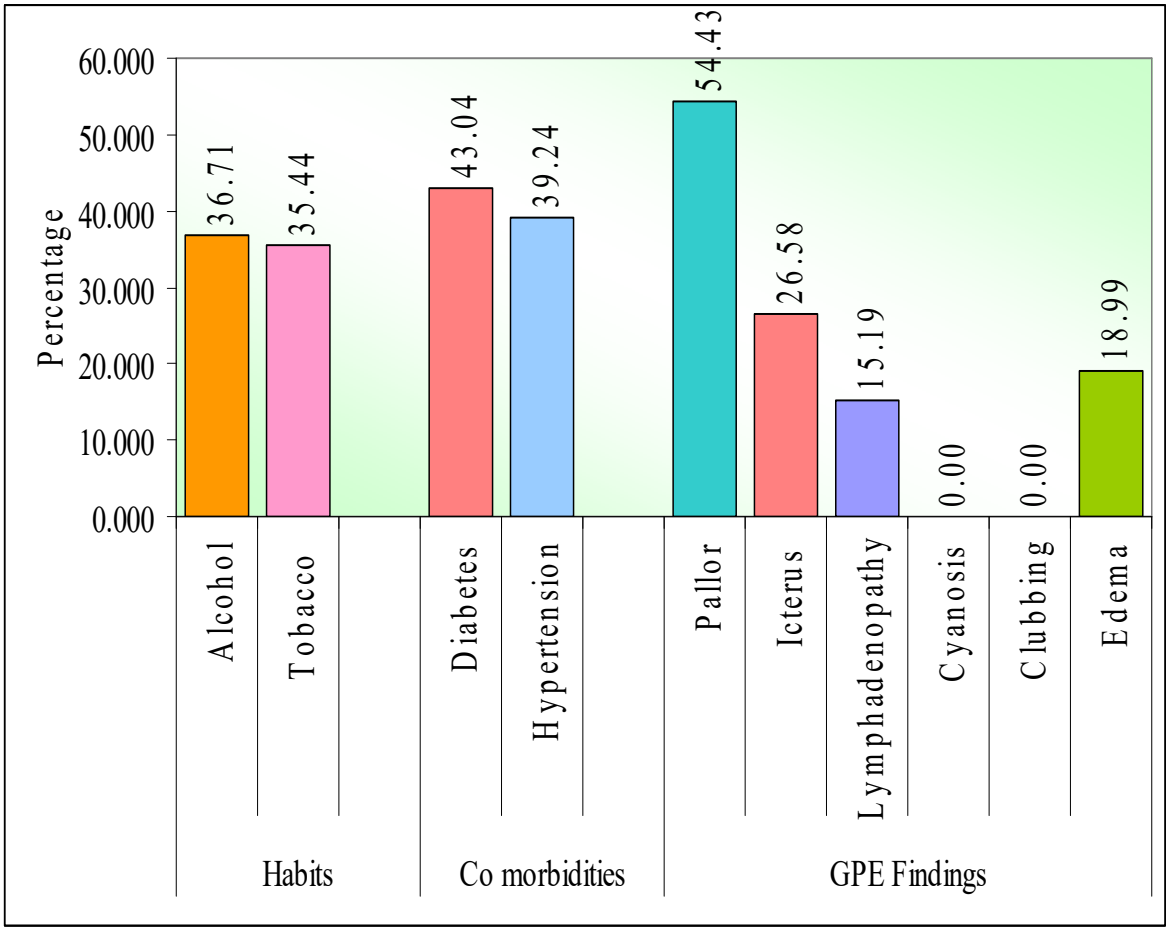
In our study, there were 57 male patients (72.15%) and 22 female patients (27.85%) accounting for a Male: Female ratio of 2.6:1.

Inference: there was a male preponderance in our study. 1

Table 3: Distribution of patients by habits, co-morbidities and GPE findings

	No of patients	% of patients
Habits		
Alcohol	29	36.71
Tobacco	28	35.44
Co morbidities		
Diabetes	34	43.04
Hypertension	31	39.24
GPE Findings		
Pallor	43	54.43
Icterus	21	26.58
Lymphadenopathy	12	15.19
Cyanosis	0	0.00
Clubbing	0	0.00
Edema	15	18.99

Graph 3: Distribution of patients by habits, co-morbidities and GPE findings



A total of 29 patients (36.71%) had habit of atleast alcohol drinking and 28 patients (35.44%) had tobacco use. 41 patients (52%) did not have any habits.

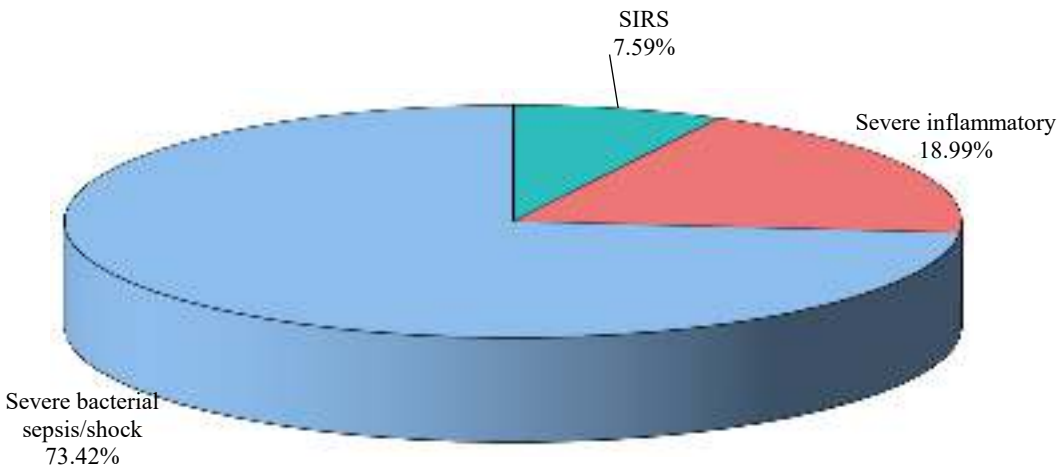
A total of 34 patients (43.03%) had atleast diabetes and 31 patients (39.24%) had atleast hypertension. 30 patients (39%) had no co-morbities.

Among the GPE findings in the study population, majority presented with palor accounting for 54.43% of patients. Cyanosis and clubbing was not seen any patients in the study population. Some patients had overlapping findings.

Table 4: Severity of sepsis wise distribution of patients

Severity of sepsis	No of patients	% of patients
SIRS	6	7.59
Severe inflammatory	15	18.99
Severe bacterial sepsis/shock	58	73.42
Total	79	100.00

Graph 4: Severity of sepsis wise distribution of patients



Among the patients in the study population, 6 patients (7.59%) had presented with SIRS, 15 patients (18.99%) with Severe inflammation and the majority presented with severe bacterial septicemia/ shock – 58 patients (73.42%).

Table 5: Association between gender and severity of sepsis

Severity of sepsis	Male	%	Female	%	Total	%
SIRS	5	8.77	1	4.55	6	7.59
Severe inflammatory	10	17.54	5	22.73	15	18.99
Severe bacterial sepsis/shock	42	73.68	16	72.73	58	73.42
Total	57	100.00	22	100.00	79	100.00
Chi-square=0.6000 P = 0.7410						

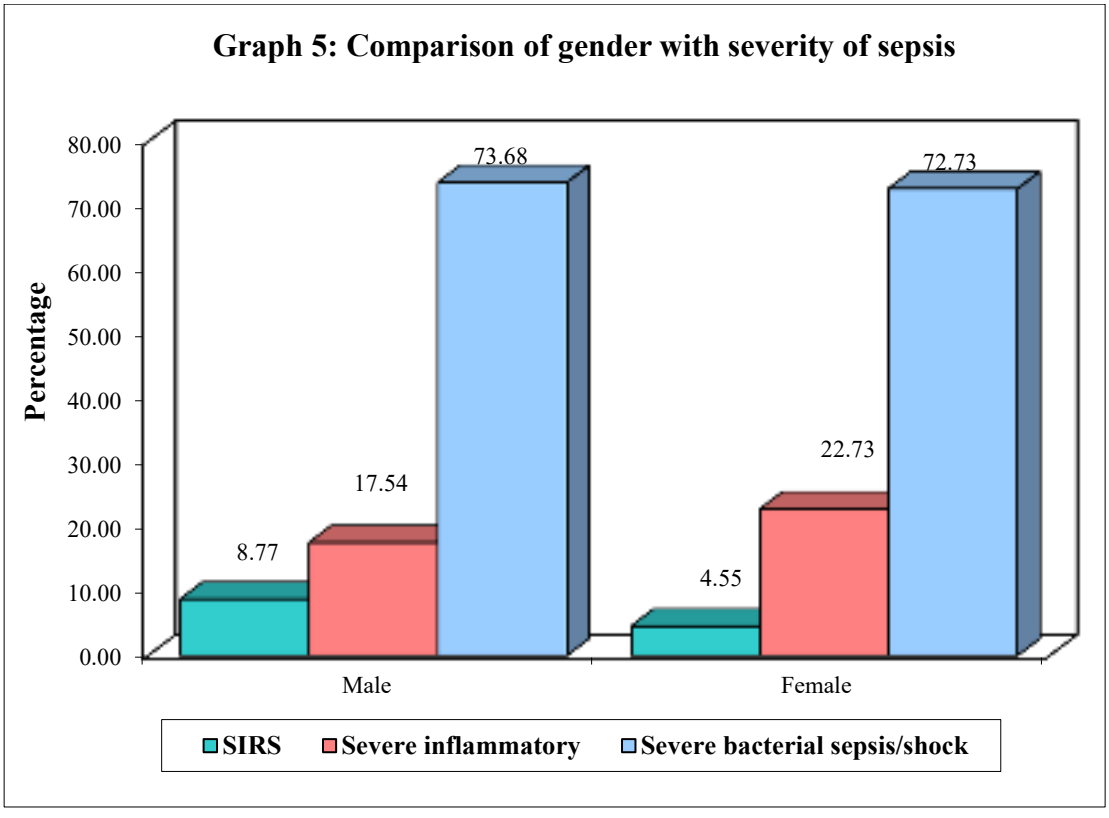


Table 6: Level of serum uric acid wise distribution of patients

Level of serum uric acid	No of patients	% of patients
<=7	25	31.65
>7	54	68.35
Total	79	100.00

Graph 6: Level of serum uric acid wise distribution of patients

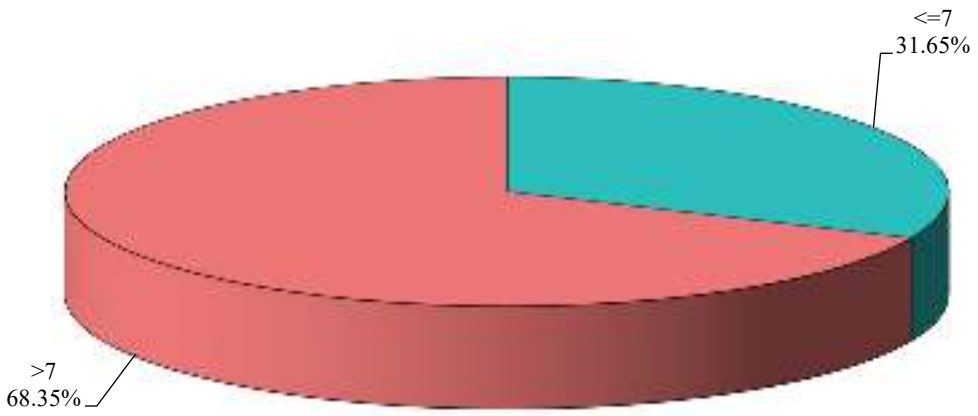


Table 7: Association between gender and levels of serum uric acid

Level of serum uric acid	Male	%	Female	%	Total	%
<=7	18	31.58	7	31.82	25	31.65
>7	39	68.42	15	68.18	54	68.35
Total	57	100.00	22	100.00	79	100.00
Chi-square=0.0001 P = 0.9840						

