"CORRELATION BETWEEN SERUM MAGNESIUM AND SEVERITY OF SEPSIS IN PATIENTS ADMITTED IN INTENSIVE CARE UNIT AT KLE DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI – A CROSS SECTIONAL BASED STUDY."

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

Date :

Place : Belagavi

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

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

KAHER
BELAGAVI
J.N. MEDICAL COLLEGE

Date :

Place : Belagavi

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Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

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Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BG0120016,
Postgraduate Student,
2020-21 Batch,
Department of General Medicine,
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE LETTER

K L E ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed to be University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
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/ \2. Date: 25/01/2021

To,

Dr. Raina N Deshlahra
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 **"CORRELATION BETWEEN SERUM MAGNESIUM AND SEVERITY OF SEPSIS IN PATIENTS ADMITTED IN INTENSIVE CARE UNIT AT KLE DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI- A CROSS SECTIONAL BASED STUDY"**, 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: Sepsis is a potentially fatal organ dysfunction produced by an exaggerate of our immune system to an infection, whereas septic shock is characterised by profound circulatory failure in the form of decreased vascular tone with some degree of hypovolemia and is linked with a higher risk of death. The present study was aimed to establish relationship between serum magnesium severity of sepsis in patients admitted in the intensive care units.

Material & Method: This cross sectional hospital based study included 100 patients to find the correlation between serum magnesium and severity of sepsis admitted in Intensive Care Unit at KLE Dr Prabhakar kore Hospital and medical research centre, Belagavi during study period. The subjects were categorized based on severity of sepsis on the basis of qSOFA score within 24 hours of ICU admission. Blood sample were taken to estimate Magnesium level at the time of ICU admission. All the data were collected in proforma and entered in excel sheet. A $p < 0.05$ was accepted as significant. Statistical analysis was performed using SPSS version 22.0 (IBM SPSS, US) software operating on windows 10.

Results: The results observed on analyzing the levels serum magnesium, there was prolonged hospital stay, ICU stay, inotrope support and ventilator support in patients of hypo magnesium group. There was positive correlation with hypo magnesium group with prolonged hospital stay, ICU stay, inotrope support, ventilator support and increased mortality.

Conclusion: Present study documented the significant correlation of hypomagnesium with severity of the sepsis and which predicts the duration of hospital stay, requirement of intensive care, inotrope support and mortality.

ABBREVATIONS

APACHE	ACUTE PHYSIOLOGY AND CHRONIC HEALTH EVALUATION
ATP	ADENOSINE TRIPHOSPHATE
CD	CELL DIFFERENTIATION ANTIGEN
CKD	CHRONIC KIDNEY DISEASE
CLD	CHRONIC LIVER DISEASE
CNS	CENTRAL NERVOUS SYSTEM
CT	COMPUTED TOMOGRAPHY
HMGB	HIGH MOBILITY GROUP BOX PROTEIN
HTN	HYPERTENSION
ICAM	INTRACELLULAR ADHESION MOLECULE
ICU	INTENSIVE CARE UNIT
MG	MAGNESIUM
MIF	MACROPHAGE MIGRATION INHIBITORY FACTOR
MODS	MULTI ORGAN DYSFUNCTION SYNDROME
MPM	MORTALITY PROBABILITY MODELS
NA	SODIUM
NEWS	NATIONAL EARLY WARNING SCORES

NO	NITRIC OXIDE
PVD	PERIPHERAL VASCULAR DISEASE
REMS	RAPID EMERGENCY MEDICINE SCORES
qSOFA	QUICK SEQUENTIAL ORAGN FAILURE ASSESSMENT
SIRS	SYSTEMIC INFLAMMATORY RESPONSE SYNDROME
SVC	SUPERIOR VENA CAVA
T2DM	TYPE 2 DIABETES MELLITUS
TNF	TUMOR NECROSIS FACTOR
US	UNITED STATES
WBC	WHITE BLOOD CELL

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INTRODUCTION

"Correlation Between Serum Magnesium and Severity of Sepsis in Patients admitted In Intensive Care Unit at KLE Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi – A Cross Sectional Based Study."

Sepsis is a potentially fatal dysfunction of multiple organ produced by an exaggerated host response to an infection, whereas Septic shock is characterised by sepsis with circulatory, metabolic and cellular abnormalities or failure in the form of decreased vascular tone with some degree of hypovolemia and has greater mortality when compared to sepsis alone. Magnesium, is generally recognised as "the forgotten electrolyte," is the 4^{th} most prevalent cation (positively charged ion) in the body and the 2^{nd} most rich intracellular cation (positively charged ion). In severely sick individuals, both high and low levels of magnesium have been seen. Hypomagnesium group can disrupt practically every organ and end in a deadly consequence (ventricular arrythmia, coronary vasospasm , even death).[1]

Over 300 enzyme systems are affected, including Na-K-ATPase-mediated transport, calcium homeostasis, nerve transmission, and skeletal muscle function. Magnesium deficiency causes systemic stress by activating the neuroendocrine pathway. Magnesium is a significant intracellular cation.[2]

Humans need a proper concentration of extracellular calcium and magnesium for appropriate neuromuscular function. Intracellular magnesium serves as a cofactor for several enzymes, transporters, and nucleic acids that are required for optimal cellular function, replication, and energy consumption.[1,3]

Several research have been conducted to investigate the association between serum magnesium and the severity of sepsis. However, the results of these investigations were conflicting, particularly in terms of serum magnesium levels and the severity of sepsis. Magnesium estimate is a low-cost, quick, and commonly performed inquiry in critically unwell patients. Magnesium metabolism disorders are among the most prevalent electrolyte disturbances in critically sick patients, and they are usually overlooked.

There are limited literature in the Indian population and also in the region of Belagavi district. Hence the present study was aimed to establish relationship between serum magnesium severity in patients of sepsis admitted in the ICU (intensive care unit) at Dr. Prabhakar Kore Hospital and MRC, Belagavi.

OBJECTIVES

Aim

To establish a proven relationship between serum magnesium and severity of sepsis in patients admitted in the intensive care units at Dr. Prabhakar Kore Hospital and MRC, Belagavi.

Objectives

- Measure the serum magnesium of all the patients with sepsis admitted to ICU (intensive care unit).
- Correlate the serum magnesium level with severity of sepsis in patients admitted to intensive care unit.

REVIEW OF LITERATURE

Sepsis is a clinical syndrome of dysfunction Ing of multiple organ caused by an dysregulated (exaggerated) host response by our immune system to an infection whereas, Septic shock is a type of vasodilatory or distributive shock that has circulatory, metabolic and cellular failure leading to decrease in vascular tone(low blood pressure) and hypovolemia which is associated with a higher risk of mortality.[4]

Systemic illness or dysfunction caused by invasion of micro-organism to a normally healthy and sterile tissue of the body is known as "sepsis." Bacterial sepsis(infection) is a potentially fatal illness that occurs as an exaggerated response of the body or host immune system to an infection which in turn leads to tissue and organ damage.[5] Sepsis which has lately been reclassified as life-threatening multiple organ dysfunction induced by an unbalanced host response or immune response to infection. Sepsis which was initially described as a 'medical illness' by Hippocrates (460–480 BC) which is derived from the Greek term sipsi, which means "to render rotten." Since then, this disease(sepsis) entity has gone through several incarnations, with advances in the late nineteenth century laying the groundwork for present knowledge of sepsis.[6]

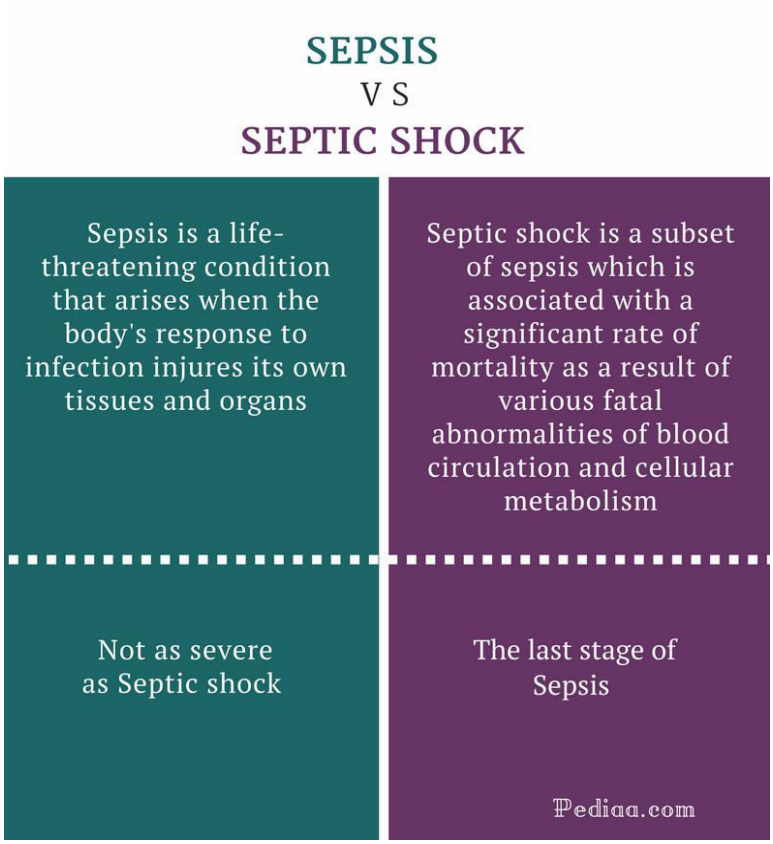
Incidence:

"It was estimated in the late 1970s that 164,000 cases of sepsis occurred in the United States (US) per year.[7] Since then, overall rates of sepsis have risen in the United States and elsewhere, but many of these figures are focused on research institutions or claims-based studies.[8–10]

Aging, immune-suppression, and multidrug-resistant infection are all potential causes of an elevated rate of sepsis.[9,11–14] "It may also be attributed to increased

detection of early sepsis as a result of vigorous sepsis education and awareness programs, but this is an unproven hypothesis.

Defining sepsis: Over the years, understanding the complex pathophysiology of sepsis has improved, and so has our ability to define sepsis. The word 'sepsis' is derived from the Greek word for "decomposition" or "decay," and its first use was about 2700 years ago in Homer's poems. It was then used by Hippocrates and Galen in later centuries.[15] In the 18 century, the "Germ theory" of disease came into light and there was some recognition that sepsis originated from harmful(bad) microorganisms. The 1st modern definition was tried in 1914 by Hugo Schottmüller who wrote that "sepsis is present if a focus has developed from which pathogenic bacteria, constantly or periodically, invade the blood stream in such a way that this causes subjective and objective symptoms". Over the years, numerous experimental and clinical trials were done which were able to demonstrate the role of the host immune response as the causative factor of sepsis. However, due to diversity of the disease process, it was difficult in recognizing, evaluating, treating, and studying sepsis.[16]"Sepsis and septic shock are defined as":



The advent of antiseptics, the Germ theory of illness, and bacteriology which led to the commonly accepted idea that Sepsis is a multi system infection caused by harmful organisms infecting the host and spreading via the bloodstream leading to septicemia. It wasn't until the extensive use of antibiotics and the discovery of endotoxins that it became clear that the pathophysiology of sepsis was significantly more complicated.[15,17]

"An International consensus meeting held in 1991, generated and defined terms, such as Sepsis, Systemic Inflammatory Response Syndrome (SIRS), and septic shock (known now as Sepsis-1). SIRS describes as the widespread inflammatory process, independent of cause based on a combination of vital signs and blood work".

SIRS includes ≥2 of the following":

- Temperature >38˚ C or < 36˚ C
- Heart rate >90 beats per minute
- Tachypnea >20 breaths per minute or PaCO2 <32 mm Hg
- White blood cell (WBC) count >11,000 cmm and <4000 cmm, or >10% immature (band) forms[18]
- SIRS as a result of an infection.
- Sepsis associated with multi organ dysfunction, hypo-perfusion, or systemic hypotension.
- Hypo-perfusion of the organs may include but are not limited to oliguria, lactic acidosis, or acute change in mental status.
- Septic shock is defined as Sepsis-induced hypotension despite adequate fluid resuscitation and require vasopressors.

The presence of progressive organ dysfunction in an acutely ill patient, such that its homeostasis cannot be maintained without any intervention is referred to as multi-organ dysfunction syndrome (MODS).

Sepsis-3 defines Sepsis as a potentially fatal multi organ malfunction produced by an unbalanced host response to infection(septicaemia). It's crucial to emphasise that not all the patients with SIRS have an infection(septicaemia), and not all patients with infections are septic. An exaggerated host reaction or response and the presence of end-organ failure distinguish sepsis from infection.[19] Sepsis with its complications comprise a spectrum of pathophysiologic and clinical severity, culminating in increasing physiologic collapse of numerous interconnected organ systems.[20,21]

Sepsis has substantial morbidity and death and is now estimated to impact about 1.7 million people in United States each year. Prior to the year 2000, fatality

rates in patients with acute sepsis and septic shock were as high as 50%.[22] "Even with modern technology and therapies, national death rate remains between 20% to 25%, along with 1 in 3 patients dying in the hospital due to sepsis". "With the documented, rising frequency of sepsis, a high priority has been put on detecting and treating this disease process(sepsis) early, with the most current budget turned up at roughly $23.7 billion in 2013". Overall mortality from sepsis syndromes can range from 30% to 50% depending on demographic characteristics such as age, race, gender, co-morbid diseases, and organ failure.[23]

Risk factors predisposing to sepsis or septic shock-

- Diabetes (type 1 and 2)
- Burns
- Malignancy(cancer)
- CKD (Chronic kidney disease)
- Liver disease
- Immunosuppressed state
- Use of steroids
- Surgery
- Prolonged hospitalization
- Trauma/accident
- Hemodialysis
- Elderly patients

Pathophysiology-

Sepsis refers to a condition that progresses as a pathophysiologic spectrum, beginning with a SIRS (systemic inflammatory response syndrome) and culminating with MODS (multiorgan dysfunction syndrome before death).[24–27]

The following are the first indications of inflammation;

- Fever (>38 degrees Celsius) or hypothermia (< 36º C)
- Tachycardia (> 90 beats per minute), Tachypnea (> 20 breaths per minute)
- With or without bandemia, leukocytosis (WBC >11,000/cu mm) or leukopenia (WBC< 4,000/cu mm) (more than 10 percent).

"Two out of four clinical symptoms are required for the diagnosis of SIRS (systemic inflammatory response syndrome). Following that, the clinical definition of sepsis is systemic inflammatory response syndrome with an infectious cause. As hypotension sets in, tissue demands are insufficiently supplied by tissue and organ oxygenation, and then patient is classified as having severe sepsis".[23,28]

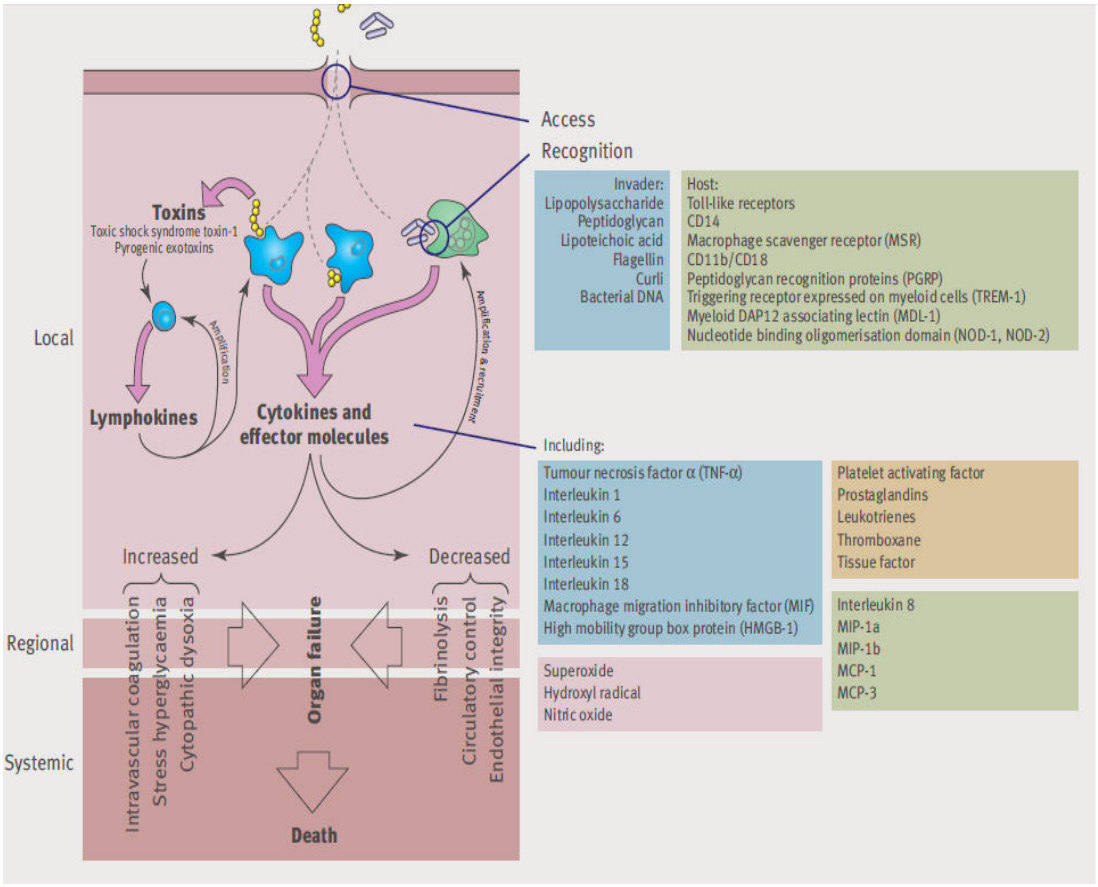


Figure 1: Pathophysiological pathway of sepsis[29]

The decrease in peripheral arterial perfusion (poor circulation) and oxygenation causes metabolic and cellular and disruptions, which leads to shift from aerobic to anaerobic metabolism leading to lactic acidosis. “Features of end-organ injury, such as transaminitis or pre-renal azotemia, can also indicate tissue hypoperfusion. During resuscitation, the imbalance in oxygen demand and supply can be monitored by trending the mixed venous oxygen (O2) saturation from a central line in the SVC (superior vena cava), if it is available”.[23,28]

Septic shock differs from other shock states in that it is a distributive form of shock. A mixture of inflammatory markers (serotonin, histamine, lysosomal enzymes and super-radicals) produced in response to bacterial endotoxins (gram negative bacteria) increases capillary permeability while decreasing peripheral vascular resistance. Decrease in venous return from third-spacing results in decrease in both

afterload and preload. The consequent drop in SV (stroke volume) is first accommodated by an increase in HR (heart rate) i.e., compensated Septic shock. Resulting in the hyperdynamic condition associated with septic shock.

