“PROGNOSTIC ACCURACY OF qSOFA SCORE COMPARED TO SOFA SCORE AMONG PATIENTS WITH SEPSIS”

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Dissertation submitted to the

Sri Devaraj Urs Academy of Higher Education and Research,

Tamaka, Kolar, Karnataka,

in partial fulfillment of the requirement for the degree of

DOCTOR OF MEDICINE (M.D.)

IN

GENERAL MEDICINE

Under The Guidance Of

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April- 2020

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ACKNOWLEDGEMENT

I thank the almighty for showering his blessings on me. I sincerely thank my respected teacher, **Dr. RAVEESHA A** for there step-by-step guidance and constant extended support with the timely advices which helped me for this study. I thank **Dr SRINIVASA S V,** Department of **MEDICINE**, for his constant guidance and advices. I thank **Dr PRABHAKAR K,** Department of **MEDICINE**, for his constant support and advices. To all my teachers throughout my life for having made me what I am today. My deep felt gratitude to my dear parents, **B H THIPPESWAMY & R N SUNANDA ,** and my sisters, SOWMYA and SUMA my brother in laws GOPAL and BHARATH whose countless sacrifices and blessings have made me who I am today. My heartfelt thanks to my Nephew MOKSHITH my niece KRITHVI and LIRISHA for the love and support. I am also thankful to my friend's, fellow postgraduate colleagues, seniors, juniors for their constant motivation and countless help. Last but not least, I thank all my patients involved in this study, without whose co-operation, this study would not have been possible.

Dr. HAMSA B T

ABSTRACT

Background & objective: Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Sepsis and septic shock are major healthcare problems, affecting millions of people around the world each year, and killing as many as one in four. According to Sepsis-3, criteria to diagnose sepsis is solely based on the change in SOFA score by 2 or more points. SOFA score consists of 6 variables, which include 2 clinical parameters and 4 laboratory parameters. In developing countries like India, with limited resource settings across the country, lack of availability of laboratory parameters makes prognostication of sepsis early becomes difficult according to SOFA score. Surviving sepsis campaign introduced a newer scoring system, the QSOFA score which uses only clinical parameters to prognosticate sepsis bed side and at the earliest. Present study in evaluating the QSOFA score compared to SOFA score as a predictor of morbidity and mortality in sepsis assumes more importance in lights of early identification and prognostication of sepsis in resourse poor settings

Materials & methods: This study was a comparative cross-sectional study conducted in R.L. Jalappa hospital among 150 individuals divided into two groups. Assessment of SOFA and QSOFA score was done and compared its significance in predicting mortality and morbidity like need for ventilatory support, inotropic support, renal replacement therapy and length of ICU stay.

Results: There were 87 males and 63 females in this study. The mean age was 51.66 years .Most common etiology for sepsis was lower respiratory tract infection. Mortality rate in the study was38.7%. The initial QSOFA score of 1,2 and 3 had mortality rate of 5.2%, 24.1% and 70.7% respectively. Initial SOFA score of <4, 4-8 and >8 had mortality rate of 5.2%, 37.9% and 56.9% respectively. The mean SOFA

score had statistically significant correlation (P value <0.001) with respect to assessment of ARDS and subsequent ventilator support whereas the mean QSOFA score had a statistically significant relation in predicting need for ventilator support, vasopressor support. Both the scores had statistically insignificant correlation with respect to assessment of AKI and need for haemodialysis and in predicting the probable length of ICU care

Conclusion: Both QSOFA score and SOFA score demonstrated fair to good accuracy for predicting in-hospital mortality when applied to patients with severe sepsis. The QSOFA scoring system can aid the physicians in early referral to health care centre, in admitting patients to ICU, monitoring the clinical course, assessment of organ dysfunction, predicting mortality, and for transferring patients out from the ICU and thus in proper utilization of ICU resources in developing countries, where the resources are limited.