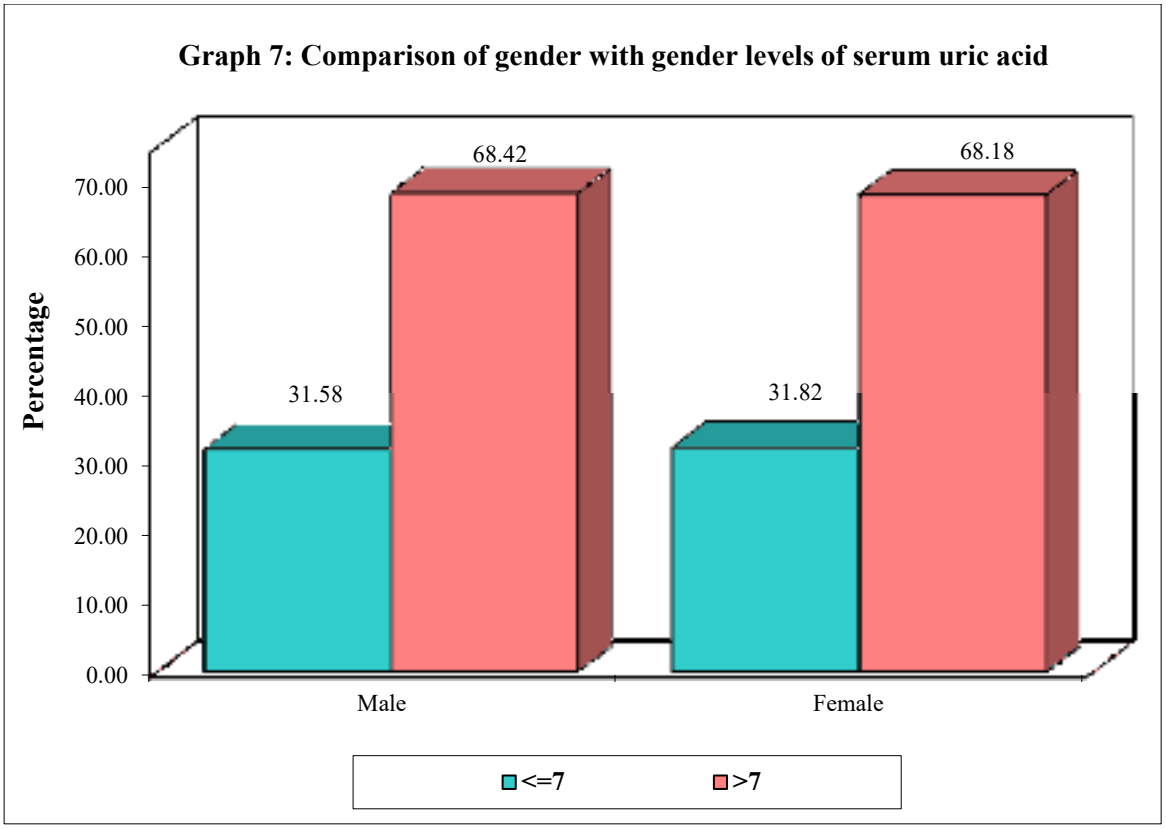


Table 8: Association between age groups and levels of serum uric acid

Age groups	<=7	%	>7	%	Total	%
<=30yrs	5	62.50	3	37.50	8	10.13
31-40yrs	0	0.00	7	100.00	7	8.86
41-50yrs	1	12.50	7	87.50	8	10.13
51-60yrs	4	22.22	14	77.78	18	22.78
61-70yrs	7	38.89	11	61.11	18	22.78
>=71yrs	8	40.00	12	60.00	20	25.32
Total	25	31.65	54	68.35	79	100.00
Chi-square=9.9380 P = 0.0770						

Graph 8: Comparison of age groups with levels of serum uric acid

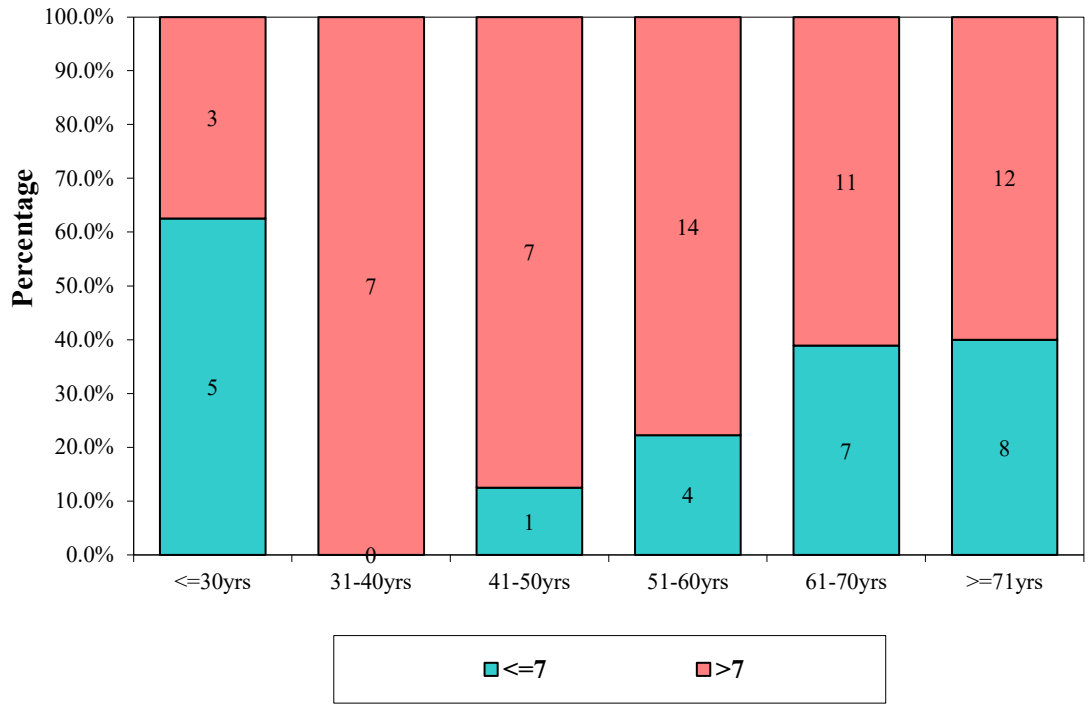
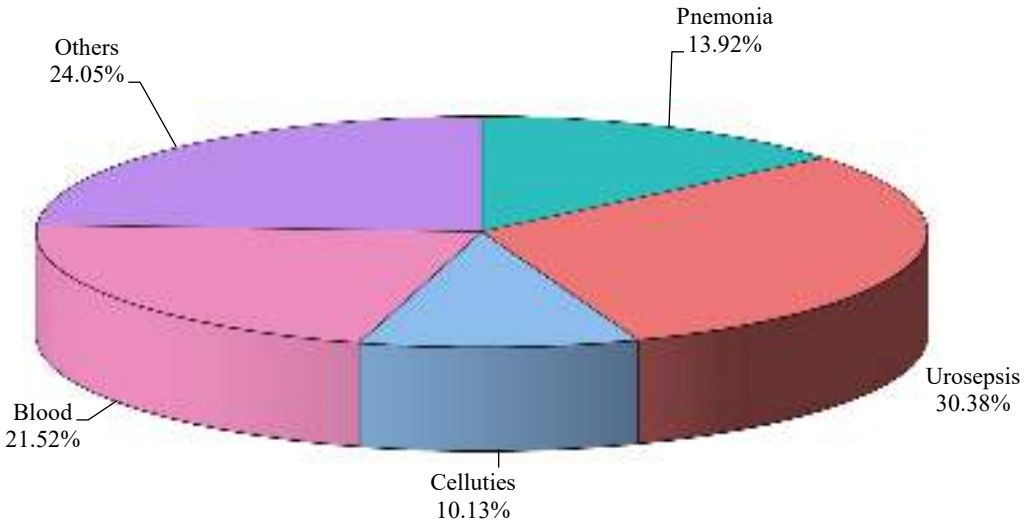


Table 9: Causes / focus of infection of sepsis wise distribution of patients

Causes / focus of infection of sepsis	No of patients	% of patients
Pnemonia	11	13.92
Urosepsis	24	30.38
Cellulitis	8	10.13
Blood	17	21.52
Others	19	24.05
Total	79	100.00

Graph 9: **Causes / focus of infection of sepsis wise distribution of patients**

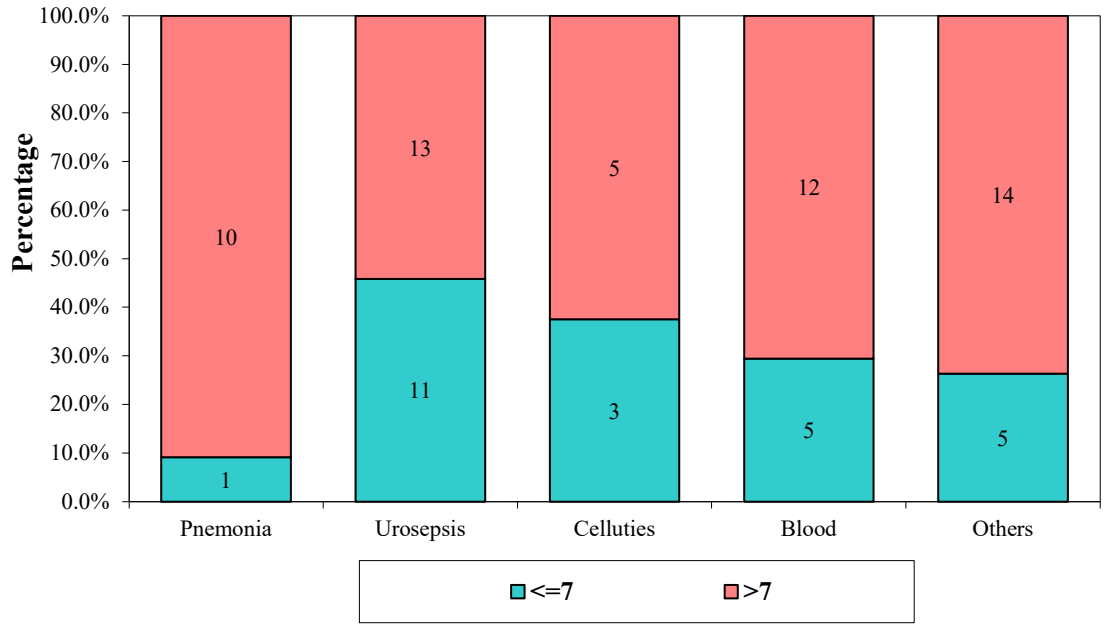


The above table shows the focus of sepsis. As tabulated, urosepsis being the major source of septicemia – 24 patients (30.38%) and cellulitis having the lowest patients (8 patients – 10.13%)

Table 10: Association between causes / focus of infection of sepsis and levels of serum uric acid

Causes / focus of infection of sepsis	<=7	%	>7	%	Total	%
Pnemonia	1	9.09	10	90.91	11	13.92
Urosepsis	11	45.83	13	54.17	24	30.38
Celluties	3	37.50	5	62.50	8	10.13
Blood	5	29.41	12	70.59	17	21.52
Others	5	26.32	14	73.68	19	24.05
Total	25	31.65	54	68.35	79	100.00
Chi-square=5.2360 P = 0.2640						

Graph 10: Comparison of causes / focus of infection of sepsis with levels of serum uric acid

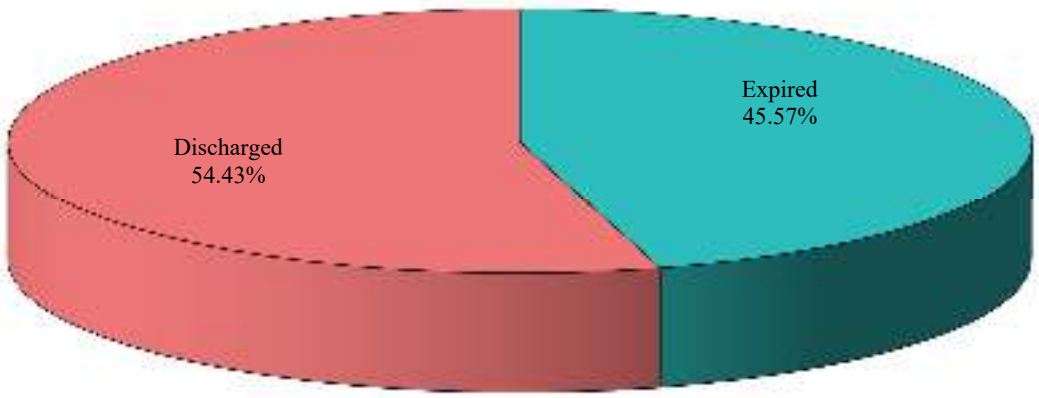


When we tried comparing the focus of septicemia with the uric acid levels, there was no significant correlation between the two variables (p value – 0.2640).