"Septic shock is defined functionally as prolonged hypotension despite sufficient fluid resuscitation of 60 mL/kg - 80 mL/kg of crystalloid or colloid fluid. At this stage, starting adequate vasoactive medicines like beta-adrenergic or alpha-adrenergic agents is critical.[30–32] The development of organ dysfunction despite high-dose vasoactive treatment characterises the condition known as multi-organ dysfunction syndrome (MODS), which has a death rate of up to 75%. While determining the precise circumstances indicating poor prognosis and mortality has proven challenging, immunologic dissonance (exaggerated pro-inflammatory response) vs immunologic paralysis (exaggerated anti-inflammatory response) has been proposed to play a role".[33]

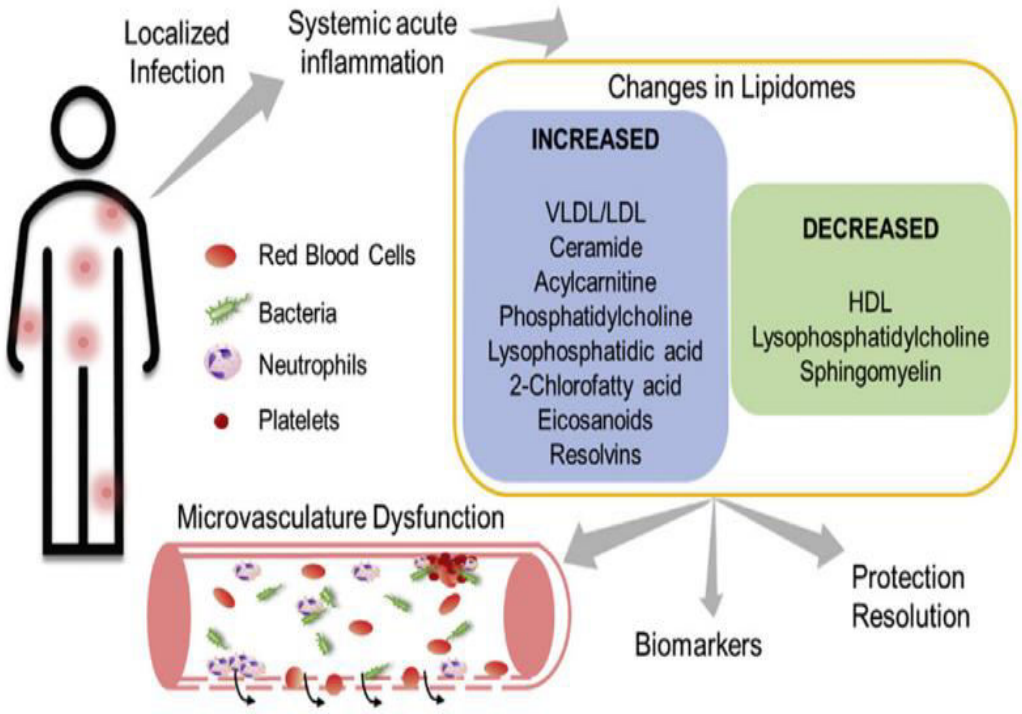


Figure 2: Sepsis and lipid changes[34]

Early signs and symptoms

Sepsis is defined as systemic inflammatory response syndrome plus an infectious source. Therefore, earlier on in the presentation of sepsis, patients present with the following vital sign changes":

- Fever
- Tachycardia
- Tachypnea

Signs and Symptoms of Severe Sepsis, is combined with end organ dysfunction

- Altered mental function
- Cyanosis
- Hypoxia
- Oliguria /anuria
- Paralytic Ileus

Patients who develop to septic shock will exhibit signs and symptoms of severe sepsis, including hypotension. Notably, blood pressure may be maintained at an early "compensated" stage of shock, and other indications of distributive shock, such as warm extremities, rapid capillary refill (less than one second), and bounding pulses, often known as warm shock, may be present. This stage of shock can be reversed if treated quickly with fluid resuscitation and vasoactive support.[23] Hypotension develops when septic shock progresses into the uncompensated stage, and patients may appear with chilled extremities, delayed capillary refill (greater than three seconds), and thready pulses, sometimes known as cold shock. With persistent tissue hypoperfusion, shock may become irreversible, swiftly progressing into multiorgan failure syndrome and death".[23]

Evaluation

Laboratory findings

The laboratory findings may show the following

- Hyperglycemia (glucose >120 mg/dL)
- Leukocytosis (WBC >11,000/mm3) or leukopenia (WBC <4000/mm3)
- Bandemia (>10%)
- C-reactive protein-CRP or Procalcitonin >2 SD above normal
- Mixed venous saturation >70%
- PaO2: FiO2 <300
- Azotemia(pre-renal)
- Coagulopathy, INR >1.5 or PTT >60 sec
- Thrombocytopenia (platelets <100,000/mL)
- Hyperbilirubinemia (total bilirubin >4 mg/dL)
- Lactic acidosis (>2 mmol/L)

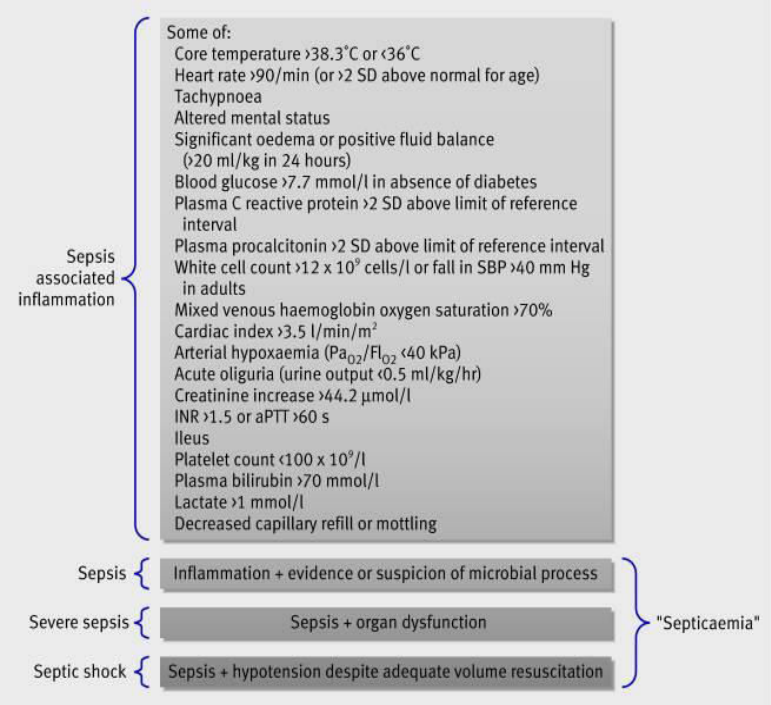


Figure 3: Definition of sepsis as severe and septic shock[29]

"Patients should be placed on continuous cardiopulmonary monitoring so that vital signs may be closely monitored. End-organ function and peripheral perfusion should be thoroughly evaluated to see where they may lie on the pathophysiologic continuum of sepsis. A Glasgow Coma Scale or mental state evaluation, lactate/mixed venous saturation test or urine output measurement should all be included (with central lines). An x-ray of the chest may show symptoms of ARDS or pneumonia. If the patient develops necrotizing fasciitis, plain x-rays of the extremities may indicate the presence of gas in the tissues". The gallbladder may be evaluated using ultrasound. A CT scan is performed to look for abscesses, intestinal perforation, or ischemia in the belly.[23]

Biomarkers - diagnosis of sepsis[29]

Total Cell counts

- Total leucocyte count- TLC
- Platelet count
- Neutrophil count
- Lymphocyte count

Leucocyte surface markers-

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

Peptides

Monocyte / macrophages

- TNF-α (Tumor necrosis factor α)

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

Leucocyte product

- Soluble L-selectin
- Soluble P-selectin

"Acute phase reactants

- Ferritin
- CRP (C-reactive protein)
- Lactoferrin
- Neopterin
- Procalcitonin (PCT)
- Serum amyloid A"

Differential diagnosis of sepsis

- ARDS (acute respiratory distress syndrome)
- Distributive/ vasodilatory shock
- DIC (disseminated intravascular coagulation)
- Hemorrhagic shock
- Cardiogenic shock
- Adrenal crisis
- Drug toxicity
- Toxic shock syndrome (TSS)

Complications

- Acute or chronic renal injury
- Acute liver failure/fulminant
- ARDS
- DIC
- Myocardial dysfunction/infarction
- Multiple organ failure (MODS)

Scoring System and Its Evolution

" Patients referred to an ED represent a broad spectrum of disease severity. In the interest of allocating resources to those who might potentially benefit most from clinical interventions, several scoring systems have been proposed as a triaging tool. McClish et al has shown that in a critical care environment, physicians outperform scoring systems when assessing groups of patients at the extremes of risk of deterioration. Patients doing very poorly or very well are easily identified, but when assessing the in-between group scoring, systems were better than clinical experience. Apart from the assessment of patient risk, scoring systems can be used in clinical trials to account for the severity of disease in the subjects included in the trial or to adjust for case-mix when benchmarking the performance of clinical units. Finally, they can be used to monitor the effect of new technology. Most systems are not developed to be used on an individual level but on groups of patients. The development of scoring systems began in the intensive care environment (ICU). Systems such as Acute Physiology and Chronic Health Evaluation (APACHE), the Mortality Probability Models (MPM) and Sequential Organ Failure Assessment (SOFA) scores were developed and validated in ICU's. Later, the Emergency

Medicine community caught on and scoring systems for this environment were developed".[35]

qSOFA (quick sequential organ failure assessment)

"The qSOFA score (also known as quick SOFA) is a bedside prompt that may identify patients with suspected infection who are at greater risk for a poor outcome outside the intensive care unit (ICU). 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). The score ranges from 0 to 3 points. The presence of 2 or more qSOFA points near the onset of infection was associated with a greater risk of death or prolonged intensive care unit stay".

National Early Warning Score (NEWS)

"The National Early Warning Score (NEWS) was first introduced by the Royal College of Physicians in 2012 as a predictor of patient deterioration. However, it was not specifically designed for septic patients. It includes seven parameters (temperature, systolic blood pressure, respiratory rate, oxygen saturation, oxygen supply, heart rate, and level of consciousness) as shown in the table. The score range is from 0 to 20. Patients are classified as a low score (NEWS 1–4), medium score

(NEWS of 5–6) and high score (NEWS≥7) (11). NEWS is a sensitive screening tool in the ED for predicting sepsis-related outcomes".[36]

Table 1 National Early Warning Score (NEWS)

Physiological Parameters	3	2	1	0	1	2	3
Respiration Rate (BPM)	≤8		9–11	12–20		21–24	≥25
Oxygen Saturation (%)	≤91	92–93	94–95	≥96			
Any Supplemental Oxygen		Yes		No			
Temperature	≤35		35.1–36.0	36.1–38.0	38.1–39.0	≥39.1	
Systolic Blood Pressure (mmHg)	≤90	19–100	101–110	111–219			≥220
Heart Rate (BPM)	≤40		41–50	51–90	91–110	111–130	≥131
Level of Consciousness				Alert			U, P or V*

Note: *Unresponsive, react to pain, or loud voice.

Rapid Emergency Medicine Score (REMS)

The Rapid Emergency Medicine Score (REMS), an attenuated version of APACHE II, allows for prompt calculation. REMS is a composite score consisting of the GCS, RR, oxygen saturation, MAP, hazard ratio and age. Among non-surgical patients who present to the ED, REMS has proven to be a valid predictor of mortality.[37]

Table 1 REMS scoring system

	Score						
Variable	0	+1	+2	+3	+4	+5	+6
Age (years)	<45		45–54	55–64		65–74	>74
MAP (mm Hg)	70–109		110–129	130–159	>159		
			50–69		≤49		
Heart rate (bpm)	70–109		110–139	140–179	>179		
			55–69	40–54	≤39		
RR (breaths/min)	12–24	25–34	6–9	35–49	>49		
		10–11			≤5		
O_2 saturation (%)	>89	86–89		75–85	<75		
GCS	14 or 15	11–13	8–10	5–7	3 or 4		

GCS, Glasgow Coma Scale; MAP, mean arterial pressure; REMS, Rapid Emergency Medicine Score; RR, respiratory rate.

Magnesium (Mg2+):

"The fourth most abundant mineral in our body is magnesium. Around 99% of the total body magnesium (Fig 5) is present in the bone, muscle and the soft tissue. Magnesium ion is present inside the cells at a concentration of 5-20mmol/L; present in the ionized form in around 1-5%, remaining is bound to the proteins, and adenosine tri phosphate (ATP). During states of acute deficiency, large amount of exchangeable pool for magnesium is from the bones. As age increases, the magnesium content of bone also decreases. Only 1% of the total intracellular magnesium is present extracellularly. The primary extracellular concentration is present in the red blood cells (RBCs) and serum. Similar to calcium, magnesium is present in three forms free/ionized, bound to proteins and anions like phosphate, bicarbonate, citrate or sulphate. The biological activity of magnesium is greater with the ionized form".

Magnesium (Mg2+) Homeostasis

"Magnesium homeostasis is achieved mainly through the bones, intestines and the kidneys. It is absorbed mainly in the small intestine by the passive transport. Only 25 – 75% of the total magnesium consumed is absorbed. The remaining is excreted in the faeces. The absorption of magnesium in the intestines depends not on the levels consumed, but on its status of the body.

Lower the magnesium levels, higher the ion is absorbed. Serum magnesium levels are principally regulated by the kidneys. Around 95% of the filtered magnesium is reabsorbed and only 3 -5% is excreted in the urine. The thick ascending limb of the loop of Henle is the primary site for reabsorption".

Atherosclerosis is the most important risk factor for the cardiovascular diseases mainly stroke and myocardial infarction. Large amount of evidence supports that the main causative factor responsible for the atherosclerotic burden in

cardiovascular disease is the inflammation and the endothelial dysfunction. Various epidemiological studies from animal models suggest that magnesium deficiency at the cellular level accelerates the inflammation and intensifies the lipid deposition in the blood vessel wall and there is an inverse correlation cardiovascular incidence and the dietary magnesium. In the follow up of the ARIC study, it was demonstrated that for every 0.1mm decline in the thickness of carotid intima- media, there was increased risk of carotid plaques development".

Antithrombotic effect of magnesium

"The protective mechanisms of magnesium are

- Reduction of the proinflammatory process.
- Stabilization of the membrane of platelets as magnesium is needed to maintain the shape of the platelets.
- Inhibition of thrombogenesis through inhibition of ADP – platelet aggregation and adhesion.
- The release of the platelets is dependent on the calcium and so magnesium physiologically inhibits its release.
- The platelet dependant thrombosis was inversely correlated with the serum magnesium levels and its supplementation positively reduced the size and the number of platelet clumps and increased the number of discrete platelets.
- Increase in the fibrinolytic activity.
- By lowering the intracellular calcium concentrations, it decreases the tonicity of the blood vessels and also inhibits the vascular calcification.
- Against oxidative stress, it helps to increase the protective enzymes.
- Increases the nitric oxide (NO) release and enhances the endothelial dependant vasodilatation and inhibits the aggregation of platelets".