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LIST OF ABBREVIATIONS

ACCP/SCCM	→	American College of Chest Physicians/Society of Critical
AKI	→	Acute Kidney Injury
APS	→	Acute Physiology Score
ARDS	→	Acute Respiratory Distress Syndrome
AUC	→	Area Under Curve
BP	→	Blood Pressure
CABG	→	Coronary Artery Bypass Graft
CM	→	Care Medicine
COPD	→	Chronic Obstructive Pulmonary Disease
DAMPs	→	Damage Associated Molecular Patterns
DIC	→	Disseminated Intravascular Coagulation
ECG	→	Electrocardiography
Ei	→	Expected Frequency
ELISA	→	Enzyme Linked Immunosorbent Assay
HIV	→	Human Immunodeficiency Virus
ICU	→	Intensive Care Unit
IHD	→	Ischemic Heart Disease
ITU	→	Intensive Therapy Unit
LODS	→	Logistic Organ Dysfunction Score
LVH	→	Left Ventricular Hypertrophy
MAT	→	Microscopic Agglutination Test
MODS	→	Multiple Organ Dysfunction Syndrome
MPM	→	Mortality Probability Model
NMUAF	→	Non-malarial Acute Undifferentiated Fever
Oi	→	Observed Frequency
OSF	→	Organ System Failure
PAMPs	→	Pathogen Associated molecular Patterns
PARs	→	Protease Activated Receptors
PRR	→	Pattern Recognition Receptors
PaO_2/FiO_2	→	Partial pressure of oxygen/fraction of inspired oxygen
pH	→	Power of Hydrogen
QBC	→	Quantified Buffy Coat
ROC	→	Receiver Operating Characteristic

SAPS	→	Simplified Acute Physiology Score
SD	→	Standard Deviation
SIRS	→	Systemic Inflammatory Response Syndrome
SOFA	→	Sequential Organ Failure Assessment
QSOFA	→	Quick Sequential Organ Failure Assessment
TISS	→	Therapeutic Intervention Scoring System
TLC	→	Total Leucocyte Count

INTRODUCTION

INTRODUCTION

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection.[1,2] Sepsis is a major healthcare problem, causing mortality among one in four and often even more. Similar to acute coronary syndrome, stroke and polytrauma , early identification and early initiation of goal directed management in the initial hours of sepsis improves outcome.[1,3]

Sepsis currently is the tenth most common cause of mortality in the United States and one of the most common causes of mortality in the non-coronary intensive care units.[4,5]

Sepsis is a syndrome of physiologic, pathologic, and biochemical abnormalities induced by infection.[1] 28 day mortality and Hospital mortality of severe sepsis in India are 57.6% and 59.3%[6] respectively. There is an elevated awareness regarding long term physical, psychological, and cognitive disabilities in patients who survive sepsis.[6]

The diagnosis as sepsis depends on overt symptoms of systemic illness causing a change in the vitals of patient and aslo indication of infection through microbial cultures and serology. While our understanding of the complex pathophysiologic alterations that occur in septic shock has increased greatly as a result of recent clinical and preclinical studies, mortality associated with the disorder remains unacceptably high.[5]

Septic is the most common cause for hospitalization in the worldwide, patients often hospitalized for prolonged periods of up to 2-3 weeks.[7] Despite the use of appropriate antimicrobial therapy and advanced supportive care, mortality in patients with sepsis has remained high since the past decade.[8,9] Elderly subjects are especially

vulnerable population and are susceptible to a wide array of infectious diseases. Estimations suggest that the global burden of sepsis and septic shock among elderly population is expected to be on the rise in the forthcoming years.[10]

Cultures and serology results will be obtained only after 24 to 72 hours. In the initial hours of sepsis which will determine the outcome and prognosis of sepsis patients, Physician should depend on clinical findings and the demographic data framed in the locality to help in initial provisional diagnosis and further management of patients. Hence various guidelines propose the use of early empirical broad-spectrum antibiotics that will cover all the likely pathogens, and also supportive care, early recognition and treatment of complications, and intensive monitoring to prevent progression of organ dysfunction.[3] In more than quarter of the patients, aetiology is never determined even till death or discharge.[11]

In India, majority of ICU burden due to sepsis is mainly attributed to multi organ dysfunction caused by various tropical infections. Majority of patients present with fever associated features like myalgia, arthralgia, icterus, rash, or acute encephalitic syndrome.[12,13] Due to varied presentation, multi organ involvement and lack of proposed clinical diagnostic and prognostic criteria these tropical infections often remain undiagnosed. [14]

Scoring systems for use in the critical care patients have been developed from the past 30 years. They aid in prognostication of illness and a probability of in-hospital mortality. Use of these prognostic models helps in providing information to physicians when counseling about patient management plan and prognosis with the patient's care takers.

In June 2016, Third international consensus definition for sepsis and septic shock was proposed to define the patient definitions and guidelines for diagnosis of

sepsis based on the advances and modifications made into epidemiology, pathobiology of sepsis and its management. According to Sepsis-3 criteria the diagnosis of sepsis is mainly based on the change in SOFA score by 2 or more points consequent to the infection.[1] SOFA score consists of 6 variables, which include 2 clinical parameters and 4 laboratory values. In developing countries like India, with limited resource settings across the country, where rural population encounter primary care centers initially, lack of availability of laboratory facilities makes early prognostication of sepsis difficult according to SOFA score. Surviving sepsis campaign has also introduced a newer scoring system, the QSOFA score which uses clinical parameters alone to prognosticate sepsis bed side and at the earliest. QSOFA not only directs for early intensive management but also to take decisions regarding early referral to a tertiary care center from resource poor settings.[1,15] Present study in evaluating the QSOFA score as prognostic marker in patients with sepsis when compared to SOFA score assumes more importance in lights of early identification and prognostication in resource poor settings.