Table 11: Outcome wise distribution of patients

Outcome	No of patients	% of patients
Expired	36	45.57
Discharged	43	54.43
Total	79	100.00

Graph 11: Outcome wise distribution of patients



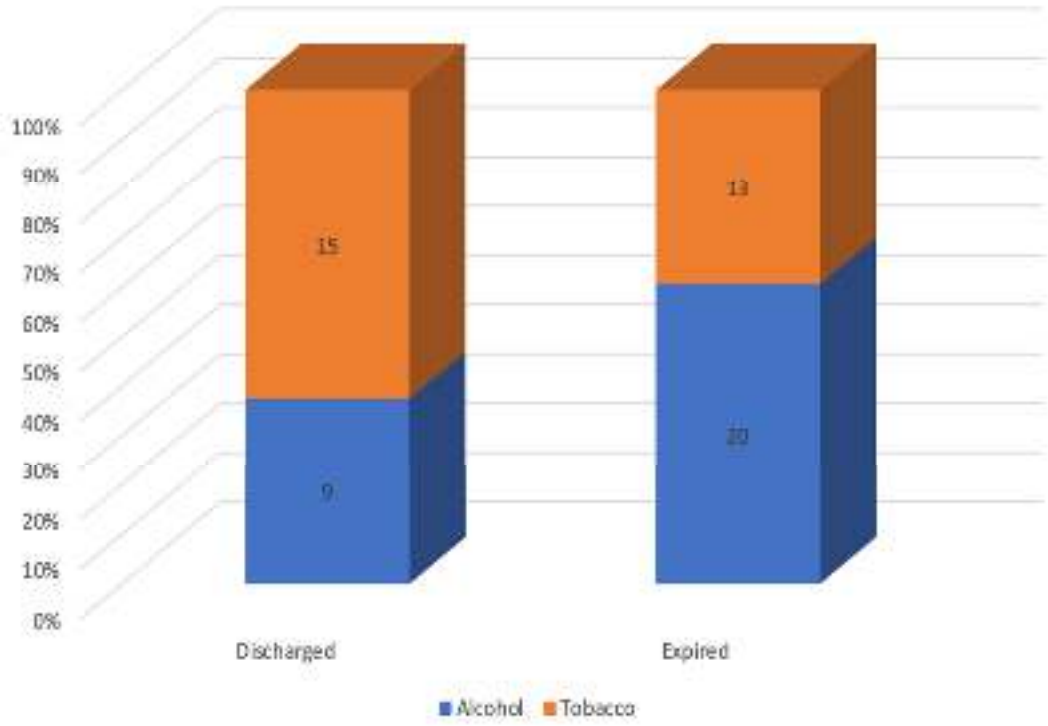
In this table above, we tried to compare the outcome of the patient on the basis of uric acid levels. Majority of the patients with septicemia were discharged – 43 patients (54.43%). Only 36 patients (45.57%) had expired.

Table 12: Comparison of outcome with status of habits

Habits	Discharged	%	Expired	%	Total	Chi-square	p-value
Alcohol	9	31.03	20	68.97	29	3.9030	0.0480*
Tobacco	15	53.57	13	46.43	28	0.0130	0.9100

*p<0.05

Graph 12: Habits vs outcome of patient



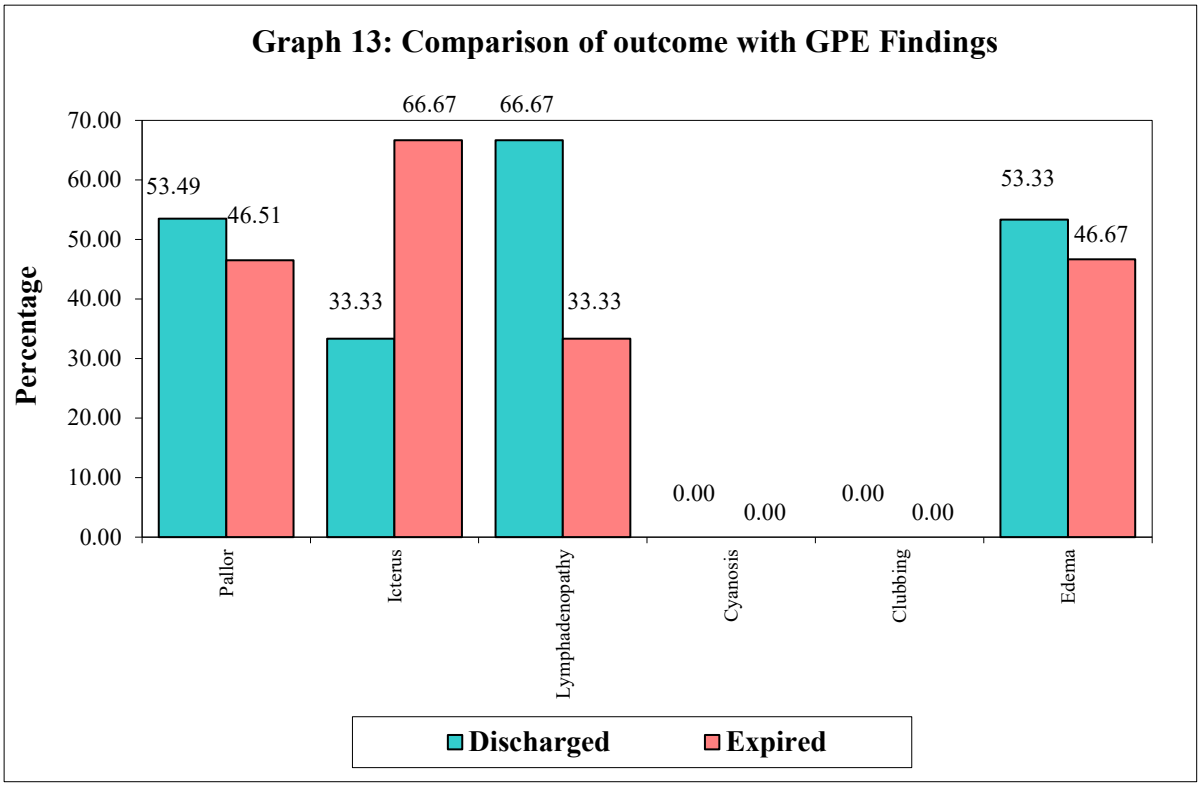
When we tried comparing the habits with outcome of the patients, we found that mortality is higher in patients having habits (alcohol). P value of 0.0480.

Table 13: Comparison of outcome with GPE Findings

GPE Findings	Discharged	%	Expired	%	Total	Chi-square	p-value
Pallor	23	53.49	20	46.51	43	0.0340	0.8540
Icterus	7	33.33	14	66.67	21	5.1330	0.0230*
Lymphadenopathy	8	66.67	4	33.33	12	0.8540	0.3550
Cyanosis	0	0.00	0	0.00	0	-	-
Clubbing	0	0.00	0	0.00	0	-	-
Edema	8	53.33	7	46.67	15	0.0090	0.9240

*p<0.05

Graph 13: Comparison of outcome with GPE Findings



The above tabulation column shows that there is significant correlation between icterus and the outcome of patient. Majority of patients having icterus have expired. P value 0.0230.

Table 14: Comparison of outcome with Level of serum uric acid, Severity of sepsis, PLT, Blood culture, Urine culture and ABG

	Discharged	%	Expired	%	Total	%	Chi-square	p-value
Level of serum uric acid								
<=7	20	80.00	5	20.00	25	31.65		
>7	23	42.59	31	57.41	54	68.35	9.6410	0.0020 *
Severity of sepsis								
SIRS	4	66.67	2	33.33	6	7.59		
Severe inflammatory response	14	93.33	1	6.67	15	18.99		
Severe bacterial sepsis/shock	25	43.10	33	56.90	58	73.42	12.5150	0.0020 *
PLT								
<150000	19	46.34	22	53.66	41	51.90	2.2480	0.1340
>=150000	24	63.16	14	36.84	38	48.10		
Blood culture								
No growth	28	56.00	22	44.00	50	63.29	0.1350	0.7130
Growth	15	51.72	14	48.28	29	36.71		
Urine culture								
No growth	25	60.98	16	39.02	41	51.90	1.4720	0.2250
Growth	18	47.37	20	52.63	38	48.10		
ABG								
Metabolic acidosis	30	57.69	22	42.31	52	65.82	5.5210	0.2380
Metabolic alkalosis	1	20.00	4	80.00	5	6.33		
Respiratory acidosis	3	50.00	3	50.00	6	7.59		
Respiratory alkalosis	5	83.33	1	16.67	6	7.59		
Normal ABG	4	40.00	6	60.00	10	12.66		
Total	43	54.43	36	45.57	79	100.00		

*p<0.05

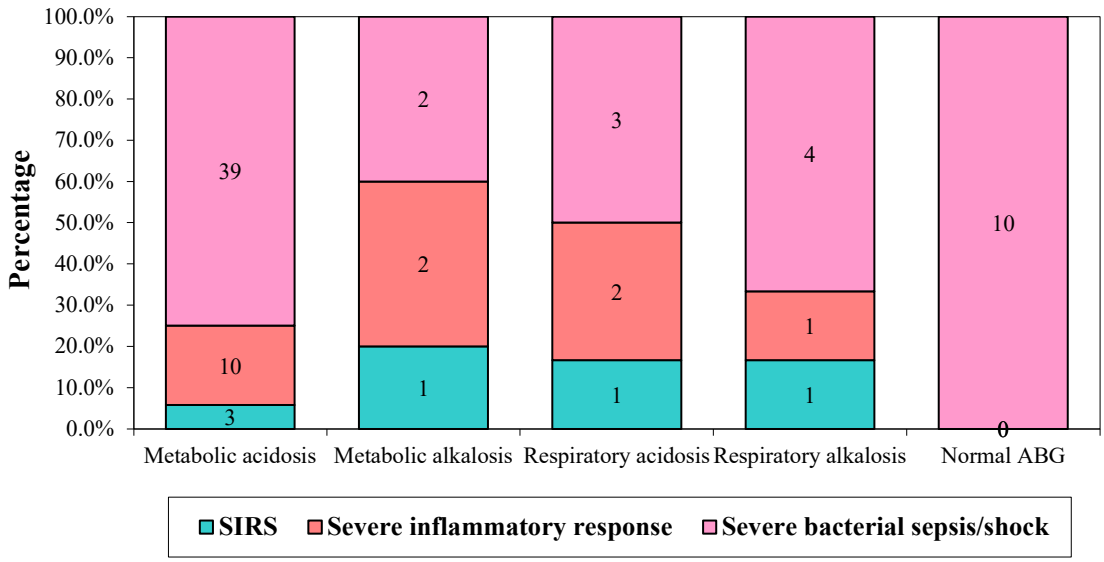
In our study, when we had tried comparing various laboratory parameters there was significant correlation between the uric acid levels and the outcome i.e., higher the uric acid levels more is the mortality. (p value 0.0020).