Various articles assessing the relation of serum magnesium with severity of sepsis;

Guerin et al conducted a prospective observational study, 179 ICU patients. On ICU admission, hypomagnesium group was observed in 79 patients (44%), hypermagnesium group in 10 (6%). High Mg level associated with higher mortality, no association between low Mg and mortality".[38]

"An observational study on 102 medical ICU patients by Reinhart et al showed that hypomagnesium group was present in 20% of patients, while hypermagnesium group was present in 9% of patients, and there were no other laboratory tests or clinical features suggesting hypomagnesium group. Of all ion levels measured routinely in these patients, serum magnesium had the highest prevalence of abnormal values".[39]

"Prospective observational study by Limaye et al., (2011) to assess the incidence of hypomagnesium group in critically ill patients. Study showed that, on ICU admission, 52% of patients had hypomagnesium group, 41% had normal serum Mg levels and 7% had hypermagnesium group. In this study, patients with hypomagnesium group had more frequent need for mechanical ventilator support (73% (38 of 52) vs. 53% (22 of 41), $P < 0.05$), longer duration of mechanical ventilation (4.27 ± 5.01 days vs. 2.15 ± 3.36 days, $P < 0.05$), increased incidence of sepsis (38% (20 of 52 patients) vs. 19% (eight of 41 patients), $P < 0.05$) and higher mortality (57.7% (30 of 52 patients) vs. 31.7% (13 of 41 patients), $P < 0.05$) compared to patients with normal Mg levels". The critically ill patients had a high prevalence of hypomagnesium group. In critically sick individuals, hypomagnesium group was linked to a greater fatality rate. Hypomagnesemia individuals required much more ventilatory assistance. Patients who were hypomagnesemia required ventilator support for a longer period of time. Sepsis and diabetes mellitus were frequently linked with hypomagnesium group. The duration of MICU hospitalisation and APACHE score on entry did not differ between

low magnesium and normal magnesium patients. Patients with hypocalcemia and hypoalbuminemia are more likely to have hypomagnesium group. [40]

In a single-center observational prospective study by Zafar et al., (2014) to assess the magnesium level in critically ill ICU patients. "The patients' ICU stay (P > 0.05), Acute Physiology and Chronic Health Evaluation-II (APACHE-II) rating (P = 0.34), and co-morbidity (P = 0.360) differed insignificantly across groups. Hypokalemia (58.82%), hyponatremia (47.05%), hypocalcemia (70.58%), and hypophosphatemia (29.41%) were associated electrolyte abnormalities in hypomagnesemia individuals. Around 76.47% of the hypomagnesemia group was on magnesium lowering medicines, while 46% of the normomagnesemic population was (P = 0.030)". The mortality rate in the hypomagnesemia group was 74.47%, while it was 36% in the normomagnesemic group. In critically ill patients, hypomagnesium group is a serious electrolyte imbalance. Individuals who are critically unwell and hypomagnesemia have a greater death rate than normomagnesemic patients.[41]

In a prospective Observational study by Kumar et al., (2015) to assess the magnesium levels in patients admitted to intensive care unit. "Over 1 year, 601 patient admitted in ICU, Hypomagnesium group in 25% of patients, associated with greater need for mechanical ventilation, longer ICU stand higher mortality (38.56% vs. 14.73%)". Approximately 25% of patients exhibited hypomagnesium group at the time of admission. There was a significant association between hypomagnesium group and outcome in terms of duration of MICU stay (5.46 (5.75) versus 3.93 (3.88), need for mechanical ventilation (56.86% vs. 24.33%), discharge/cured from ICU (61.43% vs. 85.26%), and death (38.56% vs. 14.73%) when compared to the normal Mg group. However, no significant variation in ventilation time was identified between the two groups. In critically sick individuals, hypomagnesium group is

associated with a greater fatality risk. In hypomagnesemic individuals, the demand for ventilatory assistance, but not its duration, is much greater. Sepsis and diabetes mellitus are frequently linked with hypomagnesium group. Patients with low serum Mg had a considerably longer MICU stay. [42]

In a meta-analysis study by UpalaS et al., (2016) to assess the hypomagnesium group and mortality in patients with sepsis in intensive care unit. The meta-analysis includes 6 studies with 1550 people from 30 full-text papers. With an RR of 1.90, critically sick individuals with hypomagnesium group had a statistically significant increased risk of death. The hypomagnesium group group also had an increased risk of needing mechanical breathing, with an RR of 1.65. The hypomagnesium group group also had a longer ICU stay, with a mean difference of 4.1 days. "According to the findings of this meta-analysis, hypomagnesium group is related with increased mortality, the requirement for mechanical ventilation, and the length of ICU stay among patients admitted to the ICU". [43]

"In a systematic review study by Jiang P et al., (2017) to assess the impact of hypomagnesium group in patients with illness. The patients with hypomagnesium group had higher mortality rate (risk ratio [RR] 1.76; 95% confidence interval [CI] 1.54-2.00; P <0.00001), more frequently had sepsis (RR 2.04; 95% CI 1.21-3.42; P = 0.007) and more frequent need for ventilatory support (RR 1.36; 95% CI 1.21 to 1.53; P <0.001). Length of ICU stay was also higher in the hypomagnesium group group (RR 1.85; 95% CI 0.43- 3.26; P = 0.01). Collectively, our data indicated that hypomagnesium group appears associated with greater risk of mortality, sepsis, mechanical ventilation, and the length of ICU stay in patients admitted to ICU. The role of magnesium therapy for improving outcomes in critically ill patients is needed to further study".[44]

"In a study by Khare AR et al., (2019) to assess the admission serum magnesium level in patients with septic shock and its correlation with outcome. In this study, hypomagnesium group was found in 18% of the patients, normomagnesium group in 62%, and hypermagnesium group in 20% of the patients. The mean vasopressor free days in the hypomagnesium group group (7.11±12.79 days) were greater than in the normomagnesemic (5.065±.51 days) and hypermagnesemic (1.70±3.09 days) groups. There were 18 deaths and 32 recoveries among the 50 patients. 11 of the 32 patients who recovered had aberrant entrance serum magnesium levels, while 8 of the 18 patients who died had abnormal admission serum magnesium levels. The SOFA score in hypomagnesemic patients hospitalised with septic shock was statistically significant when compared to normomagnesemic and hypermagnesemic individuals. Although SOFA score was greater in hypomagnesemic patients hospitalised with septic shock compared to normomagnesemic and hypermagnesemic patients, the author found no statistically significant link between admission magnesium levels in septic shock patients and prognosis. Serum magnesium levels may not accurately represent the body's magnesium status. RBC magnesium may need to be investigated further to see whether it is a more accurate biomarker". [45]

"In a study by Noormandi A et al., (2020) to assess the effect of magnesium supplementation on lactate clearance in critically ill patients with severe sepsis. On day 2 (27.53% vs. 23.79%, $p<0.001$) and day 3 (49.83% vs. 37.02%, $p<0.001$), the magnesium group had a considerably larger mean increase in lactate clearance than the placebo group. The magnesium group also had a considerably shorter time to lactate clearance than the placebo group (47.28 ±20.59 vs. 61.20 ±24.31 h, $p = 0.03$). Although there was no significant difference in sepsis-related mortality, the median

length of ICU stay was considerably shorter in the magnesium group than in the placebo group (8 vs. 15 days, p<0.01). In critically sick patients with severe sepsis, magnesium administration enhanced lactate elimination. Optimizing serum magnesium levels towards the upper limit of the normal range may improve the outcomes of severe sepsis". [46]

In a study by Murali PB et al., (2020) to assess the role of serum magnesium and its influence on the outcome in patients with sepsis. The study population's average age was 62.30 ±11.46 years. 63% were men and 37% were women. In the study population, 39%, 46%, and 15% of patients were hypomagnesaemic, normomagnesaemic, or hypermagnesaemic, respectively. The death rates for hypomagnesaemic, normomagnesaemic, and hypermagnesaemic patients were 76%, 18%, and 5%, respectively. The hypomagnesaemic group had significantly higher mortality and a higher SOFA score. Hypomagnesium group is frequent in people with sepsis. Serum magnesium levels must be monitored in sepsis patients to prevent significant symptoms of hypomagnesium group and thereby influence the eventual prognosis. [47]

In a retrospective cohort study by Chenwei LV et al., (2021) to assess the effect of hypomagnesium group on the prognosis of patients with sepsis in ICU. "At 28 days, 99 patients in the hypomagnesium group group (30.84%) and 123 patients in the non-hypomagnesium group group (38.0%) (P = 0.06) died. There was no link found between hypomagnesium group and 28-day mortality in sepsis patients (HR = 1.07; P = 0.87, 95% CI). The duration of mechanical breathing (P<0.01), duration of vasoactive medication usage (P 0.01), length of ICU stay (P<0.01), and length of hospital stay (P<0.01) were all longer in the hypomagnesium group group than in the non-hypomagnesium group group. In the subgroup analysis, the time spent without a

vasopressor (P<0.01) and without mechanical breathing (P<0.01) was considerably longer in the magnesium supplementation group than in the non-magnesium supplementation group. More notably, patients with magnesium treatment had reduced 14-day mortality (30.8% vs 48.9%, P = 0.01) and 28-day mortality (33.8% vs 48.9%, P = 0.03) than patients without magnesium supplementation". [48]

SMATERIALS AND METHODS

Source of data: Patients admitted to the Medical Intensive Care Unit, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi between 1st January 2021 to 31st December 2021 over a period of one year.

Study Design: A cross sectional study.

Study Period: JANUARY 2021 TO DECEMBER 2021.

SAMPLE SIZE:100

Sample size = Z^2pq / d^2

Where, z^2- standard normal variant at a 95% degree of confidence = 1.96

p - Expected proportion from population prevalence

q = 100-p

d - margin of error = 10% .

SAMPLE METHOD: Cross sectional study, all consecutive patients fulfilling the inclusion criteria will be included in the study, statistical analysis was done by SPSS using descriptive analysis and chi-square test.

Inclusion Criteria:

- Age > 18 years
- All patients of sepsis and its complication in admitted in ICU.

Exclusion Criteria:

- Patient in which day 1of ICU was missed.
- Patient admitted in general ward.

- Patient with documeted hypomagnesium group before ICU.
- Patient on Mg supplementation.
- Long term medication with PPI.
- Pregnant females.

Methodology:

- A one year Hospital based cross sectional
- Study from January 2019 to December 2019 at KLE's Prabhakar Kore Hospital and Medical Research Centre, Belagavi.
- An informed consent was obtained from all the subjects.
- Patients above the age of 18 years of age were included in the study.
- These subjects were categorised based on severity of sepsis on the basis of qSOFA score within 24 hrs of ICU admission.
- Blood sample were taken to estimate Magnesium level at the time of ICU admission.

Does the Study require any investigations or interventions to be conducted on patients or other humans or animals?

- Arterial blood gas
- Complete blood count
- Mini renal
- Live function test
- Serum electrolyte

STATISTICAL ANALYSIS

All the data were collected in proforma and entered in excel sheet. The collected data demographic data was summarized as frequency, percentage, mean and standard deviation. The summarised data were represented using tables, figures, bar diagram and pie charts. The mean difference between the continuous data was analysed using t-test, for follow-up data paired t-test and for categorical data Chi-square test was used to determine the significance between the parameters observed in this study. A $P < 0.05$ was accepted as significant. Statistical analysis was performed using SPSS version 22.0 (IBM SPSS, US) software operating on windows 10.

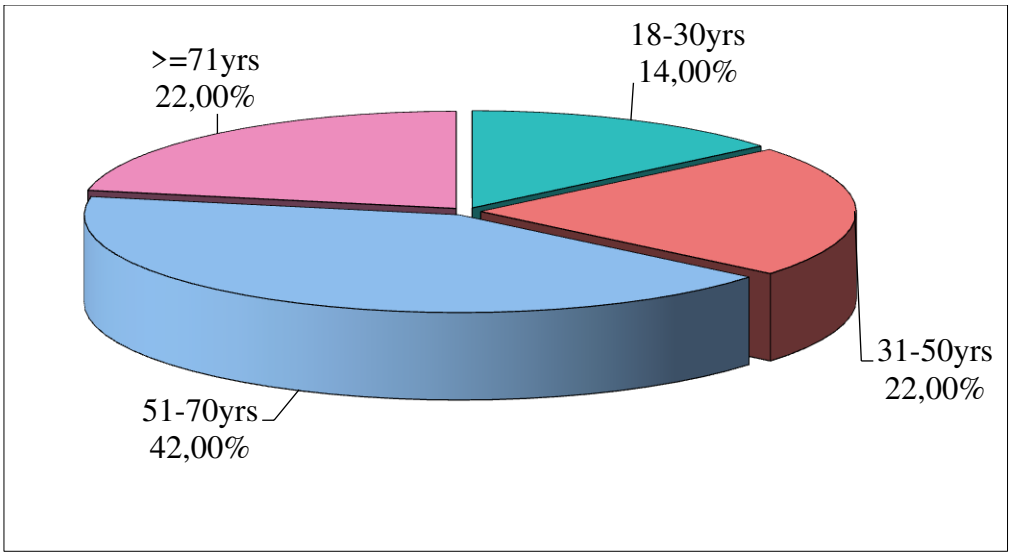
RESULTS

Table 1

Distribution of Patients Age wise.

Age groups	No of patients	% of patients
18-30yrs	14	14.00
31-50yrs	22	22.00
51-70yrs	42	42.00
>=71yrs	22	22.00
Total	100	100.00
Mean age	55.68	
SD age	17.62	

Graph 1. Distribution of Patients Age wise



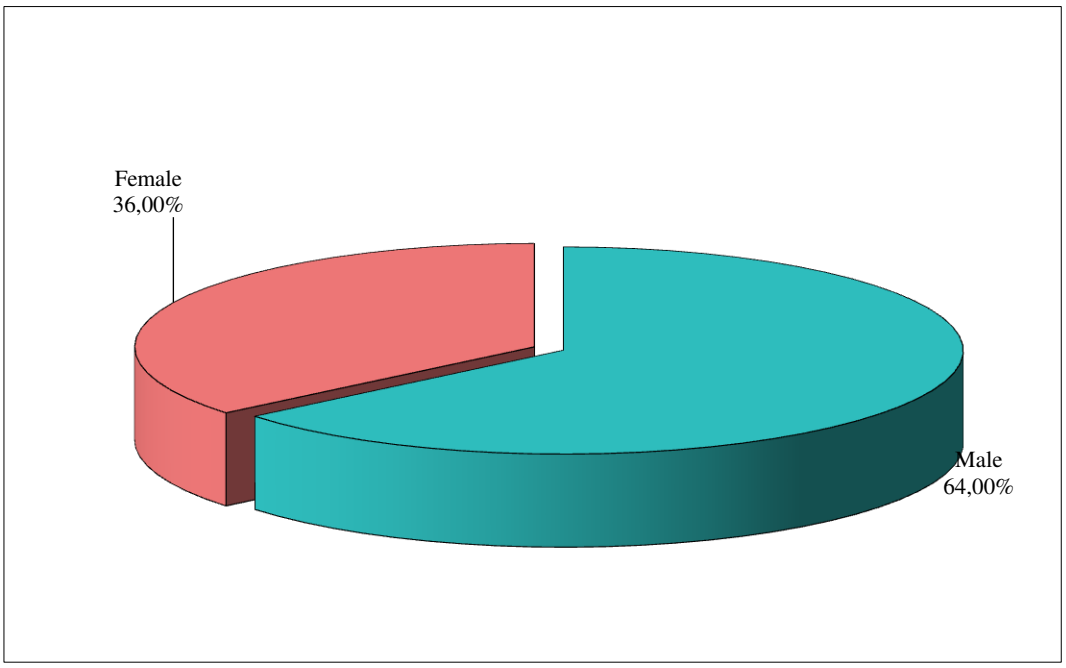
In our present study of 100 patients, the maximum number of patients were in the age group 51-70 years i.e.42 (42%), 22 each in age group of 31- 50 years and ≥71 years. There were 14 patients in the age group of 18-30 years. The youngest patient in our study population was 18 years and oldest was 85 years. Mean age of our patients was 55.68 ±17.62.

Table 2

Sex wise distribution of patients.

Gender	No of patients	% of patients
Male	64	64.00
Female	36	36.00
Total	100	100.00

Graph 2. Sex wise distribution of patients



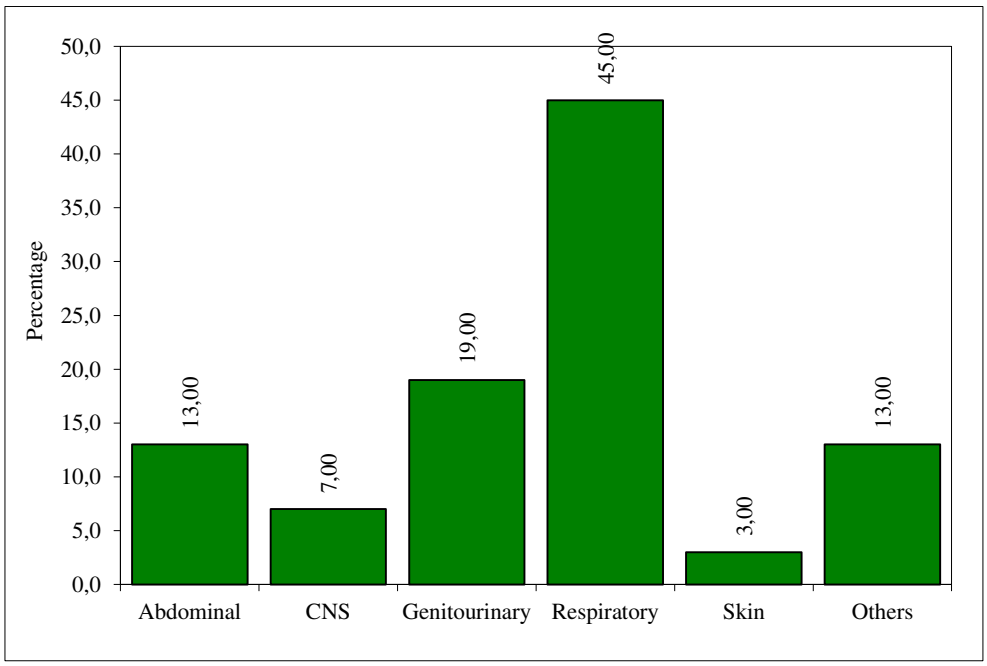
There were 64 male patients and 36 female patients in our study population. There was male preponderance observed with male: female ratio 1.7:1.