OBJECTIVES

OBJECTIVES OF THE STUDY

1) To calculate the SOFA score in patients with sepsis
2) To calculate the qSOFA score in patients with sepsis
3) To compare the above two scores with prognosis among subjects with sepsis.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORY OF SEPSIS

Sepsis is one of the oldest and most elusive syndromes in medicine. The word Sepsis is derived from the Greek sepsi meaning "make rotten", Hippocrates (460-370 BC) first coined the term sepsis to describe the unpleasant process of organic matter putrefaction.[16] Hippocrates claimed that sepsis is a process by which flesh rots, swamps generate foul airs, and wounds fester.[17]

Avicenna, the great Persian physician/scientist/philosopher, noted the frequent coincidence of blood putrefaction, known today as septicaemia, and fever in the aftermath of surgery.[18] In centuries that followed witnessed important discoveries linking germs to a varied disorders including sepsis. The germ theory of disease failed to explain the pathogenesis of sepsis since many patients succumbed to it despite successful eradication of the microbial agent. Hence, the host response to the germ, and not the germ per se, was proposed to be responsible for the pathogenesis of sepsis.[19]

Definition of sepsis

In 1914, Hugo Schottmuller in Germany introduced the modern definition of sepsis: Sepsis is present if a focus has developed from which pathogenic bacteria constantly or periodically invade the blood stream which lead to subjective and objective symptom.[20] One of the early attempts to establish a set of clinical parameters to define patients with severe sepsis came in 1989 when Roger Bone and his colleagues proposed the term "sepsis syndrome".[21]

In 1991, sepsis was defined by ICS panel defined as a systemic inflammatory response to infection, noting that sepsis can arise in response to multiple infectious

causes and that septicaemia was neither a necessary condition nor a helpful term. Instead, the consensus panel codified the term "severe sepsis" to describe instances where sepsis is complicated by acute multi organ dysfunction, and they codified "septic shock" as sepsis complicated by either hypotension that is refractory to fluid resuscitation or by hyper lactemia.[22]

Multi organ dysfunction syndrome is the presence of multiple altered organ function in a patient who is acutely ill such that without intervention homeostasis cannot be maintained. Primary MODS is the organ dysfunction which occurs early due to the direct result of a well-defined insult and can be directly ascribed to the insult itself. MODS that develops as a consequence of a host response is secondary MODS and is identified within the context of SIRS.[22]

In 2001, a second consensus panel endorsed most of these concepts, with the warning signs of a systemic inflammatory response, which include tachycardia or an elevated white-cell count, occur in many infectious and non-infectious conditions and therefore are not helpful in differentiating sepsis from other conditions.[23] Thus, "severe sepsis" and "sepsis" are sometimes used interchangeably to explain the syndrome of acute organ dysfunction due to infection. SIRS criteria were indeed too sensitive and non-specific[24] and that, in preference to the SIRS criteria, it was suggested that an expanded list of signs and symptoms must be used to reflect the clinical response to infection in sepsis.[23]

Definitions of infection and sepsis proposed during 2001 International Sepsis Definitions Conference[23]

Infection: Infection is a pathologic process which is caused by the invasion of a initially sterile tissue or body fluid by a pathogenic microorganism.

Sepsis: It is the Presence of infection, documented or strongly suspected, with a

systemic inflammatory response, as indicated by the presence of few of the features listed below.

Severe sepsis: It is the organ dysfunction complicated by sepsis.

Septic shock: It is the acute circulatory failure which is complicated by severe sepsis, characterized by persistent arterial hypotension, despite initial adequate volume resuscitation, and unexplained by other causes.

Proposed change from SIRS to a longer list of clinical findings for the diagnosis of sepsis.

Sepsis is suspected or documented infection with presence of some of the enlisted variables

General variables

- Fever (core temperature >38°C)
- Hypothermia (core temperature <36°C)
- Heart rate>90 beats/min or >2 Standard Deviations (SD) above normal range for age.
- Tachypnoea
- Altered mental status.
- Oedema or positive fluid balance (>20 ml/kg over 1 day).
- Hyperglycaemia (blood glucose level >120 mg/dl) in non-diabetics.

Inflammatory variables

- Leucocytosis: Leucocyte count >12,000/µL.
- Leukopenia: Leucocyte count <4000/µL.
- Normal Leucocyte count with >10 per centimetre band forms.
- Plasma C-reactive protein >2 SD above normal value.
- Plasma procalcitonin>2 SD above normal value.