As indicated in the tabular column, there was significant correlation between the severity of sepsis and the outcome i.e., septicemia with shock had greater mortality (p value 0.0020)

Table 15: Comparison of severity of sepsis (serum PCT) with ABG

ABG	SIRS	%	Severe inflammatory response	%	Severe bacterial sepsis/shock	%	Total
Metabolic acidosis	3	50.00	10	66.67	39	67.24	52
Metabolic alkalosis	1	16.67	2	13.33	2	3.45	5
Respiratory acidosis	1	16.67	2	13.33	3	5.17	6
Respiratory alkalosis	1	16.67	1	6.67	4	6.90	6
Normal ABG	0	0.00	0	0.00	10	17.24	10
Total	6	100.00	15	100.00	58	100.00	79
Chi-square=9.2580, p=0.3211							

Graph 14: Comparison of severity of sepsis (serum PCT) with ABG



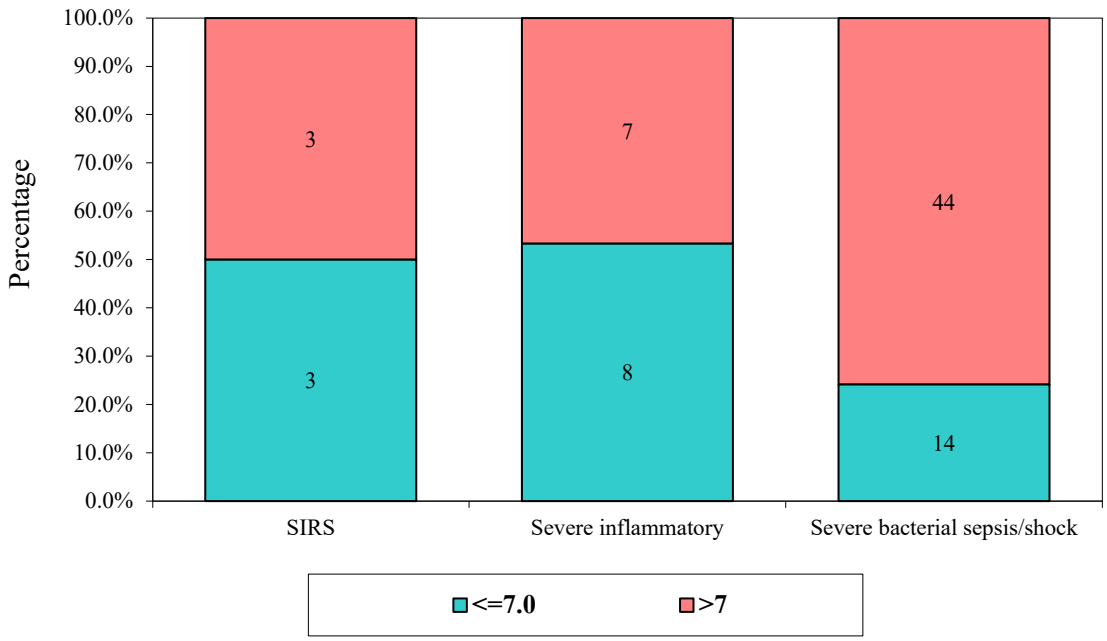
Severity of sepsis with respect to the serum PCT levels were compared with the ABG values had not shown any significant correlation.

Table 16: Association between levels of uric acid and severity of sepsis

Severity of sepsis	<=7.0	%	>7	%	Total	%
SIRS	3	50.00	3	50.00	6	7.59
Severe inflammatory	8	53.33	7	46.67	15	18.99
Severe bacterial sepsis/shock	14	24.14	44	75.86	58	73.42
Total	25	31.65	54	68.35	79	100.00
Chi-square=5.7070 P = 0.0500*						

*p<0.05

Graph 15: Comparison of levels of uric acid with severity of sepsis



In our present study of 79 patients, the data shows there is a significant correlation and association between the serum uric acid levels and severity of sepsis. Maximum number of patients with hyperuricemia have fallen under the category of severe sepsis/ shock (p value 0.0500).

Table 17: Step wise multivariate logistic regression analysis of outcome with all variables

Factors	OR	95% C.I. for OR		P-value
		Lower	Upper	
Alcohol				
No	Ref.			
Yes	0.28	0.10	0.84	0.0230*
Serum Uric acid				
<=7	Ref.			
>7	4.34	1.34	14.08	0.0150*
Severity of sepsis				
SIRS	Ref.			
Severe inflammatory	0.05	0.01	0.44	0.4430
Severe bacterial sepsis/shock	0.67	0.24	1.87	0.0070*

*p<0.05

Similarly, when a stepwise multivariate logistic regression analysis of outcome with all the variables in the study was applied to the tabulated data, we have observed a significant correlation between the alcohol consumption (p value 0.0230), hyperuricemia (p value 0.0150) and severity of septicemia (p value 0.0070).

Table 18: Summery of different lab parameters

Lab parameters	Valid N	Min	Max	Range	Mean	Median	Std.Dev.	95% CI for mean	
								Lower	Upper
PR	79	92.00	145.00	53.00	123.27	124.00	12.03	120.57	125.96
RR	79	22.00	42.00	20.00	29.48	28.00	5.13	28.33	30.63
SBP	79	70.00	100.00	30.00	89.24	90.00	7.12	87.65	90.84
DBP	79	50.00	70.00	20.00	59.37	60.00	6.86	57.83	60.90
Serum Uric acid	79	2.60	14.40	11.80	8.42	8.40	2.64	7.82	9.01
Serum PCT	79	0.13	196.00	195.87	39.90	27.90	37.74	31.45	48.35
HB	79	2.70	17.80	15.10	10.50	10.20	2.95	9.84	11.16
PLT	79	10000.00	518000.00	508000.00	157501.27	130000.00	108449.79	133209.84	181792.70
TC	79	2000.00	278000.00	276000.00	18989.87	14400.00	30499.26	12158.41	25821.34
Na	79	113.00	165.00	52.00	135.15	135.00	8.74	133.19	137.11
k	79	3.02	8.40	5.38	4.66	4.40	1.10	4.41	4.90
bicarb	79	7.00	35.00	28.00	15.53	15.00	4.94	14.43	16.64
Cl	79	32.00	127.00	95.00	98.89	100.00	11.48	96.31	101.46

Creat	79	0.20	23.87	23.67	3.65	2.27	4.04	2.75	4.56
TB	79	0.10	34.60	34.50	2.05	0.60	4.77	0.99	3.12
DB	79	0.05	28.78	28.73	1.60	0.40	4.08	0.68	2.51
OT	79	9.00	733.00	724.00	80.81	35.00	133.97	50.80	110.82
PT	79	4.00	624.00	620.00	46.28	26.00	78.49	28.70	63.86
ALP	79	26.00	388.00	362.00	127.01	112.00	68.63	111.64	142.39
total protein	79	3.70	56.00	52.30	6.53	5.90	5.71	5.25	7.80
Duration of stay in days	79	4.00	18.00	14.00	8.51	8.00	2.79	7.88	9.13
Serum Uric acid	79	2.60	14.40	11.80	8.42	8.40	2.64	7.82	9.01
Serum PCT	79	1.00	196.00	195.00	39.92	27.90	37.72	31.47	48.37
PLT		10000.00	518000.00	508000.00	157501.27	130000.00	108449.79	133209.84	181792.70

The above table shows the various numerical data which is a summary of our study. (CBP, RFT, LFT, PCT, Uric acid etc.)

DISCUSSION

In the study which we conducted at KLEH, out of 79 patients of septicemia in comparison with serum uric acid levels with various factors like age, sex, severity of sepsis, comorbidities, the following results were observed.

In our present study, the age of patients ranged from 22 to 91 years. The maximum number of patients are from the age group of >=71 years i.e., 20 patients (25%), 18 patients (23%) in the age group of 51-60 years and 61 to 70 years each respectively, 8 patients (10%) each were in the age group of less than 30 years and 41-50 years. The mean age was 57.77±17.56 years. In a study by Nitish M Bhandary,[XX] in their study group they observed more patients in age group of 31 to 64 years with mean age of 25.34± 6.27years.

In sepsis there is increase in cytokines and oxidative stress. The body has a defense mechanism to balance both pro inflammatory and antioxidant levels. in SIRS, endothelial dysfunction and free radical production via the neutrophils are responsible for the damage and can cause increased mortality in patients [36,37,38]. Conditions like sepsis increases the chances of stress in the body leading to increase in the levels of uric acid [39,40]. Uric acid has both proinflammatory and antioxidant properties. In periods of great stress such as sepsis, the protective feature of uric acid is outgunned and the pro inflammatory feature becomes predominant. At this point these elevated levels of uric acid becomes more harmful than protective. The fine balance between the levels of uric acid and sepsis becomes the corner stone of our study. thus uric acid can be an early marker for assessing the progression of sepsis in to shock and MODS ultimately leading to the mortality of the patient.

Therefore, uric acid is an important marker of stress due to conditions like septicemia. The actual mechanism for raised uric acid are not well documented and there is a void in the literature available. It can be due to both heightened production and reduced elimination out of the body.

During the conditions of sepsis and septic shock, activation of the microvascular endothelium will cause activation of the enzymes responsible for production of uric acid. These enzymes include, xanthine oxidase etc. [42,43]. Raise in the levels of uric acid in the blood vessels lead to reduced expression of genes responsible for production of vasodilator agents [3]. This further escalates the problem of sepsis leading to MODS. These factors make the hospitalization of the patient prolonged and is associated with higher consumption of resources, both human and non-human [16].

The first significant correlation that was found in our study was the relation between the outcome of the patient with habits associated with patient. It was observed that the patients having alcohol had more mortality when compared to patients with no habits. When there is excessive consumption of alcohol in the patients, there is excess load on the liver to metabolize alcohol. This leads to occurrence of hepatitis/chronic liver disease. These factors impair the excretion of uric acid from the body [44,45].

In the patients who are septicemic, the complex evolution of SIRS and MODS do occur very frequently. These complex hemodynamic changes occurring in the patients bodies are responsible for the increase in mortality of patients. The main method of uric acid causing MODS can be via direct injury to the tubular structures of kidney and indirect injury leading to the raised levels of vasoactive peptides

leading to vasoconstriction and reduced blood flow to the vital organs.

Activation of RAAS due to the elevated uric acid levels is secondary to the increased catecholamine levels in the body. These factors cause significant vasoconstriction leading to reduced perfusion to the vital organs leading to MODS. Few experiments on rats had revealed greater extents of afferent arterial constriction and reduced nitric oxide levels secondary to elevated uric acid levels [46,47].

The main dilemma in the management of patients with sepsis with elevated uric acid levels is whether there is really a proven benefit of treating the patients based on the uric acid levels. Can uric acid levels act as a marker for early sepsis? Can uric acid be used as a sensitive marker in predicting the severity of sepsis outcome of the patient? Can patients with early sepsis with raised uric acid levels be treated to reduce the degree of sepsis that it may decrease the morbidity and mortality rate in this patient population.