Table 3

Distribution of patient according to source of infection.

Severity of sepsis	No of patients	% of patients
Abdominal	13	13.00
CNS	7	7.00
Genitourinary	19	19.00
Respiratory	45	45.00
Skin	3	3.00
Others	13	13.00
Total	100	100.00

Graph 3. Distribution of patient according to source of infection



We analysed all our 100 patients according to the focus of the infection and found to have , majority of our patients i.e. 45 (45%) had a respiratory source of infections for sepsis, 19 patients had genitourinary source , 13 abdominal source, 7 central nervous system source , 3 patients had skin related infection and 13 patients we couldn't ascertain the focus of infections as a cause of sepsis.

Table 4

Distribution of patients according to Co- morbidities.

Co-morbidities	No of patients	% of patients
HTN	48	48.00
T2DM	38	38.00
IHD	6	6.00
Hypothyroidism	4	4.00
CKD	1	1.00
CLD	1	1.00
COPD	1	1.00
Haemorrhoids	1	1.00
Pulmonary tuberculosis	1	1.00
PVD	1	1.00

Graph 4. Distribution of patients according to Co- morbidities

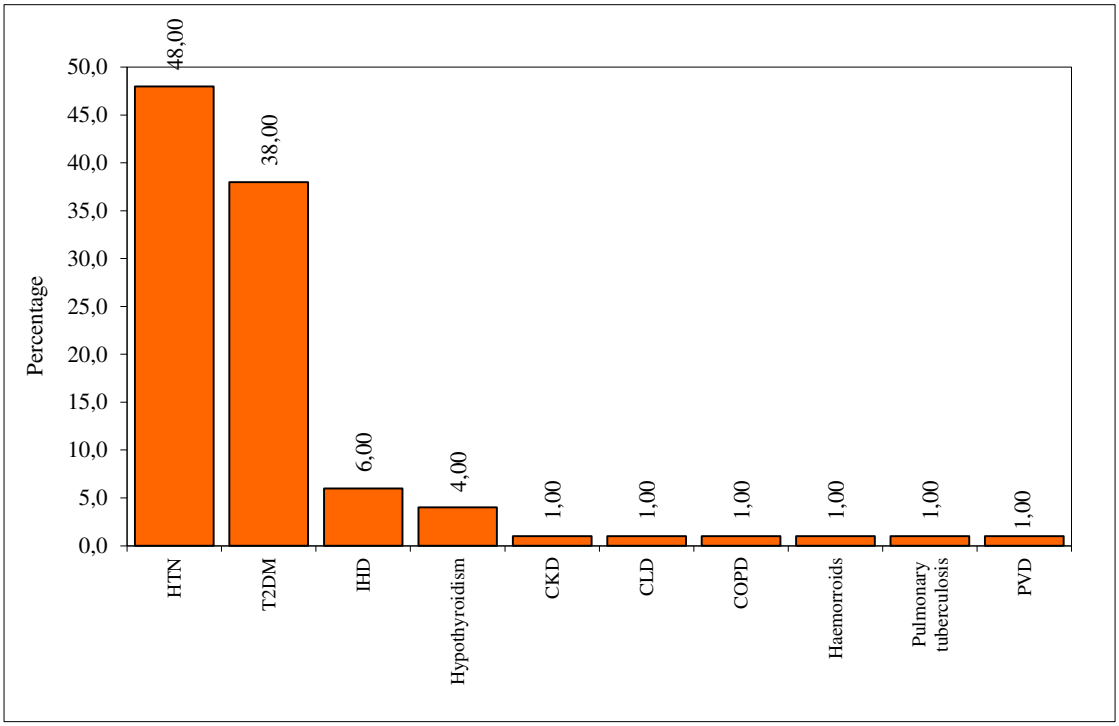


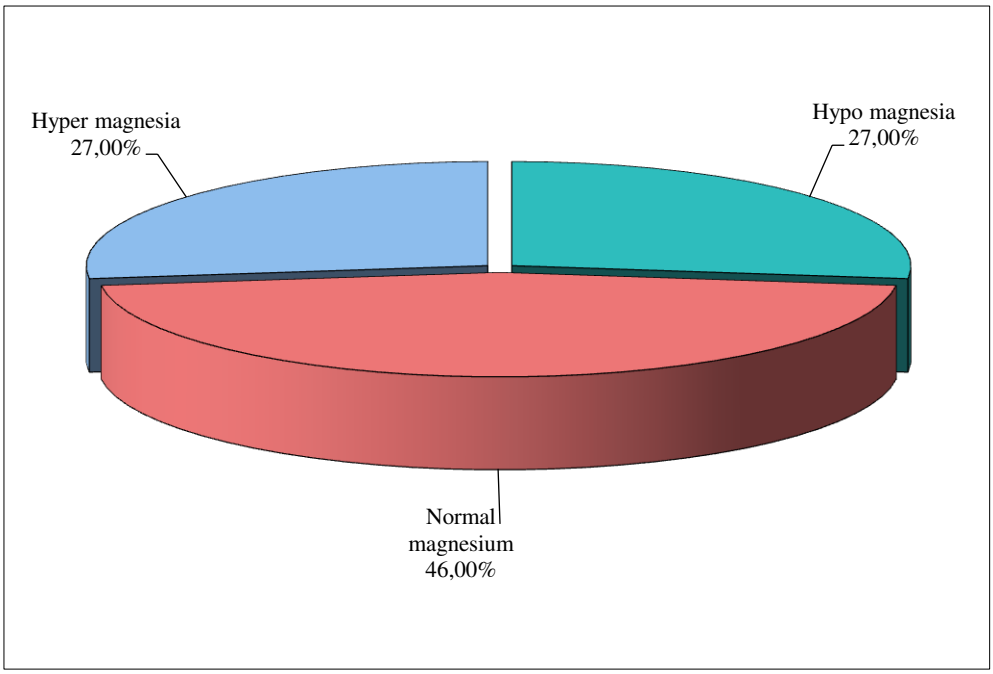
Table 4 depicts various co-morbidities our patients had. The commonest co-morbidities was Hypertension i.e. 48, 38 had Type 2 diabetes mellitus. The remaining co-morbidities are as depicted in the above table. However, there were overlapping of co-morbidities that was observed in our patients, which is shown in the table above.

Table 5

Distribution of patients based on serum magnesium levels.

Magnesium level	No of patients	% Of patients
Hypo magnesium	27	27.00
Normal magnesium	46	46.00
Hyper magnesium	27	27.00
Total	100	100.00

Graph 5. Distribution of patients based on serum magnesium levels



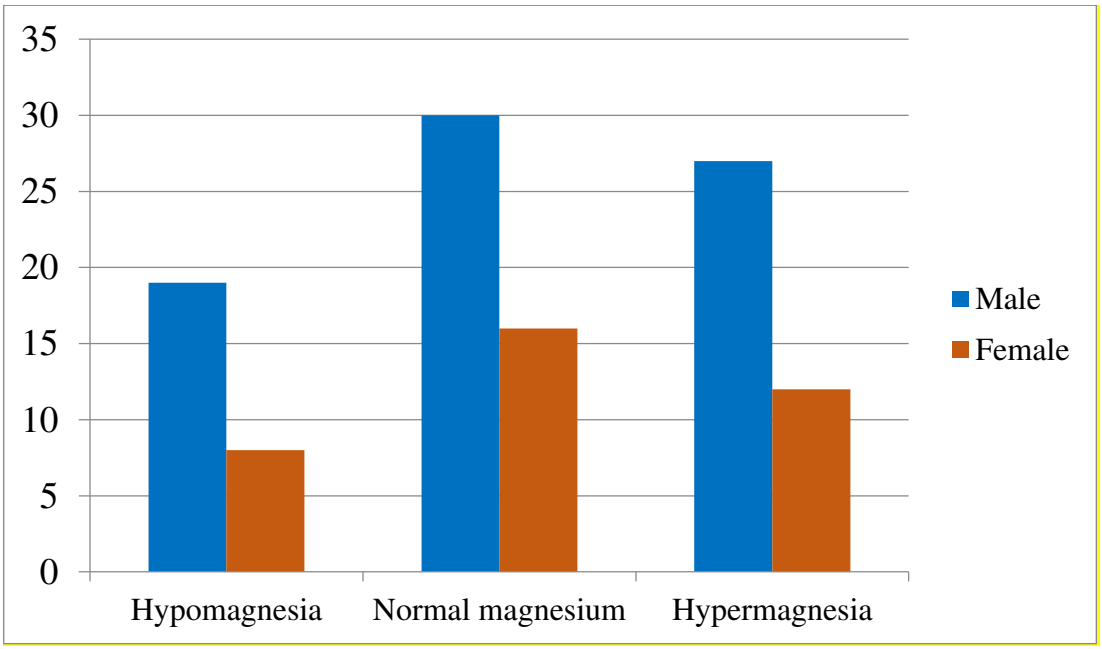
We subjected all our patients for serum magnesium estimation and results obtained were 46 patients had normal serum magnesium levels (1.6-2.2) at the time of presentation (one time estimation), 27 patients the levels were below normal range (<1.6) and remaining 27 patients had levels >2.2.

Table 6

Comparison of magnesium levels with gender.

Magnesium level	Male	Female	Total
Hypo magnesium	30	16	46
Normal magnesium	19	8	27
Hyper magnesium	15	12	27
Total	64	36	100

Graph 6. Comparison of magnesium levels with gender



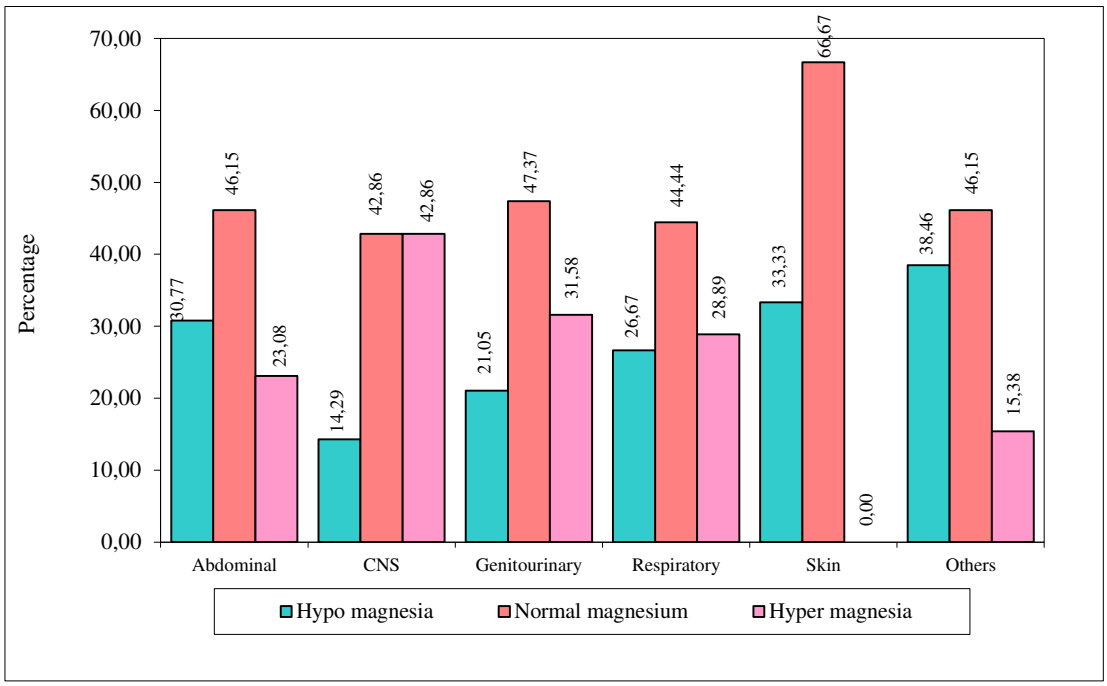
We attempted comparison of magnesium levels with gender and found there were 30 male patients with normal magnesium levels and 16 female patients. Hypomagnesium group was observed more in male patients i.e. 19 as compared to female i.e. 8 , even hypermagnesium group was more in male i.e. 15 as compared to 12 female patients.

Table 7

Levels of serum magnesium based on source of infections.

Severity of sepsis	Hypo magnesium	%	Normal magnesium	%	Hyper magnesium	%	Total	%
Abdominal	4	30.77	6	46.15	3	23.08	13	13.00
CNS	1	14.29	3	42.86	3	42.86	7	7.00
Genitourinary	4	21.05	9	47.37	6	31.58	19	19.00
Respiratory	12	26.67	20	44.44	13	28.89	45	45.00
Skin	1	33.33	2	66.67	0	0.00	3	3.00
Others	5	38.46	6	46.15	2	15.38	13	13.00
Total	27	27.00	46	46.00	27	27.00	100	100.0
Chi-square=4.1330, p=0.9410								

Graph 7. Levels of serum magnesium based on source of infections



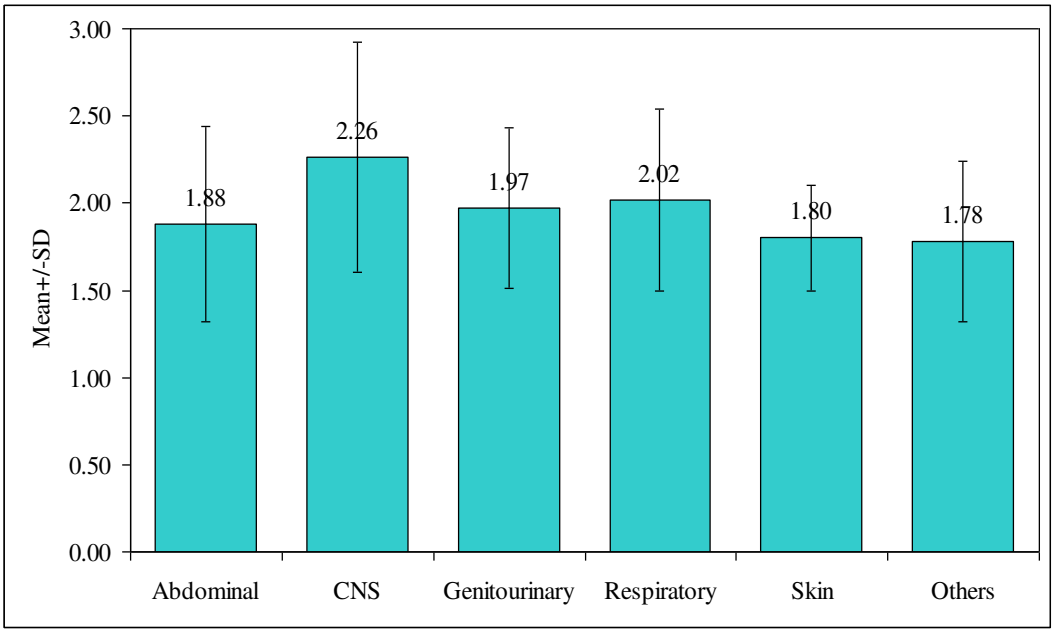
We took different source of infections of sepsis and compared with the serum magnesium levels and categorised as depicted in the above table. To our observation, there were more number of patients as far as normal levels were concerned, hypomagnesium group levels were concerned or hypermagnesium group in respiratory source of infections. The p value (0.9410) was statistically insignificant, for all the sources of infections.

Table 8

Comparison of serum Magnesium levels with source of infection by one way ANOVA method.

Severity of sepsis	Mean	Std.Dev.	Median	IQR
Abdominal	1.88	0.56	2.00	0.40
CNS	2.26	0.66	2.20	0.45
Genitourinary	1.97	0.46	1.90	0.40
Respiratory	2.02	0.52	2.10	0.40
Skin	1.80	0.30	1.80	0.30
Others	1.78	0.46	1.70	0.40
F-value	1.0472			
p-value	0.3948			

Graph 8. Comparison of serum Magnesium levels with source of infection by one way ANOVA



Further we tried comparison of serum magnesium levels with various source of infection by one way ANOVA method. The results obtained are shown above. P value was 0.3948, which was statistically insignificant for all the sources of sepsis.