Organ dysfunction variables

- Arterial hypoxemia (PaO_2/ FiO_2) <300.
- Acute oliguria: Urine output <0.5 ml/Kg/h for at least 2 hours.
- Creatinine >2.0 mg/dl.
- Coagulation defects: International Normalized Ratio (INR)>1.5 or Activated Partial Thromboplastin Time (APTT) > 60 seconds.
- Thrombocytopenia (platelet count <100,000/mL).
- Hyperbilirunemia (Serum total bilirubin>2.0 mg/dl).

Tissue perfusion variables

- Hyperlactatemia (>2mmol/L)
- Decreased capillary refill

Hemodynamic variables

- Mean arterial pressure <70mmHg
- Mixed venous oxygen saturation >70%
- Cardiac index >3.5 l min/m^2
- Organ dysfunction parameters

PIRO Model

A new concept was proposed during 2001 International Sepsis Definitions Conference that sepsis is mainly a heterogeneous condition and that it may be possible to explain sepsis based of four characteristics in the same way that cancer can be elaborated on the basis of the TMN system[22].Using a variation of the TNM approach, they developed a classification scheme for sepsis - called PIRO - that stratify patients based on their predisposing conditions, the nature and extent of the

insult(in the case of sepsis, infection), the nature and extent of the host response, and degree of concomitant organ dysfunction.

P: Predisposing Factors

Innate: Deficiencies of immune response genes and genetic polymorphisms affecting innate immune response, coagulation system, complement receptors, Toll-like receptors and intracellular signaling.

Acquired: Burns, trauma, acquired immune deficiencies.

I: Infection

Site, quantity, intrinsic virulence, and local vs. systemic infection caused by specific microbial pathogens.

R: Response

Differential responses based on hyper responsiveness vs. hypo responsiveness immunosuppression; Response modifiers such as alcohol, age, sex, nutritional status, diabetes, other preexisting diseases, and physiologic status of host.

O: Organ dysfunction

Number, pattern, and severity of organ dysfunction in response to systemic infection, primary vs. secondary organ injury; and organ injury due to pre-existing organ dysfunction vs. sepsis.

PIRO Model is now more of a framework for research than as a system that has immediate clinical application. Much work is needed to characterize those factors within each domain that affect prognosis and response to therapy.[24]

SIRS and MODS are not diseases or syndromes, but concepts. The four criteria that define SIRS are non-specific manifestations of physiologic severity, rather than distinctive manifestations of a disease process. [26]

However, these syndromes are characterized considering that shared biologic mechanisms may allow the development of effective treatment for different diseases.

Challenge lies in characterizing common pathologic processes for different diseases.[26]

Table 1: The PIRO system for staging sepsis [22]

Domain	Present	Future	Rationale
Predisposition	Premorbid illness with reduced probability of short tem survival. Cultural or religious beliefs, age, gender	Genetic polymorphisms in components of inflammatory response (e.g. Tlr, TNF, IL-1, CD14); Enhanced understanding of specific interactions between pathogens and host diseases	In the present, premorbid factors impact on the potential attributable morbidity and mortality of an acute insult; deleterious consequences of insult heavily dependent of genetic predisposition (future)
Insult (Infection)	Culture and sensitivity of infecting pathogens; detection of disease amenable to source control	Assay of microbial products (LPS, mannan, bacterial DNA); gene transcript profiles	Specific therapies directed against inciting insult require demonstration and characterization of that insult
Response	SIRS, other signs of sepsis, shock, CRP	Non-specific markers of activated inflammation (e.g. PCT or IL-6) or impaired host responsiveness (e.g. HLA-DR); specific detection of target of therapy (e.g. Protein C, TNF, PAF)	Both mortality risk and potential to respond to therapy vary with non-specific measures of disease severity (e.g. shock); specific mediator-targeted therapy is predicated on presence and activity of mediator
Organ Dysfunction	Organ dysfunction as number of failing organs or composite score (e.g. MODS, SOFA, LODS, PEMOD, PELOD)	Dynamic measures of cellular response to insult – apoptosis, cytopathic hypoxia, cell stress	Response to pre-emptive therapy (e.g. targeting micro-organism or early mediator) not possible if damage already present; therapies targeting the injurious cellular process require that it be present

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3) [1]

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. This new definition emphasizes the primacy of the no homeostatic response by the host to infection, the potential lethality that is considerably in excess of a straightforward infection, and the necessity for urgent recognition.

- Organ dysfunction can be assessed by an acute change in total SOFA score-2 points following infection.