Uric acid levels are increased in subjects with renal disease as the result of reduction in GFR and renal urate excretion. Chonchol et al. have reported that uric acid levels are associated strongly with prevalent CKD [51]. Because of progressive loss of GFR, patients with CKD have decreased renal clearance of uric acid and thus greater serum uric acid levels than the general population [52]. is. This raises the question then in the management of

In our study the levels of uric acid had significant correlation between the mortality and severity of sepsis. Although, it could not significantly predict the incidence of MODS and other organ damages. In a study conducted by Khoasla KV et al. [48] had revealed a drop in plasma nitrites levels (by product of NO

metabolism) in rats with elevated uric acid levels. in another study conducted by the French scientist, Zocalli kavilo et al. [49], had shown a significant correlation between elevated serum uric acid values and endothelial dysfunction [50].

This could potentially be due to the small patient population that we had for our study, especially since increasing uric acid levels have been reported by Nagaya et al. [53] to correlate with clinical severity of primary pulmonary hypertension and has an independent association with long-term mortality of patient with primary pulmonary hypertension. This was most likely due to the small sample size.

Regarding the outcome of septic patients, though there was a slight increase in mortality among the hyperuricemic individuals with sepsis than those with normal Uric acid levels, it was statistically significant to prove the point.

CONCLUSION

In our present study of 79 patients in sepsis, we observed insignificant correlation with various factors. Prominent features of our study are mentioned as follows.

- Among the patients who presented with anemia, majority of our patients were in age group of above 71 years.
- Age did not have an influence on uric acid levels.
- Sex of the patients did not influence the uric acid levels.
- Males were slightly more in number compared to females in our study.
- Among the GPE findings in the study population, majority presented with palor accounting for 54.43% of patients. Cyanosis and clubbing were not seen any patients in the study population. Some patients had overlapping findings.
- Majority of our patients presented with severe bacterial septicemia/ shock.
- There was significant correlation between the uric acid levels and the outcome i.e., higher the uric acid levels more is the mortality.
- There is a significant correlation and association between the serum uric acid levels and severity of sepsis. Maximum number of patients with hyperuricemia have fallen under the category of severe sepsis/ shock.
- We feel that it is worthwhile to take large sample size with confounding factors like age, sex, habits, treatment of sepsis to see whether there is a true correlation between these variables and the outcome**.**

SUMMARY

In the present study of 79 patients with septicemia, admitted in the Department of General Medicine of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi, with the study period extending from January 2020 to December 2021 was conducted to know whether serum uric acid levels have any influence of various variables like age, sex, diet, habits on severity and outcome in patients with septicemia.

The results observed were all inconclusive except for hyperuricemia and outcome in patients with habits like alcohol consumption, hyperuricemia and severity of septicemia. So, we feel with a large sample size these issues have to be addressed, comparing different grades of septicemia, focus of septicemia on the outcome of the patient. We also feel that there is necessary to follow up the patients with treatment of septicemic patients and its effect on overall wellbeing of the patient. We did not find any positive correlation with variables like age, sex, habits and severity of sepsis and its outcome.

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ANNEXURE I. ETHICAL CLEARANCE.

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed to be University)
Accredited 'A' Grade by NAAC (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
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Ref: MDC/DOME/ 207 Date: 24/12/2019

To

REGISTRATION NO: BG0119010

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 **"HYPERURICEMIA AS AN EARLY MARKER IN PREDICTING THE MORTALITY AND MORBIDITY IN PATIENTS WITH SEPSIS"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURE II

INFORMED CONSENT

Title Of Research Study:

HYPERURICEMIA AS AN EARLY MARKER IN PREDICTING MORTALITY AND MORBIDITY IN PATIENTS WITH SEPSIS"

Principal Investigator:-
REGISTRATION NO: BG0119010
Post Graduate Student,
Department Of General Medicine,
JNMC, Belgaum.

Guide:-
Dr._____
Associate Professor
Department of General Medicine,
JNMC, Belgaum.

Introduction and Purpose:- It is a well recognized clinical complication of sepsis and the presence and prompt identification of well defined precipitating factors is extremely important in diagnosis and treatment of this fatal condition.

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

-Risk and Benefits:
The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling, pain, redness (rarely happens) at the site from where the blood is drawn.You may not be benefitted by these investigations but you will be part of this study which is going to be useful to others in the future.

Alternatives:

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

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

Privacy and Confidentiality:

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

Institution / Sponsor's policy:

Does not apply to this research

Financial incentives for participation:

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:

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

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

2. Dr . _____ Associate Professor Dept of General Medicine, JNMC, Belgaum.	**REGISTRATION NO: BG0119010** Investigator, PG in General Medicine, JNMC, Belgaum. 8886355338

Dr. Roopa Bellad
Chairman,
College Ethical Dissertation
Research Committee
J. N. Medical. College
Nehru Nagar, Belagavi 590010

CONSENT FORM

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

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

Participant's name :……...

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

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

Signature / Left thumb impression :……...

Witness' name :……...

Signature / Left thumb impression :……...

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

Date:

Place:

ತಿಳುವಳಿಕೆಯ ಸಮ್ಮತಿ

ಸಂಶೋಧನಾ ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಸೆಪ್ಸಿಸ್ ರೋಗಿಗಳಲ್ಲಿ ಮರಣ ಮತ್ತು ಅಸ್ವಸ್ಥತೆಯನ್ನು ಹಿಸುವಲ್ಲಿ ಆರಂಭಿಕ ಗುರುತು ಆಗಿ ಹೈಪರ್ಲ್ಯಾಕ್ಟಿಸೆಮಿಯಾ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: -

REGISTRATION NO: BG0119010

ಸ್ನಾತಕೊತ್ತರ ವಿದ್ಯಾರ್ಥಿ,
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಮಾರ್ಗದರ್ಶಿ: -

ಡಾ. _____

ಸಹಾಯಕ ಪ್ರಾಧ್ಯಾಪಕ
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಪರಿಚಯ ಮತ್ತು ಉದ್ದೇಶ: -

ಇದು ಸೆಪ್ಸಿಸ್ನ ಉತ್ತಮವಾಗಿ ಗುರುತಿಸಲ್ಪಟ್ಟ ಕ್ಲಿನಿಕಲ್ ತೊಡಕು ಮತ್ತು ಈ ಮಾರಣಾಂತಿಕ ಸ್ಥಿತಿಯ ರೋಗನಿರ್ಣಯ ಮತ್ತು ಚಿಕಿತ್ಸೆಯಲ್ಲಿ ಉತ್ತಮವಾಗಿ ವ್ಯಾಖ್ಯಾನಿಸಲಾದ ಅವಕ್ಷೇಪನ ಅಂಶಗಳ ಉಪಸ್ಥಿತಿ ಮತ್ತು ತ್ವರಿತ ಗುರುತಿಸುವಿಕೆ ಬಹಳ ಮುಖ್ಯವಾಗಿದೆ.

ವಿಧಾನ:

ಸಂಶೋಧನಾ ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ನೀವು ಒಪ್ಪಿದರೆ, ನಿಮಗೆ ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ಕೇಳಲಾಗುತ್ತದೆ ಮತ್ತು ಸಂಬಂಧಿತ ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆ ಮತ್ತು ತನಿಖೆಗೆ ಒಳಪಡಿಸಲಾಗುತ್ತದೆ. ಅಗತ್ಯ ತನಿಖೆಗಾಗಿ ನೀವು ರಕ್ತ ಮತ್ತು ಮೂತ್ರದ ಮಾದರಿಗಳನ್ನು ಸಹ ನೀಡಬೇಕಾಗುತ್ತದೆ.

ಅಪಾಯ ಮತ್ತು ಪ್ರಯೋಜನಗಳು:

ತನಿಖೆಗಾಗಿ ನಿಮ್ಮ ತೋಳಿನಿಂದ ರಕ್ತವನ್ನು ತೆಗೆದುಕೊಳ್ಳುವಾಗ ನೀವು ಪಡೆಯುವ ಏಕೈಕ ಅಪಾಯ ಮತ್ತು ಸಂಭವನೀಯ ಅಸ್ವಸ್ಥತೆ. ಇದು ರಕ್ತವನ್ನು ಎಳೆಯುವ ಸ್ಥಳದಲ್ಲಿ ಬೆವರುವಿಕೆ, ನೋವು, ಕೆಂಪು (ವಿರಳವಾಗಿ ಸಂಭವಿಸುತ್ತದೆ) ಗೆ ಕಾರಣವಾಗಬಹುದು.

ಈ ತನಿಖೆಯಿಂದ ನಿಮಗೆ ಯಾವುದೇ ಪ್ರಯೋಜನವಾಗದಿರಬಹುದು ಆದರೆ ಭವಿಷ್ಯದಲ್ಲಿ ಇತರರಿಗೆ ಉಪಯುಕ್ತವಾಗಲಿರುವ ಈ ಅಧ್ಯಯನದ ಭಾಗವಾಗುತ್ತೀರಿ.

ಪರ್ಯಾಯಗಳು:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸದಿರಲು ನೀವು ಆಯ್ಕೆ ಮಾಡಬಹುದು.

ನೀವು ಭಾಗವಹಿಸಲು ನಿರ್ಧರಿಸಿದರೆ ನೀವು ನಂತರ ನಿಮ್ಮ ಮನಸ್ಸನ್ನು ಬದಲಾಯಿಸಬಹುದು ಮತ್ತು ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು. ನಿಮ್ಮ ನಿರ್ಧಾರವು ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೋಗ್ಯ ರಕ್ಷಣೆ ಅಥವಾ ನೀವು ಸ್ವೀಕರಿಸುವ ಇತರ ಸೇವೆಗಳನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ. ಅಧ್ಯಯನ ವೈದ್ಯರು ಅಥವಾ ಪ್ರಾಯೋಜಕರು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯನ್ನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನಿಲ್ಲಿಸಬಹುದು. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸದಿರಲು ನೀವು ಆರಿಸಿದರೆ, ನಿಮ್ಮ ಸ್ಥಿತಿಯ ರೋಗಿಗಳಿಗೆ ನೀವು ಪ್ರಮಾಣಿತ ಚಿಕಿತ್ಸೆಯನ್ನು ಸ್ವೀಕರಿಸುತ್ತೀರಿ.

ಗೌಪ್ಯತೆ ಮತ್ತು ಗೌಪ್ಯತೆ :

ಈ ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮ ಬಗ್ಗೆ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಕಾನೂನಿನಿಂದ ಅನುಮತಿಸುವ ಮಟ್ಟಿಗೆ ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ. ಈ ಸಂಶೋಧನಾ ದಾಖಲೆಯಲ್ಲಿ ಕೋಡ್ ಸಂಖ್ಯೆಗಳು ನಿಮ್ಮನ್ನು ಗುರುತಿಸುತ್ತವೆ. ಈ ಅಧ್ಯಯನದ ಮಾಹಿತಿಯನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಆದರೆ ಯಾವುದೇ ಪ್ರಕಟಣೆಯಲ್ಲಿ ನಿಮ್ಮ ಗುರುತು ಗೌಪ್ಯವಾಗಿರುತ್ತದೆ.

ಸಂಸ್ಥೆ / ಪ್ರಾಯೋಜಕರ ನೀತಿ: ಈ ಸಂಶೋಧನೆಗೆ ಅನ್ವಯಿಸುವುದಿಲ್ಲ

ಭಾಗವಹಿಸುವಿಕೆಗೆ ಆರ್ಥಿಕ ಪ್ರೋತ್ಸಾಹ :

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿಮಗೆ ಯಾವುದೇ ಉಡುಗೊರೆಗಳನ್ನು / ಪ್ರೋತ್ಸಾಹಕಗಳನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ / ನೀಡಲಾಗುವುದಿಲ್ಲ.

ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸಲು ಅಧಿಕಾರ:

ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಭಾಗವಾಗಿ ಬೆಳಗಾವಿಯ ಕೆಎಲ್ಇ ವಿಶ್ವವಿದ್ಯಾಲಯಕ್ಕೆ ರವಾನಿಸಲಾಗುತ್ತದೆ
ಎಂಡಿ ಪದವಿ, ವಿಮರ್ಶೆ ಮತ್ತು ಪ್ರಕಟಣೆಯ ಪೂರ್ಣಗೊಳಿಸುವಿಕೆಯ ಅವಶ್ಯಕತೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಅಥವಾ ಭವಿಷ್ಯದಲ್ಲಿ ನೀವು ಈ ಕೆಳಗಿನ ವ್ಯಕ್ತಿಗಳನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು,

ಡಾ._____

ಸಹಾಯಕ ಪ್ರಾಧ್ಯಾಪಕ
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

REGISTRATION NO: BG0119010

ತನಿಖಾಧಿಕಾರಿ,
ಸ್ನಾತಕೊತ್ತರ ವಿದ್ಯಾರ್ಥಿ,
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಡಾ.ರೂಪಾ ಎಂ ಬೆಲ್ಲಾದ, ಎಂಡಿ
ಅಧ್ಯಕ್ಷ, ಕಾಲೇಜು ನೈತಿಕ ಪ್ರಬಂಧ
ಸಂಶೋಧನಾ ಸಮಿತಿ ಜೆ.ಎನ್. ವೈದ್ಯಕೀಯ. ಕಾಲೇಜು
ನೆಹರೂ ನಗರ, ಬೆಳಗಾವಿ - 590010

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: .....

ಭಾಗವಹಿಸುವವರ ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: .....

ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕೃತ ಪ್ರತಿನಿಧಿ / ರಕ್ಷಕರ ಹೆಸರು: ....

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ಸಾಕ್ಷಿಯ ಹೆಸರು: ....

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ದಿನಾಂಕ:

ಸ್ಥಳ

माहितीपूर्ण संमती

संशोधन अभ्यासाचे शीर्षकः सेप्सीस ग्रस्त रूग्णांमध्ये मृत्यूची आणि रूग्णपणाची भविष्यवाणी करण्यासाठी लवकर मार्कर म्हणून हायपर्यूरिसेमिया

प्रधान अन्वेषक: -

REGISTRATION NO: BG0119010

पदव्युत्तर विद्यार्थी,
सामान्य औषध विभाग,
जेएनएमसी, बेळगावी.

मार्गदर्शन:-

डॉ. _____

सहयोगी प्राध्यापक
सामान्य औषध विभाग,
जेएनएमसी, बेळगावी.

परिचय आणि उद्देश: –

सेप्सिसची ही एक क्लिनिकल गुंतागुंत आहे आणि या प्राणघातक अवस्थेच्या निदानासाठी आणि उपचारांमध्ये योग्य परिभाषित त्वरीत घटकांची उपस्थिती आणि त्वरित ओळख करणे अत्यंत महत्वाचे आहे.

प्रक्रिया:

आपण संशोधन अभ्यासाचा भाग होण्यास सहमत असल्यास, आपणास संबंधित इतिहास विचारला जाईल आणि संबंधित क्लिनिकल परीक्षा आणि तपासणीस पात्र केले जाईल. आवश्यक तपासणीसाठी आपल्याला रक्त आणि लघवीचे नमुने देखील द्यावे लागतील.

जोखीम आणि फायदे:

तपासणीसाठी आपल्या बाहेरून रक्त घेत असताना आपल्याला फक्त धोका आणि संभाव्य असुविधाची समस्या उद्भवू शकते. ज्या स्थानावरून रक्त ओढले आहे त्या जागेवर सूज, वेदना, लालसरपणा (क्वचितच घडते) होऊ शकते.

या तपासणीमुळे आपल्याला फायदा होणार नाही परंतु आपण या अभ्यासाचा भाग व्हाल जे भविष्यात इतरांना उपयुक्त ठरेल.

विकल्प:

या अभ्यासामध्ये भाग घेणे ऐच्छिक आहे. आपण या अभ्यासामध्ये भाग न घेणे निवडू शकता.

आपण भाग घेण्याचा निर्णय घेतल्यास आपण नंतर आपले मत बदलू आणि अभ्यासापासून दूर जाऊ शकता. आपल्या निर्णयामुळे आपल्याला प्राप्त झालेल्या वर्तमान किंवा भविष्यातील आरोग्य सेवा किंवा इतर सेवा बदलणार नाहीत. अभ्यास डॉक्टर किंवा प्रायोजक या अभ्यासात आपला सहभाग कधीही थांबवू शकतात. आपण अभ्यासामध्ये भाग न घेणे निवडल्यास, आपल्या अट असलेल्या रूग्णांसाठी तुम्हाला प्रमाणित उपचार मिळेल.

गोपनीयता आणि गोपनीयता :

या अभ्यासाच्या दरम्यान आपल्याबद्दल संकलित केलेली सर्व माहिती कायद्याद्वारे परवानगी असलेल्या मर्यादेपर्यंत गोपनीय ठेवली जाईल. कोड नंबर आपल्याला या संशोधन रेकॉर्डमध्ये ओळखतील. या अभ्यासाची माहिती प्रकाशित केली जाऊ शकते परंतु आपली ओळख कोणत्याही प्रकाशनात गोपनीय असेल.

संस्था / प्रायोजक यांचे धोरणः

या संशोधनास लागू होत नाही

सहभागासाठी आर्थिक प्रोत्साहनः

अभ्यासामध्ये भाग घेण्यासाठी आपल्याला कोणत्याही भेटवस्तू / प्रोत्साहन दिले जाणार नाहीत.

परिणाम प्रकाशित करण्यासाठी अधिकृतताः

अभ्यासाचा निकाल भाग म्हणून केएलई विद्यापीठ, बेळगाव येथे पाठविला जाईल एमडी पदवी, पुनरावलोकन आणि प्रकाशन पूर्ण करण्यासाठी आवश्यक

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

डॉ. _____
सहयोगी प्राध्यापक
सामान्य औषध विभाग,
जेएनएमसी, बेळगावी.

REGISTRATION NO: BG0119010

अन्वेषक,
पदव्युत्तर विद्यार्थी,
सामान्य औषध विभाग,
जेएनएमसी, बेळगावी.

डॉ. रूपा एम बेलाड एमडी
अध्यक्ष, महाविद्यालयीन नैतिक प्रबंध
संशोधन समिती जे एन एन मेडिकल. कॉलेज
नेहरू नगर, बेलागवी - 590010

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 स्वाक्षरीः .....

तारीख:

ठिकाणः

सूचित सहमति

शोध अध्ययन का शीर्षक: सेप्सिस के साथ रोगियों में मृत्यु दर और रुग्णता के पूर्वानुमान में एक प्रारंभिक मार्कर के रूप में हाइपरयुरिसीमिया

मुख्य जाँचकर्ता:-

REGISTRATION NO: BG0119010

स्नातकोत्तर छात्र,
सामान्य चिकित्सा विभाग,
जेएनएमसी, बेलगावी.

मार्गदर्शक:-

डॉ. _____

सह - प्राध्यापक
सामान्य चिकित्सा विभाग,
जेएनएमसी, बेलगावी.

परिचय और उद्देश्य: -

यह सेप्सिस की एक अच्छी तरह से मान्यता प्राप्त नैदानिक जटिलता है और इस घातक स्थिति के निदान और उपचार में अच्छी तरह से परिभाषित अवक्षेपण कारकों की उपस्थिति और शीघ्र पहचान अत्यंत महत्वपूर्ण है।

प्रक्रिया:

यदि आप अनुसंधान अध्ययन का हिस्सा बनने के लिए सहमत हैं, तो आपको प्रासंगिक इतिहास से पूछा जाएगा और प्रासंगिक नैदानिक परीक्षा और जांच के अधीन किया जाएगा। आवश्यक जांच के लिए आपको रक्त और मूत्र के नमूने भी देने होंगे।

जोखिम और लाभ:

जांच के लिए आपके हाथ से रक्त लेने के दौरान एकमात्र जोखिम और संभावित असुविधा हो सकती है। यह उस स्थान पर सूजन, दर्द, लालिमा (शायद ही कभी होता है) हो सकता है जहां से रक्त खींचा जाता है।
आप इन जांचों से लाभान्वित नहीं हो सकते हैं, लेकिन आप इस अध्ययन का हिस्सा होंगे जो भविष्य में दूसरों के लिए उपयोगी होने जा रहा है।

विकल्प:

इस अध्ययन में भाग लेना स्वैच्छिक है। आप इस अध्ययन में भाग नहीं लेने का विकल्प चुन सकते हैं।
यदि आप भाग लेने का निर्णय लेते हैं तो आप बाद में अपना विचार बदल सकते हैं और अध्ययन से हट सकते हैं। आपका निर्णय वर्तमान या भविष्य की स्वास्थ्य देखभाल या आपके द्वारा प्राप्त अन्य सेवाओं को नहीं बदलेगा। अध्ययन चिकित्सक या प्रायोजक किसी भी समय इस अध्ययन में

आपकी भागीदारी को रोक सकते हैं। यदि आप अध्ययन में भाग नहीं लेना चुनते हैं, तो आप अपनी स्थिति वाले रोगियों के लिए मानक उपचार प्राप्त करेंगे।

गोपनीयता और गोपनीयता :

इस अध्ययन के दौरान आपके बारे में एकत्र की गई सभी जानकारी को कानून द्वारा अनुमत सीमा तक गोपनीय रखा जाएगा। कोड नंबर इस शोध रिकॉर्ड में आपकी पहचान करेंगे। इस अध्ययन से जानकारी प्रकाशित हो सकती हा□लेकिन आपकी पहचान किसी भी प्रकाशन में गोपनीय रहेगी।

संस्थान / प्रायोजक की नीति:

इस शोध पर लागू नहीं होता ह□

भागीदारी के लिए वित्तीय प्रोत्साहन :

आपको अध्ययन में भाग लेने के लिए किसी भी उपहार / प्रोत्साहन का भुगतान नहीं किया जाएगा.

परिणाम प्रकाशित करने के लिए प्राधिकरण :

भाग के रूप में अध्ययन के परिणाम केएलई विश्वविद्यालय, बेलगाम को भेजे जाएंगे एमडी की डिग्री, समीक्षा और प्रकाशन के पूरा होने की आवश्यकता।

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

डॉ. _____

सह – प्राध्यापक

सामान्य चिकित्सा विभाग,
जेएनएमसी, बेलगावी.

REGISTRATION NO: BG0119010

अन्वेषक,
स्नातकोत्तर छात्र,
सामान्य चिकित्सा विभाग,
जेएनएमसी, बेलगावी.

डॉ. रूपा एम बेलाड, एमडी
अध्यक्ष, कॉलेज नैतिक शोध प्रबंध
अनुसंधान समिति जे एन मेडिकल कॉलेज
नेहरू नगर, बेलगावी - 590010

सहमति पत्र

मैं स्वेच्छा से नीचे हस्ताक्षर करके इस अध्ययन में भाग लेने के लिए सहमत हूं। मैं किसी भी समय वापस ले सकता हूं। मैं इस फॉर्म पर हस्ताक्षर करके अपने किसी भी कानूनी अधिकार को नहीं छोड़ रहा हूं। नीचे दिए गए

मेरे हस्ताक्षर से संकेत मिलता हकि मैंने इस सहमति फॉर्म को पढ़ा हया यह मेरे लिए पढ़ा गया हयह सहमति फॉर्म और उत्तर दिए गए प्रश्नों के उत्तर हैं

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

प्रतिभागी का नाम: .....

हस्ताक्षर / बाएं अंगूठे का निशान: ....।
प्रतिभागी का

कानूनी रूप से अधिकृत का नाम: .....
प्रतिनिधि / अभिभावक

हस्ताक्षर / बाएं अंगूठे का निशान: .....

साक्षी का नाम: ....।

हस्ताक्षर / बाएं अंगूठे का निशान: ....।

अन्वेषक का नाम और हस्ताक्षर: .....