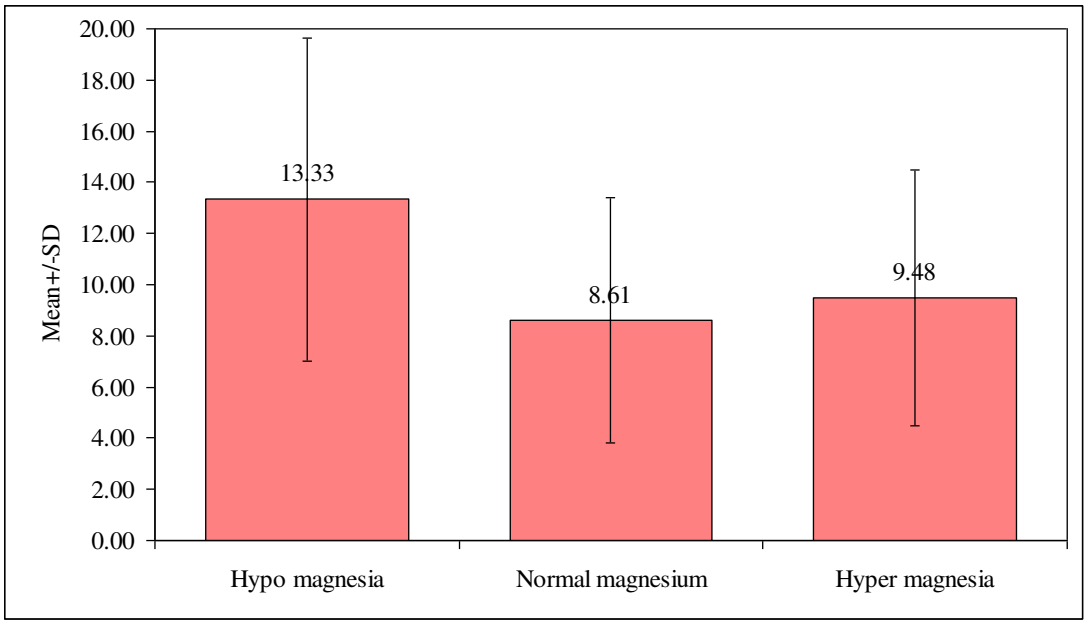
Table 9

Comparison of Magnesium levels with total duration of hospital stay by one way ANOVA method.

Magnesium level	Mean	Std.Dev.	Median	IQR
Hypo magnesium	13.33	6.33	11.00	4.00
Normal magnesium	8.61	4.77	8.00	3.00
Hyper magnesium	9.48	5.02	9.00	3.50
Total	10.12	5.61	9.50	3.25
F-value	7.0415			
p-value	0.0014*			
Hypo magnesium vs Normal magnesium	P=0.0012*			
Hypo magnesium vs Hyper magnesium	P=0.0239*			
Normal magnesium vs Hyper magnesium	P=0.7758			

*p<0.05 indicates significant

Graph 9. Comparison of Magnesium levels with total duration of hospital stay by one way ANOVA method.



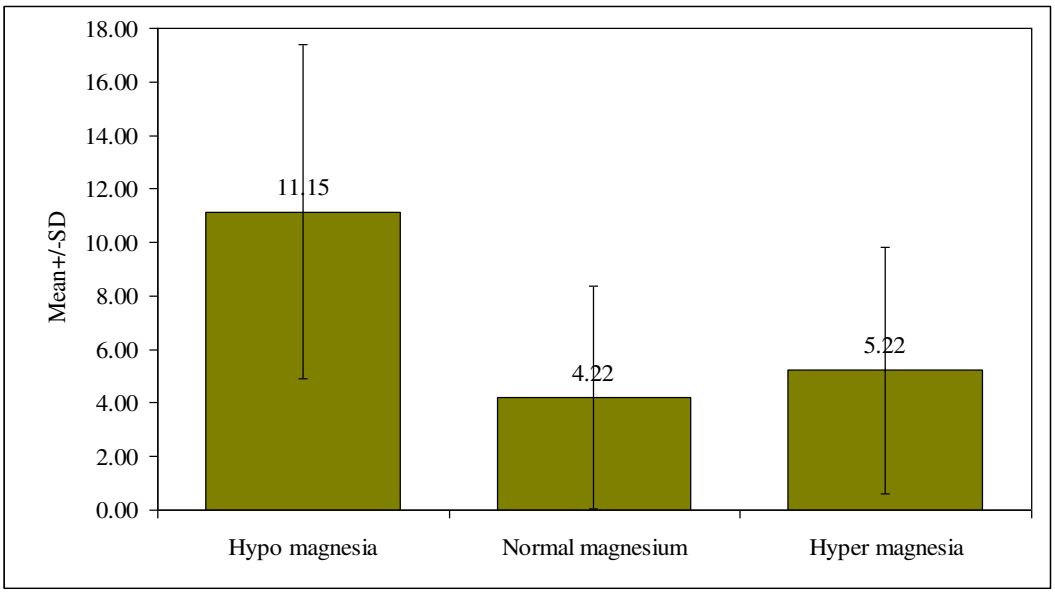
Further analysing all our 100 patients with their total duration of hospital stay which was compared with serum magnesium levels and categorised them as above. Levels were compared with normal magnesium group to hypomagnesium group, normal to hypermagnesium group and between hypomagnesium group to hypermagnesium group, the results obtained were statistically significant as depicted above.

Table 10

Comparison of Magnesium levels with mean no of days in Intensive care unit (ICU) by one way ANOVA method

Magnesium level	Mean	Std.Dev.	Median	IQR
Hypo magnesium	11.15	6.24	10.00	4.00
Normal magnesium	4.22	4.15	3.00	1.50
Hyper magnesium	5.22	4.62	3.00	3.00
Total	6.36	5.69	5.00	3.50
F-value	17.9321			
p-value	0.0001*			
Hypo magnesium vs Normal magnesium	P=0.0001*			
Hypo magnesium vs Hyper magnesium	P=0.0002*			
Normal magnesium vs Hyper magnesium	P=0.6768			

Graph 10. Comparison of Magnesium levels with mean no of days in Intensive care unit (ICU) by one way ANOVA method



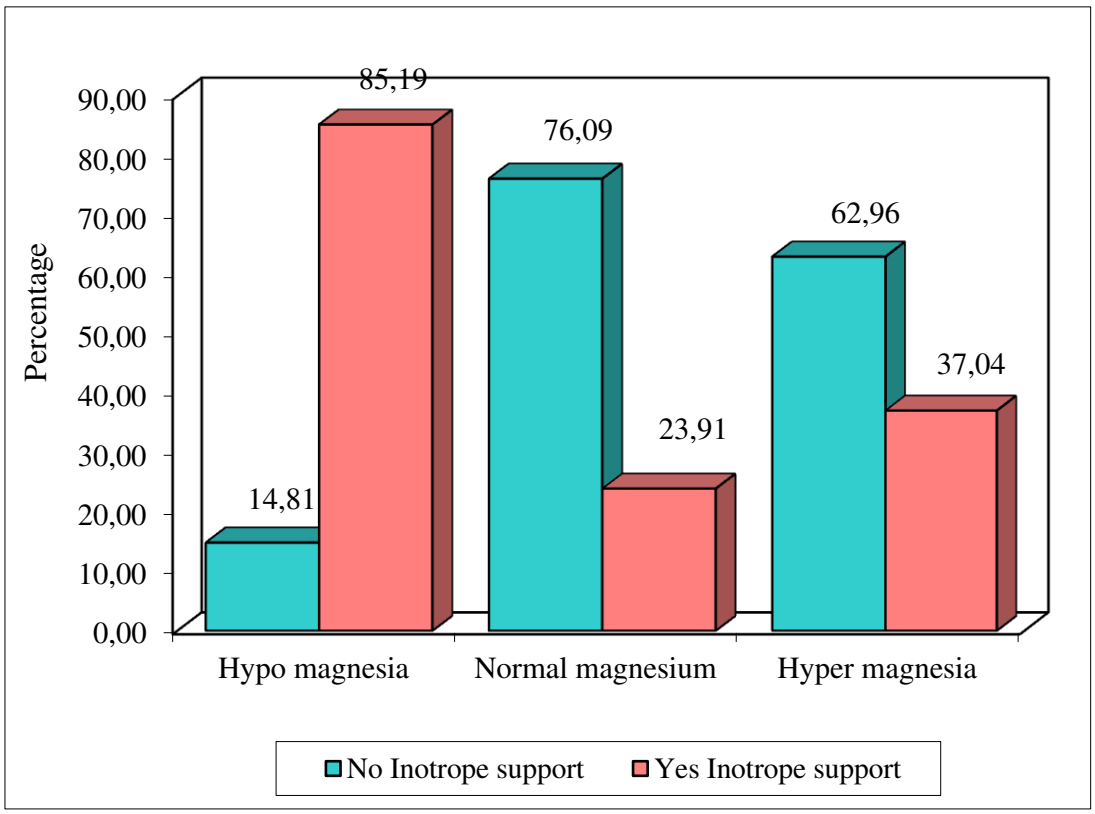
Similarly the levels were compared with number of days in ICU by one way ANOVA method and results analysed are tabulated in the above table. The mean ICU stay was more in patients with hypomagnesium group levels as compared to hypermagnesium group levels and p-value (0.0001) was statistically significant.

Table 11

Comparison of serum Magnesium levels with Inotrope support.

Magnesium level	No Inotrope support	%	Yes Inotrope support	%	Total	%
Hypo magnesium	4	14.81	23	85.19	27	27.00
Normal magnesium	35	76.09	11	23.91	46	46.00
Hyper magnesium	17	62.96	10	37.04	27	27.00
Total	56	56.00	44	44.00	100	100.00
Chi-square=26.6510, p=0.0001, S						

Graph 11. Comparison of serum Magnesium levels with Inotrope support



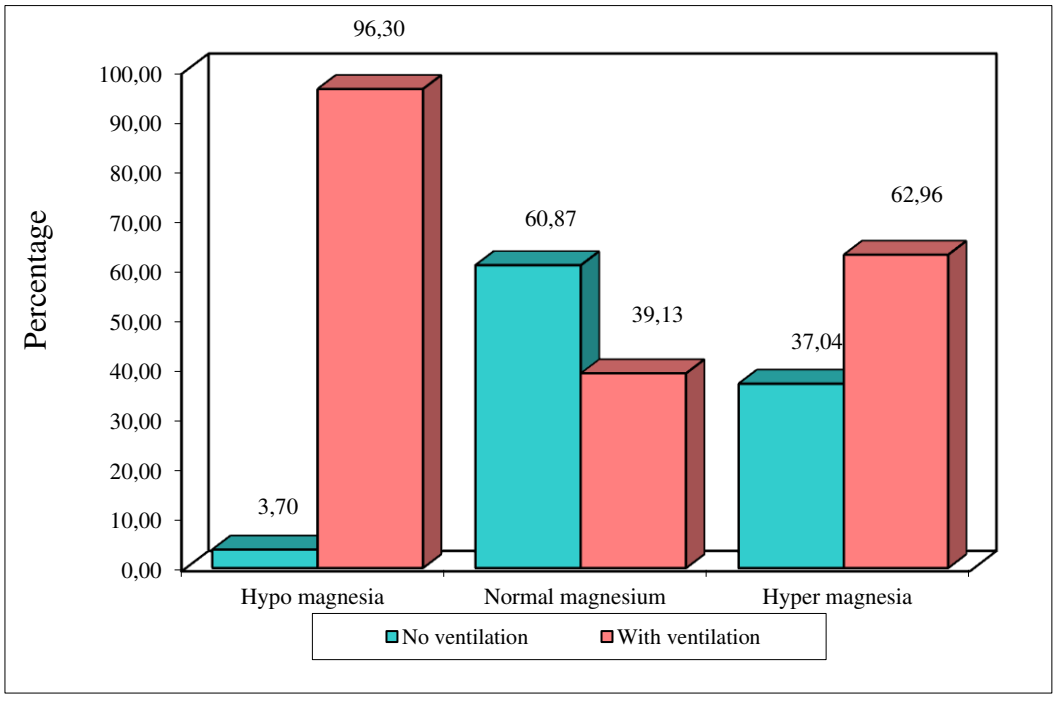
The above table depicts requirement of Inotrope support when compared with serum magnesium levels we found, 11 patients in normal magnesium group require Inotrope support, 35 did not require. 23 in hypomagnesium group require Inotrope and only 4 did not require. 10 patients in hypermagnesium group require Inotrope support and 17 did not require. P-value (0.0001) for all three groups is statistically significant.

Table 12

Comparison of serum Magnesium levels with or without mechanical ventilatory support.

Magnesium level	No ventilation	%	With ventilation	%	Total	%
Hypo magnesium	1	3.70	26	96.30	27	27.00
Normal magnesium	28	60.87	18	39.13	46	46.00
Hyper magnesium	10	37.04	17	62.96	27	27.00
Total	39	39.00	61	61.00	100	100.00
Chi-square=23.4310, p=0.0001, S						

Graph 12. Comparison of serum Magnesium levels with or without mechanical ventilatory support.



We took serum magnesium levels in all patients and compared the same with ventilator support and we found 26 patients require ventilator support whose magnesium levels were below normal levels (hypomagnesium group), however 17 patients also require ventilator support with hypermagnesium group, 18 patients with normal magnesium levels also require ventilator support. p-value (0.0001) was statistically significant as shown in table 12.

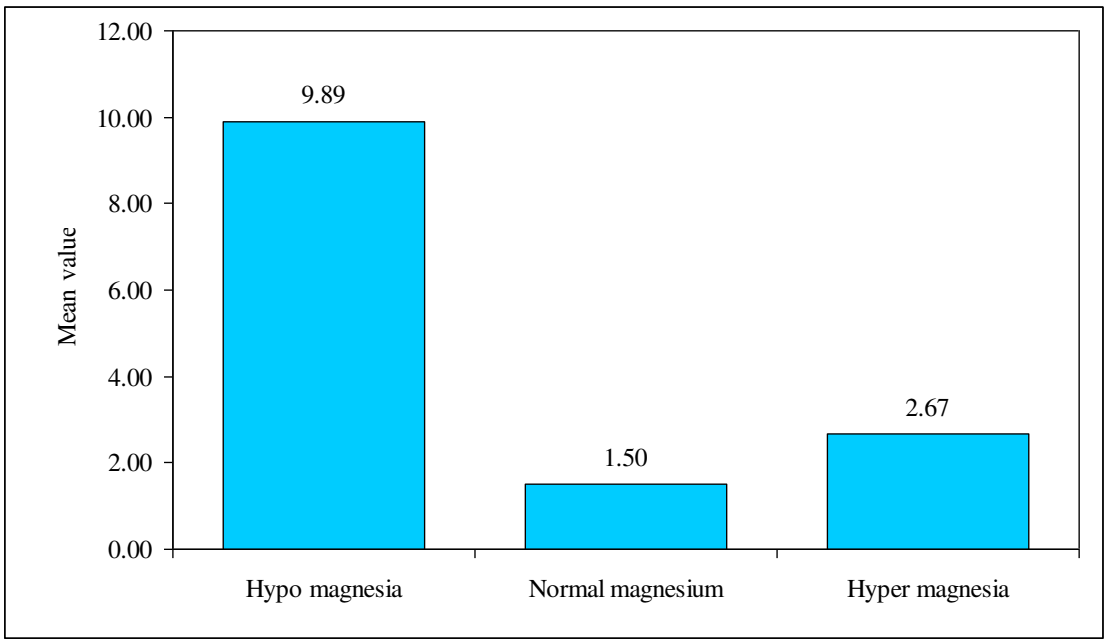
Table 13

Table: Comparison of serum Magnesium levels with total duration of mechanical ventilation by one way ANOVA method.

Magnesium level	Mean	Std.Dev.	Median	IQR
Hypo magnesium	9.89	6.94	7.00	5.50
Normal magnesium	1.50	4.12	0.00	0.50
Hyper magnesium	2.67	4.53	2.00	1.50
Total	4.08	6.21	1.00	3.00
F-value	24.1571			
p-value	0.0001*			
Hypo magnesium vs Normal magnesium	P=0.0001*			
Hypo magnesium vs Hyper magnesium	P=0.0001*			
Normal magnesium vs Hyper magnesium	P=0.6177			

*p<0.05 indicates significant

Graph 13. Comparison of serum Magnesium levels with total duration of mechanical ventilation by one way ANOVA method.



We found patients with hypomagnesium group, the mean duration of ventilator support was more and p-value was statistically significant as depicted in above table.

Table 14

Comparison of serum magnesium levels with, serum procalcitonin and total WBC counts by Karl Pearson's correlation coefficient method.