- The initial SOFA score can be taken as zero in patients who do not have pre-existing organ dysfunction.
- qSOFAscore-2 reflects an overall mortality risk of approximately 10% in patients suspected to have infection. Even patients presenting with modest dysfunction can deteriorate further, emphasizing the seriousness and the need for prompt and appropriate intervention at the earliest, if not already being instituted. Early referral, if appropriate intensive care facilities are not available.
- Patients with suspected infection can be assessed bed side with QSOFA score to know those who are likely to have a prolonged ICU stay or to die in the hospital
- Patients with septic shock can be identified as those with persisting arterial hypotension despite initial fluid resuscitation and who may require vasopressors to have MAP-65mmHg and having a serum lactate level>2 m mol/L (18mg/dl) despite adequate volume resuscitation.
- "Sepsis is a medical emergency" a concept that is paramount in the management of sepsis. As with acute myocardial infarction and stroke, prompt early identification and appropriate immediate management in the early hours after development of sepsis improves outcomes. "SSC: Guidelines for management of sepsis:2016" has developed a revised "hour 1 bundle" uplifting the need for urgent assessment and treatment, including initial fluid resuscitation while pursuing source control, obtaining further laboratory results. The main change in the revised SCC bundle is the 3-h and 6-h bundles has been clubbed into a single "hour-1 bundle" with sole intention of beginning resuscitation and management immediately.[3,4]

Hour 1 bundle includes:

- Measure lactate levels. Re measure if initial lactate >2mmol/l
- Obtain blood cultures before antibiotic administration
- Administer broad spectrum antibiotics
- Begin early administration of crystalloid at 30ml/kg for low SBP or if lactate level > 4mmol/l
- Administer vasopressors if patient is hypotensive during or after fluid resuscitation to maintain MAP of > 65mmHg.

Epidemiology of sepsis

Sepsis, a syndrome of physiologic, pathologic, and biochemical abnormalities induced by infection, remains as a major public health concern, accounting for about $20 billion (5.2%) of total US hospital costs in 2011.[27] incidence of sepsis mainly depends on how acute organ dysfunction is being defined and also the sources that are being studied. The reported incidence of sepsis is increasing[28,29,30]

Factors underlying the rising incidence of sepsis:

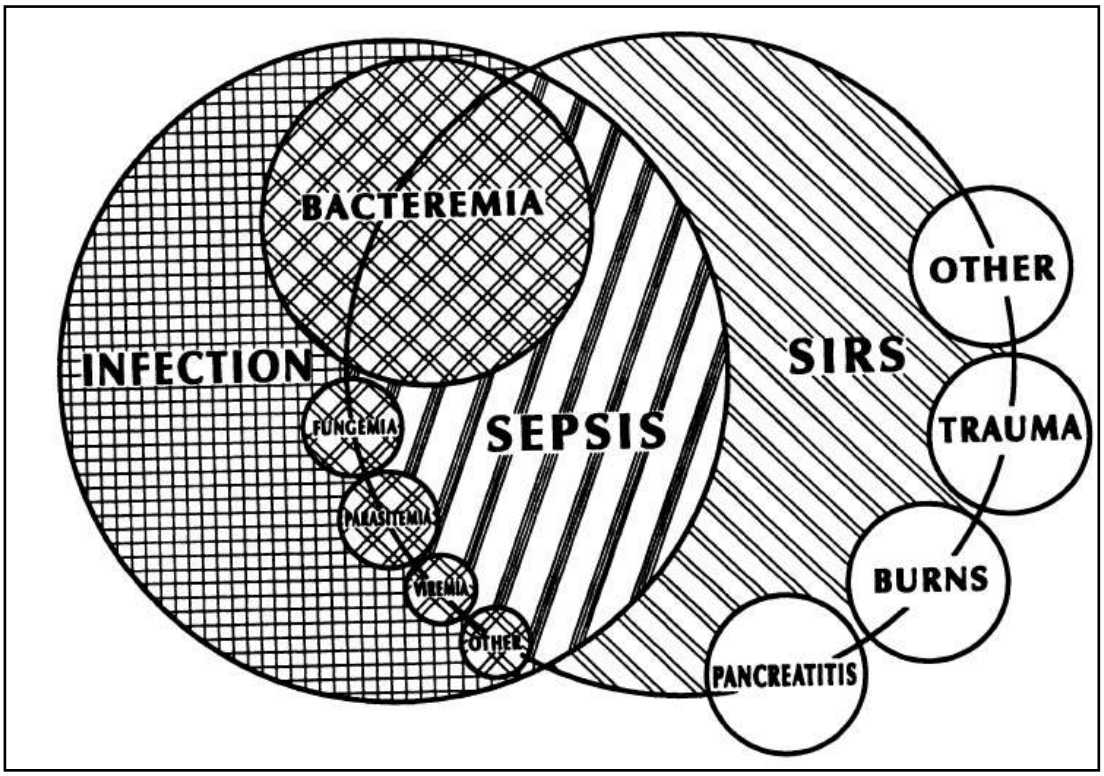
- Increasing patient age [31]
- Increase in the use of immunosuppressive therapy
- Increase in the incidence of comorbidities and concomitant illness
- Increase use of invasive procedures for diagnosis and treatment
- Emergence of antibiotic resistant organisms