दिनांक:

जगह :

ANNEXURE III

PROFORMA

CASE NO:

NAME:

AGE/SEX:

IP NO.:

ADDRESS:

OCCUPATION

COMPLAINTS AT PRESENTATION:

Past history:

Family history

Personal history

Treatment history

PHYSICAL EXAMINATION:

GENERAL CONDITION:

PALLOR- YES/NO

ICTERUS-YES/NO

LYMPHADENOPATHY-YES/NO

CYANOSIS- YES/NO

CLUBBING-YES/NO

EDEMA-YES/NO

VITALS:

TEMPERATURE

PULSE

RESPIRATORY RATE

BLOOD PRESSURE

SYSTEMIC EXAMINATION:

R. S.:

C.V.S.:

P.A.:

C.N.S.:

INVESTIGATIONS :

SERUM URIC ACID LEVELS

SERUM PCT

SERUM LDH

HSCRP

SERUM LACTATE

COMPLETE BLOOD PICTURE

BLOOD CULTURE AND SENSITIVITY

URINE CULTURES

ARTERIAL BLOOD GAS ANALYSIS

URINE ROUTINE AND MICROSCOPY

HBA1C

RENAL FUNCTION TESTS AND LIVER FUNCTION

s.no	IP.NO	Age	Sex	alcohol	tobacco	diabetes	hypertension	GPE Findings						PR	RR	BP	Serum Uric acid	Serum PCT	HB	PLT	TC	focus of infection	Blood culture	Urine culture	ABG	Urine routine	Urine Microscopy	Na	k	bicarb	Cl	Creat	TB	DB	OT	PT	ALP	total protien	Duration of stay in days
								Pallor	Icterus	Lymphaden opathy	Cyanosis	Clubbing	Edema																										
1	1052377	38	M	Y	N	N	Y	Y	Y	N	N	N	Y	95	25	90/60	8.4	2.95	9.3	35,000	15,300	5	No growth	No growth	1			152	3.69	16	121	1	16.44	14.81	126	37	144	5.4	8
2	1042851	65	F	N	N	Y	N	Y	N	Y	N	N	N	129	30	80/50	10.2	0.13	12.1	28000	20300	1	No growth	No growth	2			135	5.17	24	97	1	0.4	0.3	58	45	128	5.8	9
3	1042873	25	M	N	N	N	N	Y	N	N	N	N	N	109	28	100/60	2.6	0.44	8.7	114600	13600	4	No growth	No growth	3			133	5.29	27	96	0.7	0.4	0.2	733	48	172	6.5	5
4	1027458	35	M	Y	Y	N	N	Y	N	N	N	N	N	130	37	90/70	8.5	26.24	9.3	354000	7400	3	Acinetobactr baumani	Psedomonas aeruginoa	1			146	3.86	16	107	2.89	0.8	0.6	585	67	180	4.7	10
5	1042803	58	M	N	N	Y	Y	N	Y	N	N	N	Y	125	36	80/50	8.4	28.84	13.7	48000	21900	2	No growth	Klebsiella pneumoia	1			116	6.44	18	77	3.77	5.4	5.3	69	86	388	5.7	12
6	1025815	65	F	N	N	Y	N	Y	N	Y	N	N	Y	124	28	90/60	6.7	7.46	11.5	137000	23400	5	Burkholderia cepacia	Enerobacter cloacae	1			126	5.37	16	102	3.87	0.4	0.2	24	10	144	6.9	6
7	1040643	59	F	N	N	Y	Y	Y	Y	N	N	N	N	130	22	90/60	8.4	100	10.2	150000	12100	1	E.coli	E.coli	1			147	3.3	11	105	1.73	1.49	0.78	113	69	195	6.4	18
8	1020324	54	M	N	Y	N	Y	N	Y	N	N	N	N	140	38	70 systolic	11.2	65.14	15.4	104000	16600	2	MRSA	MRSA	1			134	5.27	12	95	3.27	4	3.72	119	79	200	6.5	15
9	1020304	74	M	N	N	Y	N	N	Y	N	N	N	Y	122	40	80/60	4.2	36.23	14.7	73000	14400	4	VRSA	E.coli	1			149	5.11	21	98	3.1	7.6	6.7	119	107	316	6.0	5
10	1021036	63	M	Y	Y	N	Y	Y	N	Y	N	N	N	118	26	90/70	3.9	15.27	9.2	351000	28200	1	No growth	No growth	4			131	3.94	16	98	1.17	0.3	0.4	130	110	184	4.0	4
11	1021519	54	F	N	N	Y	N	Y	N	N	N	N	N	122	40	80/50	6.5	6.45	9.7	285000	24200	2	No growth	No growth	1			133	3.86	12	93	3.14	0.5	0.4	22	10	241	5.6	6
12	1053076	29	M	N	Y	N	N	Y	N	N	N	N	N	96	28	100/70	11	86	7.2	113000	8600	2	Klebsiella pneumoniae	E.coli	1			131	6.35	7	94	23.87	0.3	0.4	28	22	188	5.6	12
13	1052944	63	M	N	Y	N	N	N	N	N	N	N	Y	110	42	90/70	10.2	33.19	11.5	68000	23400	2	No growth	No growth	1			139	3.65	14	97	5.82	1.29	0.9	53	23	72	6.2	10
14	1021621	75	F	N	N	Y	N	Y	N	N	N	N	N	118	27	90/70	6.7	59.90	7.1	25000	10100	3	Klebsiella pneumoniae	No growth	1			140	4.00	19	105	1.94	0.4	0.6	15	20	107	3.7	8
15	1020382	42	M	Y	N	N	N	N	N	N	N	N	Y	110	26	90/60	7.2	10.54	13.5	119000	24300	5	No growth	No growth	1			128	4.3	14	89	9	0.6	0.7	17	20	244	5.6	7
16	1043380	60	M	N	N	Y	Y	Y	Y	N	N	N	N	130	36	90/60	5.5	100	10.6	171000	32200	2	No growth	Enerobacter cloacae	3			132	4.4	10	105	7.5	1.3	1.2	33	70	180	5.2	12
17	1043213	88	M	N	N	Y	N	Y	Y	Y	N	N	N	116	28	80/50	8.0	66.2	9.1	52000	9400	4	E.coli	E.coli	4			125	5.22	12	100	5.15	6.32	5.15	28	17	98	4.5	16
18	1042635	76	M	Y	N	Y	N	Y	Y	N	N	N	N	110	24	100/70	6.5	1.49	14	94000	15000	5	No growth	No growth	4			134	4.67	22	98	2.16	1.48	1	36	24	164	6.9	5
19	1043022	51	M	N	N	N	Y	Y	N	N	N	N	Y	116	23	90/60	8.5	28.9	7.1	371000	18200	4	Klebsiella pneumoniae	No growth	1			141	3.02	20	100	0.7	0.7	0.4	21	8	113	5.3	9
20	1053054	55	F	N	N	Y	N	Y	N	N	N	N	N	100	26	100/60	8.5	96	10.4	518000	21200	5	No growth	Candida species	1			131	4.61	14	100	2.27	0.4	0.1	26	24	153	5.6	13
21	1015959	35	F	N	N	N	N	N	Y	N	N	N	N	130	35	90/60	9.1	100	11.3	98000	8900	5	No growth	No growth	1			143	4.41	10	105	5.3	2.4	1.6	77	31	62	56	11
22	1037360	86	M	N	N	Y	Y	N	N	N	N	N	Y	122	26	90/60	11.2	80	13.5	106000	7700	5	No growth	No growth	1			141	6.2	14	110	6.32	0.8	0.5	57	26	164	7.3	10
23	1027233	18	M	Y	N	N	N	Y	N	N	N	N	N	130	42	80/50	4.1	100	8.0	209000	23800	4	Pseudomonas aeruginosa	No growth	1			136	5.2	10	96	2.2	0.8	0.7	112	86	114	6.2	7
24	1027124	57	M	Y	N	Y	Y	Y	Y	N	N	N	Y	114	24	90/70	13.1	26.6	9.4	30000	17000	2	Klebsiella pneumoniae	No growth	1			116	7.13	10	76	7.29	1.4	0.7	29	16	281	5.8	6
25	991629	57	M	N	N	N	Y	Y	N	Y	N	N	N	120	24	90/70	9.5	25	9.5	130000	17300	5	No growth	Mixed flora	1			134	4.6	16	101	1.74	0.4	0.3	112	45	98	3.8	8
26	1024664	21	M	N	N	N	N	N	N	N	N	N	N	98	25	100/60	4.6	5	17.1	183000	13600	2	No growth	Acinetobactr baumani	4			136	4.2	19	101	2.36	4.75	1.23	69	57	108	5.2	6
27	1031237	72	F	N	N	Y	N	N	N	N	N	N	Y	110	22	90/60	11.8	4.91	11.1	156000	17300	1	No growth	No growth	3			134	7.15	10	101	0.5	0.2	0.89	17	26	75	6.5	9
28	1030653	63	M	N	N	N	Y	Y	Y	N	N	N	N	130	30	90/60	7.0	80	8.6	25000	26500	2	Cogulase negative staphylococcus	E.coli	1			126	4.16	12	95	3.68	2.16	2.08	42	15	178	4.8	6
29	1035140	72	F	N	N	N	Y	Y	N	N	N	N	N	110	24	90/60	6.5	32	7.4	222000	10800	3	No growth	No growth	5			133	5.6	14	109	5.1	0.1	0.05	15	14	134	6.0	9
30	1053143	67	F	N	N	N	N	Y	N	N	N	N	N	92	28	100/60	8.9	28.8	7.3	196000	11600	4	No growth	CONS	1			126	4.5	16	102	1.8	0.5	0.4	45	24	26	4.8	7
31	1027521	72	F	N	N	Y	Y	N	N	N	N	N	N	130	30	90/60	6.0	46.71	10.2	197000	17000	2	No growth	No growth	5			117	6.32	13	86	4.78	0.7	0.4	24	24	66	5.4	14
32	1027459	70	F	N	N	Y	N	Y	N	N	N	N	N	110	27	100/50	6.7	12.61	9.3	277000	35600	2	E.coli	E.coli	1			136	8.4	9	97	17.69	0.4	0.2	18	16	137	6.8	8
33	1028215	70	M	Y	Y	Y	N	N	N	N	N	N	N	120	31	100/60	5.0	7.6	12	245000	12000	5	No growth	No growth	1			165	3.6	19	127	5.67	0.6	0.24	121	41	90	5.6	5
34	1034399	64	M	Y	Y	N	Y	Y	N	N	N	N	N	130	40	90/60	7.1	100	7.4	317000	9400	2	No growth	Enerobacter Species	5			136	5.8	10	104	14.5	0.3	0.2	21	18	68	5.8	11
35	747376	65	F	N	N	N	Y	Y	N	Y	N	N	N	118	28	90/70	8.6	2.34	9.3	321000	11500	5	No growth	No growth	1			143	4.5	15	108	2.35	0.3	0.2	44	32	112	5.5	8
36	962551	56	M	Y	Y	Y	Y	N	N	N	N	N	N	141	28	80/50	13.8	60.72	12.8	212000	9100	4	E.coli	Enerobacter cloacae	4			142	3.9	21	104	0.87	0.9	0.3	24	11	63	5.1	14
37	1033415	73	F	N	N	Y	Y	N	N	N	N	N	N	110	25	100/60	9.0	15	10.3	263000	19900	5	No growth	No growth	3			135	4.3	20	100	1.97	0.5	0.3	33	25	78	4.2	10
38	1035217	65	M	N	Y	N	N	N	Y	N	N	N	N	118	30	80/50	12.5	14.8	11.2	30000	20800	3	No growth	No growth	1			137	3.5	22	95	2.12	1.2	1.1	35	31	108	6.5	9
39	1035564	40	M	Y	Y	N	N	N	N	N	N	N	N	108	25	100/50	14.4	18	14.4	382000	14100	4	No growth	Psedomonas aeruginoa	5			138	4.4	17	101	1.71	0.3	0.1	31	27	63	7.3	10
40	1035554	87	M	N	N	Y	Y	N	N	N	N	N	N	130	34	80/60	6.0	3.95	11.5	178000	21100	2	No growth	No growth	1			124	5.2	10	100	3.4	0.2	0.1	15	14	66	6.4	7
41	1032381	26	M	N	N	N	N	Y	N	N	N	N	N	130	28	90/60	7.7	28	8.2	285000	8400	3	No growth	No growth	1			143	5.29	10	113	18.01	0.3	0.1	112	13	110	5.5	9
42	1032014	75	M	N	N	Y	Y	N	N	Y	N	N	N	110	24	100/60	9.0	18	10.8	290000	10000	1	No growth	E.coli	5			137	4.3	16	101	1.43	0.8	0.4	59	30	98	5.0	12
43	1032469	48	M	Y	Y	N	N	N	N	N	N	N	N	122	24	80/50	7.9	16.4	14.1	51000	22800	5	No growth	No growth	4			130	5.99	10	96	4.12	2.2	1.2	69	42	243	5.9	8
44	1030745	57	M	Y	Y	Y	Y	Y	N	N	N	N	N	140	36	90/60	11.2	24	8.4	177000	2000	4	Staph Haemolyticus	No growth	5			138	3.19	35	32	1.13	1.01	0.4	35	32	56	6.4	5
45	1037360	86	M	Y	N	Y	N	N	N	N	N	N	N	108	25	90/60	11.2	36	13.5	106000	7700	1	Burkholderia cepacia	No growth	3			141	6.23	14	110	6.32	0.8	0.5	57	26	164	7.3	7
46	1029186	62	F	N	N	N	Y	Y	N	N	N	N	N	110	24	100/60	7.4	24	6.8	351000	13400	5	No growth	Mixed flora	5			136	5.7	22	102	2.1	0.5	0.4	9	4	56	6.9	5