Variables	Correlation between magnesium levels with		
	r-value	t-value	p-value
Total WBC	-0.0857	-0.8513	0.3967
Serum procalcitonin	0.0079	0.0783	0.9377
Total counts	-0.0903	-0.8980	0.3714

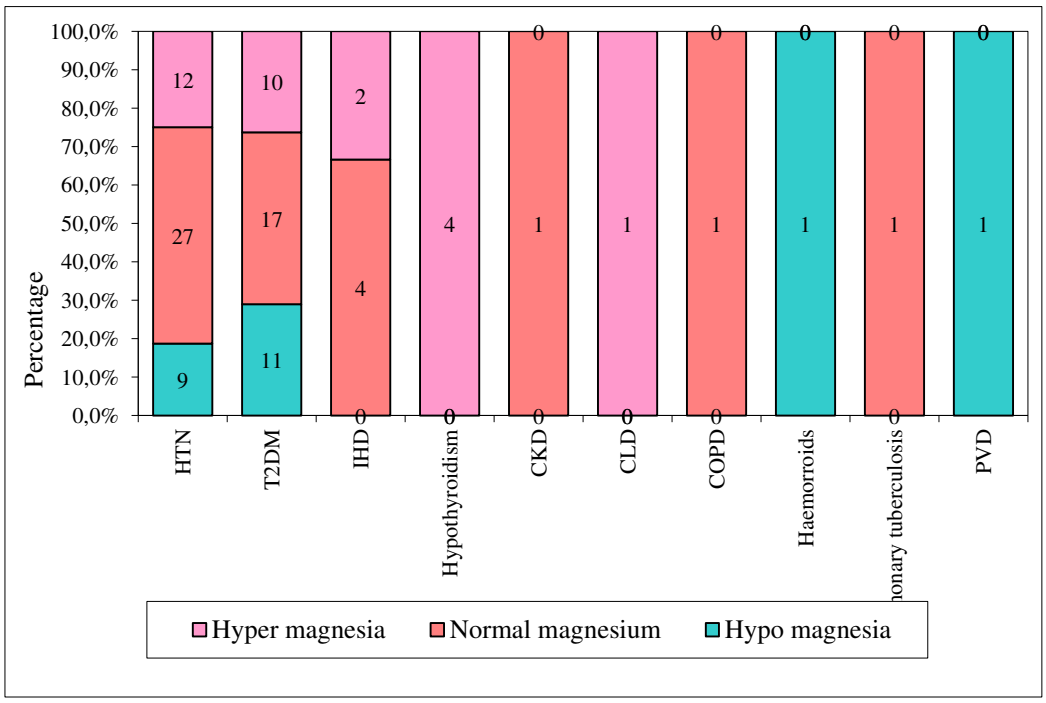
We attempted comparison of serum procalcitonin and total WBC with serum magnesium levels and found to have no co-relation. P-value was statistically insignificant for all.

Table 15

Comparison of serum magnesium with Co-morbidities.

Co-morbidities	Hypo magnesium	Normal magnesium	Hyper magnesium	Total
HTN	9	27	12	48
T2DM	11	17	10	38
IHD	0	4	2	6
Hypothyroidism	0	0	4	4
CKD	0	1	0	1
CLD	0	0	1	1
COPD	0	1	0	1
Haemorroids	1	0	0	1
Pulmonary tuberculosis	0	1	0	1
PVD	1	0	0	1

Graph 14. Comparison of serum magnesium with Co-morbidities.



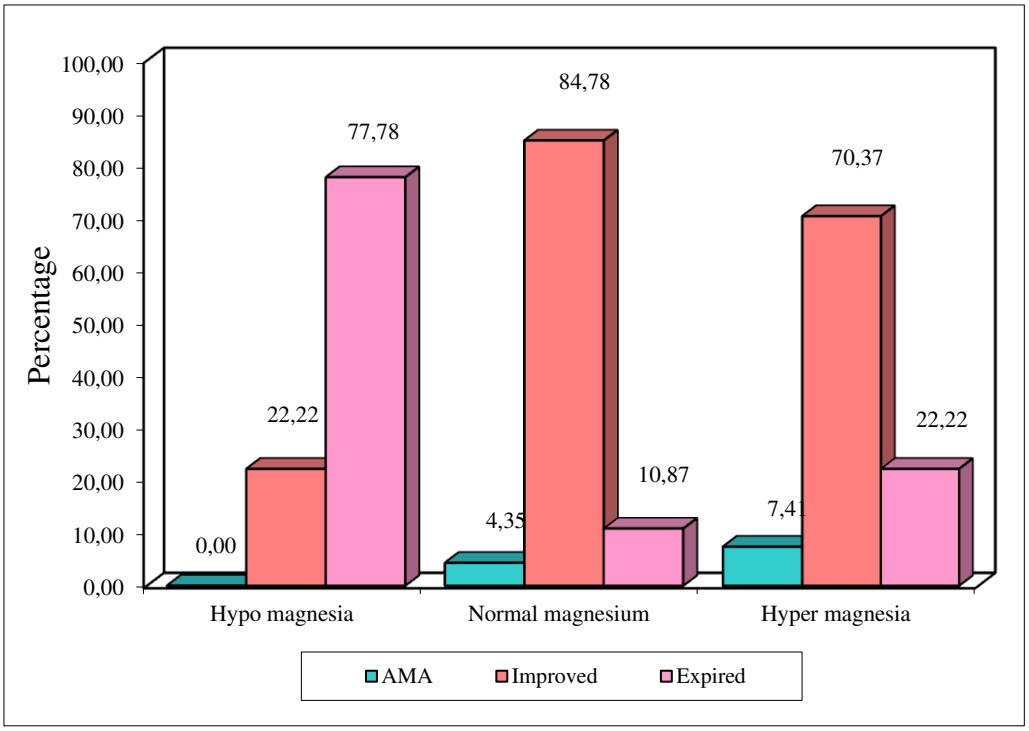
Serum magnesium levels were compared with different Co-morbidities as shown in above table and the results obtained are shown above.

Table 16

Comparison of serum Magnesium levels with mortality.

Male	Male	Male	Male	Male	Male	Male	Male	Male
Hypo magnesium	0	0.00	6	22.22	21	77.78	3	23.08
Normal magnesium	2	4.35	39	84.78	5	10.87	2	28.57
Hyper magnesium	2	7.41	19	70.37	6	22.22	4	21.05
Total	4	4.00	64	64.00	32	32.00	100	100.00
Chi-square=37.4230, p=0.0001, S								

Graph 15. Comparison of serum Magnesium levels with mortality.



Finally we tried to compare all our patients with their serum magnesium levels and outcome and found to have in group with normal magnesium levels 5 patients expired, in hypo magnesium group 21 patients expired and in hyper magnesium group 6 patients expired. P-value (0.0001) was statistically significant.

DISCUSSION

In the present study of 100 patients, the serum magnesium levels were estimated to assess the severity of sepsis. Serum magnesium levels were divided into three groups as normal magnesium group, hypomagnesium group, hypermagnesium group and same was compared with patients of sepsis by qSOFA scoring method and results obtained were analysed. All our 100 patients who presented with sepsis, their age range from 18-85 years (youngest 18 years old and oldest 85 years old). There were 42 patients in the age group of 51-70 years, 22 patients each in 31-50 years and ≥71 years and 14 patients in 18-30 years. The mean age was 55.68±17.6. A study by Murali et al.,[47] also found same observation in their study population. Another study by arun et al.,[49] also found similar observation in their study population but their sample size was 50.

In our study group there was male preponderance 64(64%) and female 36 (36%) with male to female ratio of 1.7:1. A study by Chenwei et al.,[48] of , also observed in their study group the male preponderance. Another study by Murali et al.,[47] also observed a male preponderance in their study population with 63 male patients and 37 female patients.

Further we analysed all our 100 patients taking into consideration the source of infections foe sepsis and found the respiratory infections was commonest source of sepsis i.e., 45 patients (45%), 19 patient's genitourinary source, abdominal 13, central nervous system 7 patients, skin 3 patients. However, 13 patients we could not ascertain source of infection responsible for sepsis. A study by motiul et al.,[50] also found similar observation in their study population the source of infection was respiratory. A study by Erickson et al.,[51] in their large study population of 2589 patients observed the commonest source of infection for sepsis was cardiovascular ,

haematological related infectious disease , respiratory and gastrointestinal infections in the following order.

In our study majority of our patients had hypertension as the commonest co-morbidities i.e. 48(48%), type 2 diabetes mellitus 38 patients, ischemic heart disease 6 patients, hypothyroidism 4. The other co-morbidities are depicted in table 4. There were overlapping of co-morbidities among the patients enrolled in the study. A study by Erickson et al.,[51]in their large study population have also found hypertension as most common co-morbidity. Even they observed overlapping of co-morbidities in their study population. A study by huabin et al.,[52] have observed anaemia as the commonest cause of sepsis. Other causes were congenital heart disease, diabetic ketoacidosis, liver related problems and malignancy.

We grouped our patients into 3 categories as normal magnesium group, hypo magnesium group and hyper magnesium group levels and found 46 patients had normal magnesium levels (1.6-2.2), 27 each in hypomagnesium group (<1.6) and hypermagnesium group (>2.2). A study by Murali et al.,[47] also have categorized in three groups as normal , hypo and hyper magnesium levels and found normal magnesium levels in 46% of patients , 39% in hypo magnesium group and 15% in hyper magnesium group. *Akshay et al.,[45]* have also categorized their patients in above three category and found 62% in normal magnesium group , 18 in hypo magnesium group and 20% in hyper magnesium group. In most of the studies it is shown that patients of hypo magnesium group is commonly observed in patients of severe sepsis and septic shock. A study by motiul et al[50]who showed the occurrence of sepsis was more commonly seen in patient of hypo magnesium group. A study by Safaviet al.,[53] have also observed severe sepsis and septic shock was more common in patients of hypo magnesium group. As we know magnesium play important role in sepsis, as

magnesium ions are essential for several important immunological functions and also serve as natural calcium antagonist and important step in causing a cellular injury, it is observed in animal model studies, magnesium deficiency may increase inflammatory cytokines with increase in harmful endotoxins which may further lead to cellular inflammation, injury and sepsis. It is also observed magnesium deficiency is strongly associated with increased mortality in experimental sepsis. The supplementation of magnesium has help in protection of these patients with sepsis against endotoxin challenge.

We tried comparison of magnesium levels with gender and found 30 out of 100 patients were male patients with normal magnesium levels. There were 19 patients with hypo magnesium levels who were males and 8 were females, remaining 27 patients of which 15 males had hyper magnesium levels and similarly 12 female patients. A study by Sunil et al.,[42] in their study population observed a similar finding, males were more in all 3 categories as compared to their counter parts. Another study byChenwei et al.,[48] who had large population of sample size in their study group, they also observed more number of males in all the 3 categories of hypo magnesium , normal magnesium and hyper magnesium .

We tried to look for magnesium levels based on their source of infections and results found were as tabulated in table 7. Though p-value was not statistically significant as far as levels and source of infection was concerned. A study by Erickson et al.,[51] in their large sample size of 2589 had different magnesium levels based on source of infection of sepsis. They also did not find any statistical significance in their study population. Another study by motiul et al.,[50]also compared magnesium level with source of infection and found to have insignificant p-value. In our study the respiratory source of infection had more number of patients in all 3

groups. The studies quoted i.e.Charat et al.,[51] they found the commonest source of infection for sepsis was cardiovascular related problems. Similarly , a study by Motiul et al., [50] they also found all the three categories was more observed in respiratory source of infection, almost similar to our study. A study by Motiul et al., [50] proposed there is a no clear cut mechanism for magnesium levels related to source of infection for sepsis. To best of our knowledge, various authors have also said the same thing, as there is no significant correlation observed for levels of magnesium and source of infections (sepsis).

We further compared the magnesium levels with one way ANOVA method and found no significant correlation between magnesium levels and different source of infection of sepsis. P-value (0.3948) was statistically insignificant (table 8). Most of the authors have not done comparison of levels of magnesium by one way ANOVA method.

Similarly we tried to compare magnesium level (one time estimation) with total hospital stay with one way ANOVA method and found to have some relevance with magnesium levels i.e. normal magnesium levels vs hypo magnesium, normal magnesium with hyper magnesium and hypo magnesium with hyper magnesium. P-value were statistically significant for all the 3 levels of comparison as stated above (9a- bar diagram, 9b- scatter diagram representation).A study by Chenweiet al.,[48]compared magnesium levels with number of days in hospital ,they also found significant p-value as far as hypo magnesium group was concerned. Whereas study by huabin et al.,[52]did not find a correlation with magnesium levels with hospital stay and p – value was statistically insignificant.

Similarly we attempted to estimate serum magnesium levels and compared the same with patients stay in Intensive Care Unit (ICU) by one way ANOVA method.

The results observed were compared between normal magnesium group with hypo magnesium group, normal magnesium and hyper magnesium group and hypo magnesium with hyper magnesium group. In all the 3 groups p-value was statistically significant. Also observed in hypo magnesium group the patients stay in ICU was more i.e., 11.15 ± 6.24, slightly more than mean stay was also observes in hyper magnesium group i.e. 5.22 ± 4.62. In patient with normal magnesium group the mean value (stay in ICU) was 4.22 ± 4.15 (table 10 a and 10 b). A study by Sikarin et al.,[43] who also observed the length of stay in ICU was more in hypo magnesium group in their study population. Another study by motiul et al., [50]also observed more number of days in ICU in patients with low magnesium levels that was 9.13 ± 5.10. Study by Chenweiet al.,[48] in their large study population of 645 observed more number of days, patients with low magnesium levels stayed in ICU as compared to normal levels. Magnesium is an essential co-factor of several important enzymes in a human body. Magnesium plays an important role in normal physiological functions of organs like brain, heart, and skeletal muscle. Magnesium levels are low in critically ill patients, otherwise also in general population. Overall, 65% low levels were observed in patients who were critically ill. Low levels of magnesium may affect neuromuscular and cardiovascular function. Deficiency of same may present with seizure, muscular weakness, and respiratory depression due to neuromuscular junction hyper excitability. It may be also responsible for coronary artery disease, cardiac arrhythmias, heart failure and can eventually lead to death of patients in the serious patients. Some studies have found the higher magnesium levels may also lead to severe muscle weakness, respiratory depression, hypotension, fatal arrhythmias leading to sudden cardiac death. The high level of magnesium is a risk for increased mortality especially in patient of renal failure and heart failure patients. Many studies

have quoted the increased risk of complications in critically ill patients with hypo magnesium group than hyper magnesium group. The disordered magnesium regulation may be due to gastrointestinal loss of ions because of diuretics or renal tubular dysfunction. It may be due to tissue sequestration also. Hence it is warranted to check the serum magnesium levels in the critically ill patients (sepsis) and if required correction of the same. Another explanation for hypo magnesium in such critically ill patients is because of the dialysis but may believe the disordered metabolism leading on to hypo magnesium or hyper magnesium with increased risk of mortality is highly controversial. In another large study it was observed hypo magnesium does occur >40% of hospitalized patients and in 60% in Inpatients post operative. In setting of medical ICU, it was observed to the tune of 65% and almost 90% in surgical ICU.

Further we analysed patients with magnesium levels with or without Inotrope support and results obtained were, 23 patients required inotrope support in hypo magnesium group, 10 patients in hyper magnesium group required inotrope support and in patients with normal magnesium group 11 patients required the support. The p-value (0.0001) was statistically significant (table 11). A study by Chenwei et al.,[48] in their large study population 645, more than 235 patients required inotrope support in hypo magnesium group . However, 222 required pressure support with inotrope whose magnesium levels was normal. Another study by motiul et al.,[50] 61% of their patients required inotrope support in patients of low magnesium group. However, they also observed 38.9% required inotrope support with normal magnesium levels.

We also attempted comparing magnesium levels in patients requiring assisted ventilator support and found there were 26 patients in our study who required ventilator support who's magnesium levels were low.17 patients who had hyper

magnesium also require ventilator support. However, 18 patients with normal magnesium levels required ventilator support. Totally 61 patients were on ventilator support. In hypo magnesium group only 1 patient did not require ventilator support, 10 in hyper magnesium group and 28 patients with normal magnesium did not have ventilator support need. Total patients in all 3 groups did not require the ventilator support. The p-value (0.0001) was statistically significant inpatients requiring ventilator support in hypo magnesium group. A study by Erickson et al.,[51]in their large study population 2589 there were total of 1872 in hypo magnesium group of which 510 required ventilator support, in hyper magnesium group there 312 patients of which 128 required ventilator support. Total 405 patients in normal magnesium group of which 131 required ventilator support. In their study group also the p-value was statistically significant in patient with hypo magnesium group requiring ventilator support. In sharp contrast to study by motiul et al.,[50] in their study population , more than 75% with low magnesium levels requiring ventilator support as compared to normal magnesium levels. In humans' magnesium is the common abundant cation. Most of the time deficiency is seen as common electrolyte imbalance yet, in the clinical practice which is under diagnosed. It has been observed more than 50% of patients, hypo magnesium is observed in ICU settings. Many studies have reflected that these patients have significant higher morbidity and mortality. Soto identify this, whether it is true in patients of sepsis, we undertook this study. There were many factors which need to be considered during application of this fact, because many of times magnesium levels are influenced by diuretics, aminoglycosides, and sepsis itself. It has been observed in animal studies, there is increase in proliferation in proinflammatory cytokines like interleukin-6, TNF –alpha, in hypo magnesium group. Also, it is observed hypo magnesium may lead to increased risk of inflammation by

activation of macrophage, neutrophils and endothelial cells. The animal model study in rats has shown increase apoptosis in thymus in magnesium deficiency rats. Magnesium helps in release of nitric oxide from the cells and which seems as a penetrating factor, infections in body cavities such as sinuses, respiratory infections like pneumonia and many infections in mucosa of many organs. Hypo magnesium may prevent free release of nitric oxide from cells which may have in turn effect on blood vessels dilatation of blood vessels, which may increase risk of repeated infections. In gastro intestinal tract, hypo magnesium may be required by thiamine pyrophosphate formation from thiamine and as a result of this the low levels of gastric acid production, which may lead to increased risk of gastro intestinal infection. Erickson et al.,[51] stated hypo magnesium may be responsible for increased risk for acute bacterial infection including sepsis, bronchopneumonia and urinary tract infections.