Although the true incidence is unknown, conservative estimates show that sepsis is the leading cause of mortality and critical illness worldwide.[31,32] Furthermore, there is increasing evidence that patients who survive sepsis often have

long-term physical, psychological, and cognitive disabilities with significant health care and social implications.[8]

In high income countries, sepsis is a significant public health burden, whereas in developing low and middle income countries its burden remains even higher due to increased incidence of infectious disease and communicable diseases. Case fatality rates are higher in these countries when compared to developed countries.

ETIOLOGY[32]



Sepsis may be a response to any class of microorganism. In fact, blood cultures yield bacteria or fungi in only -20-40% of cases of sepsis and 40-70% of cases of severe sepsis. Individual gram-negative or gram-positive bacteria account for 70% of these isolates; the remainder are fungi or a mixture of microorganisms.[33]

The etiology of sepsis has been determined by medical advances that has led to increased use of invasive devices and antibiotics.[34] Historically, gram-negative

rods were the predominant etiologic agent; however in recent years sepsis by gram positive cocci and fungal organisms is in the rise.[35]

1. Gram-negative bacteria-Enterobacteraceae , pseudomonas, Haemophilus species
2. Gram-positive bacteria-Staphylococcus aureus, coagulase-negative staphylococci, enterococci, Streptococcus pneumonae , other streptococci.
3. Fungi
4. Polymicrobial
5. Classic pathogens -Neisseria meningitides, S.pneumoniae, Haemophilus influenzae, and Streptococcus pyogenes

Table 2: Micro organisms involved in episodes severe sepsis

Micro organisms	Episodes with Bloodstream Infection, % (n= 436)	Episode with Documented Infection but No Blood stream Infection, % (n= 430)	Total Episodes,% (n= 866)
Gram-ve organism	35	44	40
Gram +ve organism	40	24	31
Fungi	7	5	6
Poly-microbial	11	21	16

The most common foci of infection include respiratory and urinary tract. The respiratory and genitourinary systems combined are the source in 65.3% of patients with sepsis aged ≥65 years, vs. only 49.3% in those younger patients. Whereas younger patients are at increased risk of gastrointestinal sources, skin and soft tissue sources compared to older adults.[36]

We are in a way unfortunate in that we see merely the usual causes of sepsis and MODS encountered in West, but also certain infections peculiar to tropical and developing countries.

These infections to which we are exposed are not just related to geography or climate, but are significantly related to environmental and socioeconomic conditions that prevail in involved part of the world.[37] Infections like fulminant falciparum infections, severe leptospiral infections and hemorrhagic fevers sometimes cause life threatening organ dysfunction and have several overlapping features.

Pathophysiology.

Sepsis is triggered most often by bacteria or fungi that do not ordinarily cause systemic disease in immunocompetent hosts. To survive within the human body, these microbes often exploit acquired deficiencies in host defenses, indwelling catheters or other foreign matter, or obstructed fluid drainage conduits.

Host Response to infection:

Earlier days it was assumed that the development of clinical features in sepsis is mainly due to the overly exuberant inflammation, whereas recent evidences conclude that the initial inflammatory response will give way for the development of compensatory anti-inflammatory response. It has become apparent that that initial infection will trigger more complex and variable host response. Hence host response will include both pro-inflammatory and anti-inflammatory immunosuppressive response. The extent of these two reactions are determined by both the host factors (age, comorbid illness, genetic characteristics, medications) and the pathogen factors (virulence and microbial load). Host response is mainly aimed at initiating tissue repair following pathogen invasion.

Pathogens express molecular patterns on their surface which are recognized by the pattern recognition receptors(PRR)which are present over host cell surface, these receptors mainly include the toll-like receptors (TLRs) and the C Type lectin receptors (CLRs). Collateral tissue damage and necrotic cell death occurs as a consequence of exaggerated inflammation, this results in release of danger molecules which will perpetuate inflammation.