s.no	IP.NO	Age	Sex	alcohol	tobacco	diabetes	hypertension	GPE Findings						PR	RR	BP	Serum Uric acid	Serum PCT	HB	PLT	TC	focus of infection	Blood culture	Urine culture	ABG	Urine routine	Urine Microscopy	Na	k	bicarb	Cl	Creat	TB	DB	OT	PT	ALP	total protien	Duration of stay in days
								Pallor	Icterus	Lymphaden opathy	Cyanosis	Clubbing	Edema																										
47	1040929	60	M	N	Y	N	N	N	Y	N	N	N	N	126	28	90/60	9.1	100	11.3	98000	8900	2	staph aureus	pseudomons	1			143	4.41	10	105	5.3	2.4	1.6	77	31	62	5.6	11
48	1015305	72	M	N	N	Y	Y	Y	N	N	N	N	Y	130	26	100/60	7.3	196	10.3	196000	11600	1	No growth	E.coli	5			138	4.4	12	100	2.2	0.3	0.1	23	15	112	5.7	8
49	1041561	42	M	Y	N	N	N	Y	Y	N	N	N	N	110	25	100/60	6.0	30	4.7	150000	7800	3	E.coli	E.coli	5			135	3.8	15	111	1.34	0.4	0.2	22	12	80	5.1	6
50	1034973	53	M	N	N	Y	N	N	N	N	N	N	N	145	36	90/60	6.3	100	16.4	206000	9000	2	E.coli	E.coli	1			138	3.7	22	101	1.8	1.4	0.5	22	10	72	7.5	9
51	1032905	73	M	Y	Y	Y	N	Y	N	N	N	N	N	130	27	90/60	6.2	10	10.4	84000	6600	4	Streptococcus agalactiae	Enerobacter cloacae	1			125	4.4	13	96	3.8	0.6	0.5	19	33	86	4.7	6
52	1051557	45	F	Y	Y	N	Y	N	N	N	N	N	N	140	36	80/50	7.1	27	7.5	75000	9100	2	No growth	E.coli	1			130	3.5	16	98	1.5	0.1	0.1	19	18	205	6.1	10
53	1045945	68	M	N	N	Y	Y	Y	Y	N	N	N	Y	135	26	80/50	3.2	57.6	8.9	189000	11900	5	No growth	No growth	5			129	3.4	18	89	1.3	1.2	1.2	51	46	128	5.9	8
54	1045396	77	M	N	N	N	N	N	N	N	N	N	N	120	28	90/60	12.2	100	10.1	196000	17800	2	No growth	MRSA	1			137	4.2	20	100	1.78	0.5	0.3	34	17	40	5.5	10
55	1051000	20	M	N	N	N	N	N	N	Y	N	N	N	140	28	90/70	10.3	20.4	13.8	199000	7500	5	No growth	No growth	1			135	3.9	19	101	6.01	0.3	0.2	102	33	61	6.2	7
56	1008719	69	M	Y	Y	N	Y	Y	N	N	N	N	Y	130	24	80/60	11.3	59.98	9.9	50000	12300	4	Cogulase negative staphylococcus	Klebsiella pneumoia	1			140	6.2	12	96	3.8	0.2	0.3	20	18	84	4.9	12
57	1011440	58	F	N	N	Y	N	Y	N	N	N	N	N	132	27	100/50	10.2	46	6.0	288000	3800	5	No growth	No growth	1			136	4.8	10	100	0.8	0.2	0.1	17	10	233	8.3	6
58	1023800	55	F	N	N	Y	Y	N	N	N	N	N	N	122	26	90/60	9.3	44	10.6	72000	24000	2	E.coli	E.coli	1			131	3.3	10	93	5.5	0.3	0.2	31	31	146	7.1	8
59	1019224	38	M	Y	Y	N	N	Y	N	N	N	N	Y	140	26	80/50	10.1	96	8.5	76000	18000	4	Klebsiella pneumoniae	No growth	1			135	4.1	10	84	2.4	1.8	1.2	460	320	112	7.3	8
60	1026792	40	M	N	Y	Y	N	Y	N	N	N	N	N	136	28	90/60	10.5	84	12.8	113000	5800	5	No growth	No growth	2			117	3.9	16	96	3.0	4.2	2.4	126	110	98	6.6	11
61	1033107	47	M	Y	Y	N	N	N	Y	N	N	N	N	128	32	90/70	8.3	27.9	14.7	27000	18000	4	Pseudomonas species	Acinetobactr baumani	1			132	3.3	12	100	1.89	2.5	1.8	220	146	112	5.8	8
62	1055332	46	M	Y	N	N	N	Y	Y	N	N	N	N	130	40	80/50	9.8	17.23	9.3	119000	39000	3			1			120	5.7	10	86	5.28	10.3	9.4	135	43	147	6.4	5
63	1055028	67	M	Y	Y	N	N	Y	N	Y	N	N	N	120	26	90/60	13.3	4.85	7.1	59000	278000	1	No growth	No growth	3			134	5.9	17	104	2.6	0.8	0.4	15	10	164	5.5	6
64	1055134	26	F	N	N	N	N	Y	Y	N	N	N	N	122	28	90/70	5.0	100	7.1	10000	41000	5	No growth		1			143	3.5	18	110	1.1	34.6	28.78	182	101	118	4.4	5
65	1054659	43	M	Y	Y	N	N	Y	N	N	N	N	N	130	32	100/70	12.4	1.19	2.7	57000	5300	4	No growth	No growth	1			135	3.69	15	104	2.18	0.5	0.2	46	25	125	5.6	6
66	1054658	86	M	N	N	Y	Y	N	N	N	N	N	N	133	32	90/60	6.8	1.74	12	201000	24600	2	E.coli	E.coli	1			140	3.2	22	100	1.34	0.3	0.1	35	45	73	6.2	6
67	1019376	87	F	N	N	N	Y	Y	N	N	N	N	N	120	30	90/70	8.3	9.2	9.2	75000	10700	2	No growth	Enterobacter aerogens	2			134	4.6	15	94	1.32	1.2	0.54	15	6	48	6.0	7
68	1019619	56	M	N	Y	N	N	Y	Y	Y	N	N	Y	134	32	90/70	8.6	27	6.8	12000	19600	4	MRSA	enterococcus feacalis	1			137	4.2	13	98	3.94	16.9	14.5	63	60	293	4.7	9
69	1026324	61	M	Y	Y	N	Y	N	N	N	N	N	N	126	28	90/60	9.4	1.05	8.6	57000	9800	2	No growth	E.coli	1			131	6.4	12	95	0.8	0.4	0.2	16	18	93	7.0	8
70	1031982	30	M	Y	Y	N	N	Y	N	N	N	N	N	142	38	80/50	6.8	46	8.1	118000	10900	2	No growth	Acinetobactr baumani	1			131	5.34	15	97	5.4	0.4	0.2	19	10	50	6.5	12
71	1032020	82	F	N	N	Y	N	N	Y	N	N	N	N	130	27	90/60	7.5	36	10.9	28000	15800	1	No growth	E.coli	1			113	3.5	22	77	0.9	0.5	0.2	18	11	117	6.6	8
72	1041799	81	M	N	N	Y	Y	N	N	N	N	N	N	136	28	90/60	7.5	62	10.8	172000	17800	1	No growth	No growth	1			138	4.5	17	100	2.6	0.8	0.3	41	15	173	4.3	6
73	1015730	31	F	N	N	N	N	N	Y	N	N	N	N	124	30	80/50	9.2	36	14.4	50000	22300	3	Staph Haemolyticus	No growth	2			134	4.1	11	97	0.7	1.2	0.6	694	624	52	6.5	8
74	1029523	67	M	Y	Y	Y	N	N	N	Y	N	N	N	134	32	90/60	13.8	5.6	17.8	308000	16700	5	No growth	No growth	1			155	6.2	16	112	1.1	0.6	0.1	25	26	113	6.7	7
75	1016440	56	M	N	Y	N	N	Y	N	N	N	N	N	126	28	80/50	6.9	8	13.3	58000	14400	4	Pseudomonas species	No growth	2			133	3.3	20	87	0.5	1.2	0.8	56	43	110	5.8	8
76	1040172	61	M	Y	N	N	N	N	N	N	N	N	N	122	26	90/60	3.4	10	8.2	220000	15700	2	No growth	No growth	1			137	3.79	22	101	0.2	0.1	0.1	15	18	68	6.8	7
77	1055551	56	M	Y	N	N	N	N	N	N	N	N	N	136	34	90/70	9.1	7.61	8.7	274000	13500	2	Cogulase negative staphylococcus	Citrobacter freundi	1			141	5.1	10	110	5.84	0.2	0.1	55	32	87	6.3	8
78	1051610	46	M	Y	Y	N	N	N	N	Y	N	N	N	124	28	80/50	7.2	12	15.8	240000	15000	4	No growth	No growth	1			151	4.16	20	111	0.81	0.4	0.2	22	24	66	7	7
79	1041876	72	M	N	Y	N	Y	N	N	N	N	N	N	144	38	80/50	12	86	15.4	118000	8900	1	No growth	Acinetobacter	1			143	4.1	22	110	0.9	0.4	0.2	52	73	77	6.8	10

dis	exp	exp	dis	exp	dis	dis	exp	exp	exp	dis	dis	exp	dis	exp	exp	exp	dis	dis	dis	dis	dis	exp	dis	exp	exp	exp	dis	dis	exp	dis	dis	exp	exp	dis	dis	exp	exp	dis	dis	dis	dis	exp	dis	exp	exp	exp	dis	dis	dis	exp	dis	dis	exp	dis	dis	exp	dis		**Outcome**

exp	dis	dis	dis	dis	pl	exp	exp	exp	dis	dis	exp	exp	pl	exp	exp	dis	exp	dis	exp	exp	pl	dis	exp	dis	dis	dis	dis	pl	pl	exp	dis	exp	exp		**Outcome**