Similarly we compared the levels of serum magnesium with duration of mechanical ventilation in our patients and found mechanical ventilator support required more number of days in hypo magnesium group i.e. 9.89 ± 6.94. In normal magnesium group the number of days required was 1.50 ± 4.12 and in hyper magnesium group, it was 2.62 ± 4.83. The p-value was statistically significant in patient of hypo magnesium who required a greater number of days with ventilator support (table 13a- bar diagram 13b- scatter diagram). Study by Amin et al.,[3] also observed in their study population the duration of ventilator support was more in hypo magnesium group i.e. 12 ± (4.00 – 14.25). In patient with normal magnesium group the mean duration was 3.00 (0.00-8.00). P-value was statistically significant in their study population also. This is in sharp contrast to study by Sunil et al.,[2] who found in their study population the mean duration of ventilator support in days was (3.07 ±

5.05) in hypo magnesium group. Where as in normal magnesium group it was (2.15±3.46). The p-value was not statistically significant (0.09).

Further we took procalcitonin and total WBC count and compared same with magnesium levels and found to have no correlation. A study by Sunilet al.,[2] have compared total WBC count with magnesium levels which was statistically insignificant. Another study by Amin et al.,[3] have also compared total WBC count. They also found no significant correlation between hypo magnesium and total WBC count. To best of our knowledge no author has compared serum magnesium levels with serum procalcitonin.

Similarly, the serum magnesium levels were compared with different co-morbidities in our study and did not reflect any correlation which influenced associated co-morbidities with that of serum magnesium levels (table 15). A study by huabin et al.,[52] have compared serum magnesium levels with patients co-morbidities and found no significant correlation to the levels of serum magnesium. P-value was also statistically insignificant. Another study by Erickson et al.,[51]also compared serum magnesium levels with co-morbidities of their patients which did not reflect any correlation as far as co-morbidities was considered with different levels of magnesium.

A sincere attempt was made to compare serum magnesium levels with mortality and there was significant correlation was observed as far as mortality was concerned in patients with hypo magnesium group (table 16). P-value was statistically significant (0.0001). A study by Murali et al.,[47] have also observed increased mortality in their study population with patients who had hypo magnesium group. Another study by huabin et al.,[52] also found there was increased mortality in patients

of hypo magnesium group . P-value was also statistically significant in their study population.

More often than not the estimation of serum magnesium is a neglected investigation (entity) by doctors. Though it is a very important body constituent which has got reflection as far as infection is concerned (sepsis), outcome of patients and mortality but yet it's a underdiagnosed problem. So we feel it's worthwhile to carry out simple investigation which can be done in all hospital settings and can be made use in clinical practice especially so in patient who are critically ill, which would guide us to decide the outcome of patient as well as correcting low levels (hypo magnesium) and same was observed by many authors, as we have stated in our study. So we feel that a routine magnesium level should be asked for in patient with sepsis and ICU setting which would help us to treat our patients. We found age was not influencing the levels in our small size of 100 patients, the males were more in number and hypo magnesium was observed more in males because of this reason. As far as source of infections was concerned the respiratory source was more and hypo magnesium was observed more in it. Various studies have given different levels of magnesium. We have stated in our study normal levels as 1.6-2.2, hypo magnesium as <1.6 and hyper magnesium as >2.2. Patients who had hypo magnesium the stay was also more in the hospital (ICU) as well as their requirement of ventilator support was also for more number of days. Even we observed in our study the inotrope support was more in patients with hypo magnesium group. Finally there was increase mortality in our small size if 100 patients with hypo magnesium. Finally we concluded our discussion saying, it's worthwhile to subject all our patients to magnesium estimation, which is a simple tool, which can be made use in our day to day practice.

CONCLUSION

In our present study of 100 patients with sepsis (qSOFA score method) there were more number of patients in the age group of 51-70 years followed by ≥71 years. There was male preponderance observed in our study, the respiratory source of infection was more as a source of sepsis in our study population. The most common co-morbidities observed was hypertension followed by type 2 diabetes mellitus. There were many patients who had overlapping of co-morbidities. We had almost 46% patients whose magnesium levels were normal and equal number of patients with hypo magnesium and hyper magnesium was observed. Hypo magnesium was more observed in male gender compared to female since there was male preponderance in our study population could be the reason. Hypo magnesium was more observed in respiratory infections as compared to other source of infection for sepsis. Similarly patient with hypo magnesium had longer duration of hospital stay that was observed in our study. Even the patient who required ICU stay, even in this had longer ICU stay. We observed the Inotrope support was more required in hypo magnesium group. Patient who required assisted mechanical ventilation the hypo magnesium group there were more number of patients and the duration of mechanical ventilation was also more in hypo magnesium group. The comparison of magnesium level with serum procalcitonin and total WBC count had no relation. The co-morbidities did not have any correlation with serum magnesium. Finally we sum up saying there was increase mortality in patient of hypo magnesium group.

SUMMARY

In the present study of 100 patients of correlation between serum magnesium and severity of sepsis admitted in Intensive Care Unit at KLE Dr Prabhakar kore Hospital and medical research centre, Belagavi during study period from January 2021 To December 2021 was undertaken to find the effect of serum magnesium levels on outcome of our patients. The results observed on analyzing the levels serum magnesium, there was prolonged hospital stay, ICU stay, inotrope support and ventilator support in patients of hypo magnesium group. There was positive correlation with hypo magnesium group with was prolonged hospital stay, ICU stay, inotrope support, ventilator support and increased mortality.

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ANNEXURE I

INFORMED CONSENT

Title Of Research Study:

"Correlation Between Serum Magnesium And Severity Of Sepsis In Patients Admitted In Intensive Care Unit At KLE Dr. Prabhakar Kore Hospital And Medical Research Centre, Belagavi – A Cross Sectional Based Study".

Principal Investigator:-
REG NO: BG0120016

Post Graduate Student,

Department Of General Medicine,

JNMC, Belgaum.

Guide:-

Dr. _____

MD (General Medicine)

Professor of General Medicine and Unit Chief.

Department of General Medicine,

Jawaharlal Nehru Medical College, Belagavi**.**

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.

All cost for investigation will be bared by the investigator.

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,

Dr._____ MD (General Medicine) Professor of General Medicine and Unit Chief. Jawaharlal Nehru Medical College, Belagavi	**REG NO: BG0120016** Post Graduate Student, Department Of General Medicine, JNMC, Belagavi.

DR HARSHA HEGDE,

Chairperson, JNMC, IEC & Scientist D, ICMR

National Institute of Traditional Medicine

Belagavi. - 9480422500

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:

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ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: – REG NO: BG0120016
ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಮಾರ್ಗದರ್ಶಿ: ಡಾ. _____

ಎಂಡಿ (ಜನರಲ್ ಮೆಡಿಸಿನ್)
ಪ್ರೊಫೆಸರ್ ಮತ್ತು ಯುನಿಟ್ ಚೀಫ್
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಪರಿಚಯ ಮತ್ತು ಉದ್ದೇಶ: –
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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:
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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:
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ಸಂಸ್ಥೆ / ಪ್ರಾಯೋಜಕರ ನೀತಿ:
ಈ ಸಂಶೋಧನೆಗೆ ಅನ್ವಯಿಸುವುದಿಲ್ಲ

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ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿಮಗೆ ಯಾವುದೇ ಉಡುಗೊರೆಗಳನ್ನು / ಪ್ರೋತ್ಸಾಹಕಗಳನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ / ನೀಡಲಾಗುವುದಿಲ್ಲ.

ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸಲು ಅಧಿಕಾರ:
ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಎಂಡಿ ಪದವಿ, ವಿಮರ್ಶೆ ಮತ್ತು ಪ್ರಕಟಣೆಯ ಪೂರ್ಣಗೊಳಿಸುವ ಅಗತ್ಯತೆಯ ಭಾಗವಾಗಿ ಬೆಲ್ಗಾಮ್‌ನ ಕೆಎಲ್‌ಇ ವಿಶ್ವವಿದ್ಯಾಲಯಕ್ಕೆ ರವಾನಿಸಲಾಗುತ್ತದೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಅಥವಾ ಭವಿಷ್ಯದಲ್ಲಿ ನೀವು ಈ ಕೆಳಗಿನ ವ್ಯಕ್ತಿಗಳನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು,

ಡಾ. _____

ಎಂಡಿ (ಜನರಲ್ ಮೆಡಿಸಿನ್)
ಪ್ರೊಫೆಸರ್ ಮತ್ತು ಯುನಿಟ್ ಚೀಫ್
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್‌ಎಂಸಿ, ಬೆಳಗಾವಿ.

REG NO: BG0120016
ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್‌ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಡಾ.ರೂಪಾ ಎಂ ಬೆಲ್ಲದ
ನೈತಿಕ ಸಮಿತಿಯ ಮುಖ್ಯಸ್ಥ
ಮಾನವ ಸಂಶೋಧನೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

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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

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माहितीपूर्ण संमती

"के.एल.ई. डॉ.प्रभाकरकोरे हॉस्पटिल अँड मेडकिल रसिर्च सेंटर, बेलगावी मध्ये - इंटेंसीव्ह केअर युनटिमध्ये दाखल झालेल्या रुग्णांमध्ये सेरम मॅग्नेशयिम आणि सेपसिची तीव्रता यांच्यात सहसंबंध क्रॉस सेक्शनल बेस्ड स्टडी ."

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एमडी (सामान्य औषध)
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सामान्य औषध वभिाग,
जे.एन.एम.सी, बेळगावी.

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आपण संशोधन अभ्यासाचा भाग होण्यास सहमत असल्यास, आपणास संबंधति इतहिास वचिारला जाईल आणि संबंधति क्लनिकिल 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 असुवधिाची समस्या उद्भवू शकते. ज्या स्थानावरून रक्त काढले आहे त्या जागेवर सूज, वेदना, लालसरपणा (क्वचतिच घडते) होऊ शकतो. या तपासणीमुळे आपल्याला फायदा होणार नाही परंतु भवष्यिात इतरांना उपयुक्त ठरणाया या अभ्यासाचा तुम्ही भाग व्हाल.

वकिल्पः

या अभ्यासामध्ये भाग घेणे ऐच्छकि आहे. आपण या अभ्यासामध्ये भाग न घेणे नविडू शकता.
आपण भाग घेण्याचे ठरवल्यिास आपण नंतर आपले मत बदलू आणि अभ्यासापासून दूर जाऊ शकता. आपल्या नरि्णयामुळे आपल्याला प्राप्त झालेल्या सद्य कवि भवष्यिातील आरोग्य सेवा कवि इतर सेवा बदलणार नाहीत. अभ्यास डॉक्टर कवि प्रायोजक या अभ्यासात आपला सहभाग कधीही थांबवू शकतात. आपण अभ्यासामध्ये भाग न घेणे नविडल्यास, आपल्या अट असलेल्या रूग्णांसाठी तुम्हाला प्रमाणति उपचार मळिेल.

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 अभ्यासावरील माहिती प्रकाशित केली जाऊ शकते परंतु आपली ओळख कोणत्याही प्रकाशनात गोपनीय असेल.

संस्था / प्रायोजक यांचे धोरण:

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

परिणाम प्रकाशित करण्यासाठी अधिकृतता:

अभ्यासाचे निकाल एमडी पदवी, आढावा आणि प्रकाशन पूर्ण करण्याच्या आवश्यकतेनुसार केएलई विद्यापीठ, बेळगाव येथे पाठवले जातील.

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

डॉ. _____
एमडी (सामान्य औषध)

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सामान्य औषध विभाग,
जे.एन.एम.सी, बेळगावी.
मोबाइल - 9845710945

REG NO: BG0120016

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

डॉ. रूपा एम बेल्लद
अध्यक्ष, नैतिक समिती मानव संशोधन
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 कवि कायदेशीररत्या अधकृित प्रतनिधिीची सही / डावा अंगठा प्रटि

सहभागीचे नाव: ....

स्वाक्षरी / डावा अंगठा ठसा: ....

सहभागीचा

कायदेशीररीत्या अधकृित नाव: ....

प्रतनिधिी / पालक

स्वाक्षरी / डावा अंगठा ठसा: ....

साक्षीचे नाव: ....

स्वाक्षरी / डावा अंगठा ठसा: ....

अन्वेषकांचे नाव आणि स्वाक्षरीः ....

तारीख:

ठकिाणः

ANNEXURE II

PROFORMA

CASE NO	

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

Complaints at presentation	
Past history	
Family history	
Personal history	
Treatment history	

VITALS:

Temperature	
Pulse	

Respiratory rate	
Blood pressure	

PHYSICAL EXAMINATION:

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

SYSTEMIC EXAMINATION:

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

INVESTIGATIONS:

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

FOCI OF SEPSIS

Respiratory	
Genitourinary	

Abdominal	
C.N.S	
Skin	
Others	
Non identified	

SL.NO.	IP NO	AGE	SEX	ADDRESS	OCCUPATION	CO-MORBIDITIES	FOCI OF SEPSIS	DIAGNOSIS	BLOOD PRESSURE	ionotrop support	RESPIRATORY RATE	GCS	qSOFA SCORE	PULSE	SERUM PROCALCITONIN	TOTAL COUNT	MAGNESIUM	TOTAL HOSPITAL STAY	NO OF DAYS IN ICU	NO OF DAYS IN VENTILATION	Outcome
1	1067395	72	F	Hubli	Shopkeeper	HTN, T2DM, Hypothyroidism	Respiratory	CKD with right Nephrectomy with pnuemonia	130/80	no	26	10	2	100	2.86	30000	2.6	5	3	2	AMA
2	1066942	52	M	kallehol	labourer	HTN,T2DM,hypothyroidism	others	CKD with congestive cardiac failure with severe PAH with severe anaemia	106/60	no	18	4	2	98	26.9	25,800	2.3	19	8	5	I
3	1067152	63	M	Hirebagewadi	Farmer	HTN	Abdominal	Acute pancreatitis secondary to gall stone	180/110	no	26	13	2	100	3.2	16700	1.7	10	3	0	I
4	1067019	55	F	Angol	Housewife	Haemorroids	Respiratory	Acute respiratory failure in septic shock secondary to pneumonia	80/40 on ionotropes	yes	24	10	3	96	100	24700	1.5	12	10	7	E
5	1066094	49	M	Hanuman nagar ,belagavi	Clerk	T2DM	Respiratory	COVID 19 pnuemonia with ARDS	98/70	no	31	15	2	96	0.09	19700	2	10	5	0	I
6	1067469	32	M	Raibag	Driver	HTN	Abdomial	Decompesated CLD with SBP	98/60	no	25	15	2	84	1.07	18900	1.8	12	5	0	I
7	1067630	70	M	Athani	Retired	HTN	Abdominal	Acute pancreatitis,peptic ulcer disease, AKI secondary to sepsis	110/70	no	26	13	2	92	100	11100	2	6	1	0	I
8	1071664	28	M	Athani	Clerk	HTN	Respiratory	Decompensated liver disease , pnuemonia,acute kidney injury	120/80	yes	24	10	3	110	2.01	13900	1.5	10	2	0	E
9	1076156	35	M	Jamkhandi	shopkeeper	nil	Respiratory	Antipsychotic drug overdose , schizophrenia	90 /50	no	26	3	3	140	1.09	14100	1.9	10	10	5	I
10	1073070	30	M	karnataka	Buisness	HTN	Genitourinary	CKD	160/90	no	26	13	2	100	0.9	16300	2.8	7	2	0	I
11	1078339	83	M	karnataka	retired	HTN	Respiratory	acute exacerbation of COPD , pnuemonia , CVA	90/60	yes	26	12	3	110	11.2	35200	2	8	2	0	I
12	1078154	64	M	kalloli	cooli	HTN	central nervous system	CVA, AKI secondary to sepsis , Epilepsy , IHD	80 SBP	yes	24	10	3	100	5.92	17300	2.6	10	2	0	I
13	1077655	78	M	Belagavi	Retired	Nil	Respiratory	Hemoptysis secondary to koch	100/60	yes	26	9	3	94	1.67	15600	1.4	10	5	4	E
14	1078309	65	F	Kallehol	Housewife	HTN	CNS	Severe subarachnoid haemorrhage, polycystic kidney disease	150/90	yes	24	3	2	60	1.34	15800	3.5	4	4	4	E
15	1067641	62	M	Belgundi	Farmer	Nil	Others	Metastasis to lung and spine	80/50	yes	24	15	2	82	2.09	20500	1.5	3	3	3	E
16	1064882	48	M	Hukkeri	Storeworker	Nil	Others	CA buccal mucosa	100/70	yes	26	15	2	78	2.3	25000	1.4	28	20	20	E
17	1067480	25	M	Sattegiri	Farmer	HTN	CNS	Acute meningitis encephalopathy	120/80	no	25	10	2	82	1.89	19400	2.3	12	10	4	I
18	1067671	27	M	Athani	Student	T2DM	Respiratory	Left lung emphysema	100/60	yes	24	10	3	80	0.9	38300	2.4	4	3	2	AMA