The innate immunity response is the first line of defense towards invading pathogens. Recognition of microbe and its components, activation of phagocytosis, activation of complement system and coagulation cascade and also production of acute phase reactants. Adaptive immunity includes responses of cell-mediated and humoral immunity.[38]

Innate immune response

The natural mechanical barriers to pathogen invasion are formed by the skin externally and by mucous membranes internally. These mechanical barriers are in co-operation with the commensal flora. In the hospital, patient's indwelling catheters and intravenous cannulas must be considered as potential sources of infection.[39] various structural components of the pathogen may be involved in the pathogenesis of sepsis, detection of which may aid in development of therapeutic targets. Endotoxin and exotoxin produced by bacteria trigger the immune cells via molecular patterns expressed over pathogen.[40]

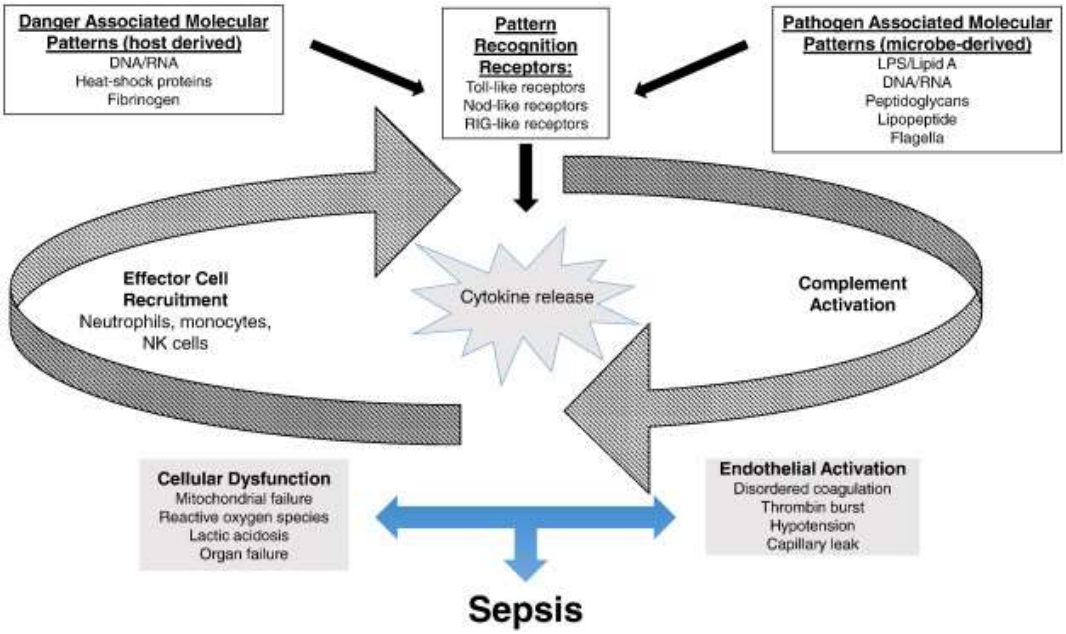


Figure 1: Role of innate immune response in sepsis [41]

The invading pathogen will activate the immune cells by interaction with pattern recognition receptors (PRR).[42]

four main classes of PRR include- toll-like receptor, C Type lectin receptors, retinoic acid inducible gene 1-like receptors and the nucleotide-binding oligomerization domain like receptors. These receptors will recognize unique cell-wall molecules present over microbes known as pathogen-associated molecular patterns (PAMPs). This will result in initiation of innate immunity by up regulation of inflammatory gene transcription. The same molecular receptors also sense the endogenous molecules which are released from the damaged cells, called as damage associated molecular patterns (DAMPs). Macrophages and monocytes secrete pro inflammatory cytokines. Adhesion molecules on endothelium are produced by activated Neutrophils and endothelial cells, these adhesion molecules help to kill the pathogens, but also cause damage to the endothelium. Activated Macrophages release VEGF-like

mediators, which will increase the vascular permeability and contribute to coagulation and inflammatory processes.[42,43]

Adaptive immune response

Following the initial pathogen invasion and host-pathogen interaction there will be activation of adaptive immune response, this immune response coordinates the defense responses involving both the humoral and cellular immune response . The humoral immune response is mediated by the antibodies which are produced by plasma cells which belong to B cell lineage, whereas the cellular immune effectors are the T Lymphocytes.[44] Activated phagocytes destroy the pathogen with the help of complement activation or by recognition by antibodies.[45]

Effector T cells are mainly secreted by Thymus in early life, maintained throughout life by the peripheral lymphoid organs. Upon exposure to antigen there will be activation of macrophages, natural killer cells, cytotoxic T-lymphocytes and various inflammatory cytokines. Viruses and the intracellular bacteria are mainly targeted and destroyed by Cytotoxic T-lymphocytes (CD8). The Helper T cells (CD4) differentiate into type 1 helper T-cells (Th1) and type 2 helper T-cells (Th2) and secrete cytokines. Th1 secretes pro-inflammatory cytokines which trigger inflammation whereas the Th2 cells secrete anti-inflammatory cytokines which tries to curtail inflammation. Pro inflammatory mediators mainly include (e.g. interleukin-1 (IL-1), interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) and anti-inflammatory mediators mainly include (e.g. interleukin-4 (IL-4) and interleukin-10 (IL-10)) [46,47].