19	1067894	78	M	shadashiv nagar	retired	HTN	Respiratory	pnuemonia	100/60	no	25	15	2	90	1.9	12700	2.6	5	1	0	I
20	1072256	65	M	Belagavi	Farmer	HTN, T2DM	CNS	CVA	140/80	no	25	12	2	76	0.01	15300	1.5	10	7	6	I
21	1073421	38	F	Gokak	Housewife	Nil	Respiratory	Pnuemonia	120/80	no	25	14	2	92	2.4	12700	1.9	5	1	0	I
22	1075709	82	F	Bailhongal	Housewife	Nil	Genitourinary	Septic shock secondary to urosepsis, old CVA	90/50	yes	26	9	3	89	2.89	18700	2.4	8	8	0	I
23	1075742	47	M	Angol	Worker	Nil	Abdomial	Decompensated CLD	90/60	yes	27	10	3	78	1.89	15800	2.5	10	2	0	I
24	1075210	49	M	Sadashiv nagar	Shopkeeper	T2DM, IHD	Respiratory	Rhinocerebral mucormycosis,septic shock,AKI secondary to toxic nephropathy	90/60	no	24	15	2	88	2.4	15800	2.5	14	10	3	I
25	1075340	47	M	belgundi	driver	HTN	abdominal	Decompensated ALD with hepatic encephalopathy with AKI	90/60	no	25	15	2	84	0.1	16700	2.2	14	3	0	I
26	1075023	70	F	Mudalagi	Housewife	HTN,T2DM	Respiratory	Acute pulmonary Edema ,Inflammatory Bowel Disease	100/62	yes	24	15	2	78	1.23	17000	2.1	5	5	1	E
27	1074452	57	M	Belgaum	Farmer	T2DM	Genitourinary	Left pyelonephritis with sepsis with AKI ,CLD	90/60	yes	26	15	2	80	1.67	11200	2.2	15	3	1	I
28	1074681	60	M	Yaradal	Daily wage worker	Nil	CNS	Fungal sinusitis with rhinocerebral involvement with sepsis with AKI ,old CVA	110/70	no	26	9	2	88	2.45	24600	2.2	2	2	0	AMA
29	1078307	30	M	Yaradal	Driver	Nil	Others	Rickettsial fever ,sepsis with MODS ,with AKI	90/60	yes	27	3	3	112	3.56	25700	1.3	15	15	15	E
30	1089077	52	F	Kallehol	Housewife	T2DM,HTN,hyperthyroidism	Abdominal	AKI with MODS,decompensated cirrhotic liver disease	96/70	no	28	15	2	100	1.07	11000	2.4	7	4	0	I
31	1089001	30	M	Bagewadi	Business	T2DM, HTN	Respiratory	viral pnuemonia	80/60	yes	29	10	3	84	1.9	13500	1	23	23	23	E
32	1087565	73	M	Sadashiv nagar , belagavi	Retired	HTN, T2DM, IHD	Respiratory	Severe COVID 19 pnuemonia	100/60	no	28	15	2	92	0.82	16500	2.5	10	5	2	I
33	1088474	73	M	Kangrali,belagavi	Farmer	HTN	Others	Encephalopathy secondary to sepsis ?viral	140/80	no	25	10	2	88	1.2	17500	1.3	18	10	7	I
34	1088233	45	M	Shivaji Nagar	Buisness	CLD, HTN	Respiratory	Sepsis in mods , bilateral pnuemonia , chronic liver disease	110/70	no	29	9	2	86	2.7	19000	2.6	6	2	1	I
35	1088598	77	M	Vijay Nagar, belagavi	Retired	HTN	Respiratory	Viral pnuemonia	100/60	no	28	15	2	86	0.43	12800	2.2	8	2	1	I
36	1088627	24	M	Belagavi	Student	Nil	Abdominal	Acute edematous pancreatitis secondary to unknown etiology	100/60	no	52	15	2	78	0.43	18300	2.1	5	1	0	I
37	1088557	85	M	Belagavi	Farmer	HTN	Respiratory	Heart failure secondary to anaemia , hypoalbuminemia	90/60	no	25	15	2	102	0.3	12300	2.1	5	3	0	I
38	1086641	71	M	Belagavi	Retired	Nil	Respiratory	viral pnuemonia	110/70	yes	28	13	3	78	7.4	18900	1.5	14	10	7	E
39	1087937	30	M	Ram Durg,belagavi	Autodriver	HTN	others	Rickettsial fever ,sepsis with MODS ,with AKI	80 SBP	yes	26	15	2	82	1.7	16400	2.2	5	1	0	I

40	1086529	82	F	Belagavi	Housewife	Type 1 DM	Genitourinary	Septic shock secondary to do stenting , type 1dm , CKD	100/60	yes	28	15	2	88	1.2	31500	1.2	22	22	22	E
41	1087888	67	F	Adarsh Nagar, belagavi	Housewife	CKD,COPD,IHD	Respiratory	Acute exacerbation of COPD with secondary infections	110/70	no	28	13	2	98	0.09	16900	2	5	1	0	I
42	1087654	33	M	Mutaga	Banker	HTN	genitourinary	Septic shock secondary to urosepsis, old CVA	110/70	no	26	10	2	88	1.2	19400	1.9	5	1	1	I
43	1086571	24	M	Muchandi ,belagavi	Buisness	NIL	others	Encephalopathy secondary to sepsis ?viral	90/60	yes	25	15	2	82	18.93	24200	1.6	25	22	22	E
44	1086616	48	M	Hindalgo	Buisnessman	T2DM , HTN	Respiratory	viral pnuemonia	90/60	yes	28	9	3	92	2.56	12300	1.2	22	20	20	E
45	1086378	38	F	Gokak	Housewife	NIL	genitourinary	urosepsis	120/70	yes	28	10	3	98	4.25	18540	1.3	25	22	22	E
46	1086811	70	M	Belagavi	Farmer	T2DM, HTN	Skin	Penile abscess	100/50	no	15	12	2	78	0.04	15200	2.1	7	2	0	I
47	1086641	71	M	Nisargi	Retired	T2DM, HTN	respiratory	acute exacerbation of COPD	100/60	yes	25	10	3	82	8.87	16100	1.3	14	10	7	I
48	1085881	59	F	Gandhi Nagar, belagavi	Housewife	HTN	Genitourinary	AKI on CKD	90/60	no	28	15	2	92	30.25	18540	1.6	6	3	1	I
49	1085354	74	M	Parishwad	Ex service man	HTN , T2DM	Skin	cellulitis	100/60	no	24	15	2	74	21.09	22400	1.8	2	1	0	I
50	1085416	65	M	Yellur	Shopkeeper	Nil	Respiratory	Old pulmonary Koch , bronchiectasis, pulmonary arterial hypertension	98/60	no	29	15	2	86	1.55	18900	2.2	2	1	0	I
51	1084406	65	M	mudalagi	farmer	HTN	Respiratory	pnuemonia with COPD	100/60	yes	29	15	2	82	2.78	22600	2.1	11	5	2	E
52	1084407	71	F	raibag	housewife	nil	genitourinary	AKI secondary to acute gastroenteritis	98/70	no	27	15	2	78	0.09	21500	2.8	9	5	2	I
53	1083962	66	M	gokak	cooli	pulmonary tuberculosis	Respiratory	post TB sequelae	100/60	no	27	15	2	92	0.71	13500	2.1	5	2	0	I
54	1082762	56	M	yellur	ex service man	Type2DM	Abdominal	Acute kidney injury, acute liver disease , varicose veins	110/60	yes	30	15	2	90	28.6	24000	0.9	12	12	12	E
55	1082493	29	M	Belagavi	Buisness	Nil	Others	Dengue fever with thrombocytopenia, heart block	120/90	yes	28	15	3	62	0.9	13200	1.2	11	11	10	E
56	1082246	56	M	Hosatti , belagavi	Retired	HTN	others	Dengue fever with thrombocytopenia, heart block	140/90	no	24	10	2	78	1.2	15600	2.7	10	2	1	I
57	1081974	48	M	station road, belagavi	buisnessman	HTN, T2DM	Respiratory	COPD	110/70	yes	27	13	3	112	0.32	23900	2.2	21	18	17	E
58	1079292	58	M	Mallikarjun nagar, belagavi	Ex service man	HTN	Abdominal	Decompensated liver disease, PR BLEED secondary to haemorroids	100/70	no	26	13	2	100	0.5	13200	2.1	7	7	0	I
59	1078473	58	F	Belagavi	Housewife	Nil	Respiratory	Rheumatic heart disease with mitral stenosis with atrial fibrillation	80/50	yes	34	15	2	88	42.47	25400	2.3	22	22	22	E
60	1078381	64	F	Mudalagi	Housewife	T2DM	Respiratory	COPD with pnuemonia	90/60	yes	28	10	3	82	10.23	15420	1.5	10	7	6	E

61	1078002	65	M	Ram Durg,belagavi	Retired	HTN, T2DM	Abdominal	Hypoxic brain injury secondary to cardiac arrest , s/p ERCP cholelithiasis, myoclonus seizures	100/50	yes	26	3	3	93	1.5	16200	1.3	8	8	8	E
62	1077643	56	F	Uchagaon	Housewife	Hypothyroidism	Respiratory	Rheumatic heart disease with mitral stenosis with mitral regurgitation with atrial fibrillation, moderate pulmonary arterial hypertension, bilateral pnuemonia	100/50	no	24	15	2	82	0.42	13900	2.3	15	7	0	I
63	1075023	70	F	Hindalga	Housewife	HTN	respiratory	Acute Pulmo edema , acute kidney injury , inflammatory bowel disease	120/80	no	24	15	2	78	5.2	14800	3.6	5	1	0	I
64	1076523	64	F	Hukkeri	Retired	Nil	Respiratory	old kocks, pnuemonia	130/80	no	26	12	2	82	2.87	15700	2.6	17	10	0	I
65	1090143	41	M	Ramadurg	Buisness	HTN	Respiratory	pnuemonia	100/60	no	25	15	2	88	1.31	14830	2.2	11	4	4	AMA
66	1090778	35	F	Sutapatti	Housewife	T2DM	genitourinary	sepsis secondary to urosepsis with MODS	98/60	no	26	15	2	78	8.23	19200	1.5	10	8	4	I
67	1091122	18	F	Belagavi	Student	Nil	Respiratory	Bilateral pneumothorax	100/60	yes	26	15	2	118	4.75	6400	1.4	11	9	8	E
68	1091343	70	F	Savadatti	Housewife	T2DM	genitourinory	urosepsis	100/70	no	27	15	2	84	5.23	24000	1.7	6	3	1	I
69	1091981	60	F	Gokak	Housewife	HTN,T2DM	Respiratory	Accelerated hypertension , pulmonary edema, recurrent CVA, HTN, T2DM	160/90	no	24	13	2	88	2.45	12100	1.7	11	3	0	I
70	1092069	52	F	Raybag	Housewife	T2DM	Others	Partial hanging suicidal attempt secondary to depressive episode, uncontrolled T2DM with DKA , Delirium.	100/60	no	27	15	2	84	4.5	20300	1.8	8	5	1	I
71	1092181	80	F	Belagavi	Housewife	HTN	CNS	Septic encephalopathy with COVID 19 pnuemonia	100/60	no	18	5	2	92	7.56	29400	2	13	5	1	I
72	1092044	54	M	Belagavi	Buisnessman	T2DM	Respiratory	pnuemonia	100/60	yes	25	13	3	100	8.56	17800	2.6	9	9	9	E
73	1092448	75	F	Mattikatte, badami	Housewife	HTN,T2DM	Others	Left supracondylar femur fracture , sepsis with septic shock , ischaemic hepatopathy , AKI .	100/70	no	24	15	2	72	3.86	18000	1.7	9	3	0	I
74	1092458	33	M	Sawantwadi	Worker	T2DM	Abdominal	acute gastroenteritis	80/60	yes	29	15	2	110	63.24	16200	2.8	18	10	8	E
75	1092511	38	M	Mekalamardi	worker	Nil	Respiratory	COPD with pnuemonia	100/60	no	24	15	2	80	100	15300	1.9	3	1	0	I
76	1092321	65	F	Siddeshwar nagar	housewife	HTN	Respiratory	Congestive cardiac failure , Dilated cardiomathy with EF 35% with severe MR with TR , COPD , o/p/c CA breast	80/60	no	26	15	2	82	23.78	15300	1.6	7	2	1	I
77	1092345	45	F	Sadashiv nagar	Housewife	T2DM	Genitourinary	Urosepsis	100/70	no	26	15	2	98	18.9	18900	2	8	3	0	I
78	1122286	60	M	Belagavi	Buisnessman	T2DM	Respiratory	Operated case of CA oesophagus	100/50	yes	30	7	3	127	26	18500	2	10	5	1	I
79	1093854	20	F	ambedkar ,mudalgi	student	Nil	Genitourinary	Urosepsis	120/80	no	28	12	2	90	100	12100	2.3	9	3	1	I
80	1094022	61	M	Jadhav nagar	engineer	T2DM, PVD	Respiratory	Ca lung, with metastasis ,post covid .	100/60	yes	27	10	3	110	1.1	19500	1.5	7	7	7	E
81	1123266	62	M	Illkal	Housewife	HTN	Respiratory	Pulmonary edema secondary to LVH with HTN urgency	100/60	no	30	15	2	96	5.65	21500	2.2	5	2	1	I

82	1122633	21	F	Gokak	Student	Nil	Respiratory	Sepsis with AKI with postpartum hypernatraemia with osmotic demyelination with ARDS with GTCS	120/90	no	28	12	2	98	0.43	23300	2.8	5	1	0	I
83	1120735	72	F	Belagavi	Housewife	T2DM, HTN	Genitourinary	Sepsis secondary to urosepsis with MODS	100/60	yes	26	11	3	146	10.89	27300	1.9	2	1	0	I
84	1094477	54	M	yaligar,dharwad	farmer	T2DM	Respiratory	type2 Respiratory failure secondary to chronic hypersensitivity pnuemonitis	90/64	yes	30	15	2	112	3.16	15100	2.9	6	2	2	E
85	1094258	70	F	Belagavi	Housewife	Nil	Genitourinary	Urosepsis with AKI with septic meningoencephalitis	100/60	no	24	15	2	80	9.35	18900	2.4	5	2	2	I
86	1094989	46	M	Bhutramatti	farmer	T2Dm	Genitourinary	Septic shock with urosepsis with aspiration pnuemonia old CVA	120/60	yes	28	15	2	84	22.35	18200	2.4	5	3	2	E
87	1095658	61	M	Hukkeri	Buisnessman	HTN,T2DM,IHD	Others	Sepsis in septic shock with MODS , IHD s/p CABG	100/60	no	26	10	3	100	100	10600	2.2	7	5	0	I
88	1096186	67	M	Gokak	Retired	HTN	Respiratory	Acute excarberation of COPD with secondary infection	100/60	no	24	15	2	78	20.95	19300	2.1	12	7	0	I
89	1096979	36	M	Belagavi	Worker	Nil	Genitourinary	Severe metabolic acidosis secondary to sepsis in MODS with AKI on RRT	80SBP	yes	36	9	3	130	0.8	15000	1.5	10	10	10	E
90	1096980	76	F	Ilkal	Housewife	Nil	Genitourinary	Cardiogenic shock secondary to heart failure with reduced ejection fraction , urosepsis	90/60	yes	26	15	2	80	9.28	15500	1.9	15	9	6	E
91	1096241	74	M	Ramdev Pallu,belagavi	Retired	T2DM	respiratory	old pulmonary Koch , bronchiectasis, pulmonary arterial hypertension	100/60	no	24	15	2	82	9.78	22800	1.7	14	8	0	I
92	1099526	83	F	Nanawadi	Housewife	Nil	Abdominal	Acute gastroenteritis	90/50	yes	28	10	3	110	8.99	18100	1.2	14	14	14	E
93	1099651	68	F	Gandhi Nagar, belagavi	Buisness	HTN , T2DM	Respiratory	Septic shock with DIC secondary to APML , Left Lobe pnuemonia.	90/40	yes	18	3	2	80	2.82	16300	1.4	7	5	4	E
94	1101090	40	F	Laxigalli	Housewife	Nil	Others	Organophosphorus poisoning	108/60	no	28	15	2	82	10.32	22200	1.9	13	7	0	I
95	1102516	28	F	Sainik nagar	Housewife	Nil	Genitourinary	5months of gestation with urosepsis secondary to pyelonephritis	100/50	no	27	15	2	84	0.97	19100	1.7	6	2	0	I
96	1102214	52	M	Khanagaon	Buisnessman	HTN,T2DM,IHD, CVA	C.N.S	meningitis, obstructive hydrocephalus, bed sore, sepsis with AKI	80/50	yes	28	6	3	113	0.8	20200	1.7	12	7	2	I
97	1119250	82	M	Gokak	Farmer	Nil	Skin	Right cellulitis with septic shock S/p fasciotomy with AKI with CLD with Varicose vein	100/60	yes	28	13	3	96	3.89	48400	1.5	7	4	1	I
98	1120064	48	M	Sadashiv nagar	Buisnessman	Nil	Abdominal	Acute severe necrotising pancreatitis with bronco pleural peritoneal fistula , old tuberculosis	100/60	yes	26	15	2	100	8.25	17700	1.4	20	20	18	E
99	1125355	78	F	Belagavi	Housewife	IHD ,T2DM,HTN	Genitourinary	IHD with cardiogenic shock with urosepsis with diabetes mellitus	100/60	no	26	15	2	88	30.78	19800	1.9	8	2	0	I
100	1085416	65	M	Yellur	Shopkeeper	Nil	Respiratory	Old pulmonary Koch , bronchiectasis, pulmonary arterial hypertension	90/60	no	29	15	2	86	1.55	18900	1.4	7	7	2	I