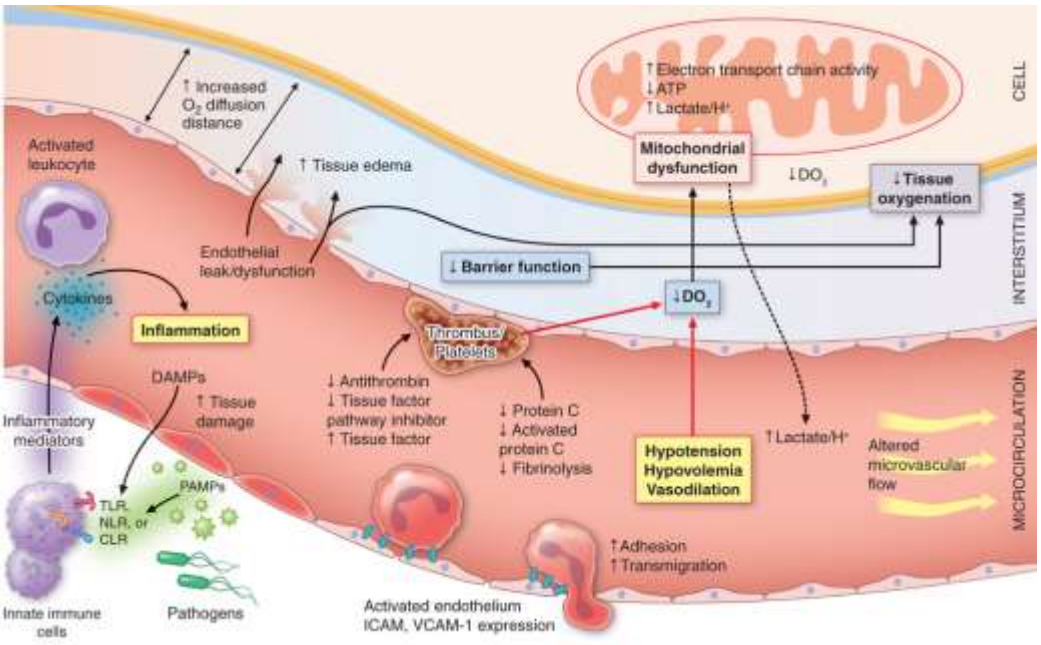


Figure 2 : Pathophysiology of Sepsis [42]

Coagulation abnormalities

Altered coagulation profile invariably accompanies severe sepsis in most patients. This frequently leads to disseminated intravascular coagulation (DIC). Excessive fibrin deposition is mainly driven by the coagulation system via activated Tissue factor which is a trans-membrane glycoprotein and by impaired anticoagulation mechanisms which include protein C system and the Antithrombin. Molecular link between the coagulation pathway and the inflammatory cascade is formed by the Protease- Activated Receptors (PARs). There are four subtypes of PARs, in sepsis PAR1 has been implicated. When PAR1 is activated by low dose of thrombin or by Protein C it exerts cyto-protective effect, but when this PAR1 is activated by high doses of thrombin it mainly exerts disruptive effects on the endothelium [42].

There will be activation of coagulation as well as concurrent impairment of anticoagulation system due to the deceased activity of endogenous anticoagulant system which is mediated by the activated protein C, anti-thrombin and the inhibitor of tissue factor pathway, this will ultimately lead to the formation of micro vascular thrombosis in sepsis, this micro vascular thrombosis is further augmented by the impaired fibrinolysis via excessive release of PAR1. Dying neutrophils release Neutrophil Extracellular Traps (NETs) which will further facilitate thrombus formation. Thrombus formation will ultimately lead to tissue hypo perfusion which will be further aggravated by the vasodilatation and hypotension in severe sepsis.[42]

Complement cascade.

Complement cascade also gets activated in par with the coagulation system, both the pathways mutually interact at several stages and aggravate the inflammatory response. [47] The major function of the activated complement cascade is to defend against the pyogenic bacterial infections, it acts as a bridge between the adaptive and the innate immune system. Activated complement pathways plays a role in elimination of products of inflammatory injury and to clear the immune complexes.

Complement components in the circulation are activated by three pathways:

1. The classical pathway which is initiated by the binding of complement component C1q to the antigen antibody complex,
2. The lectin pathway which is initiated by the binding of mannose-binding lectin that are present over the bacterial cell wall
3. The alternative pathway which is initiated after exposure to invading pathogens surface molecules

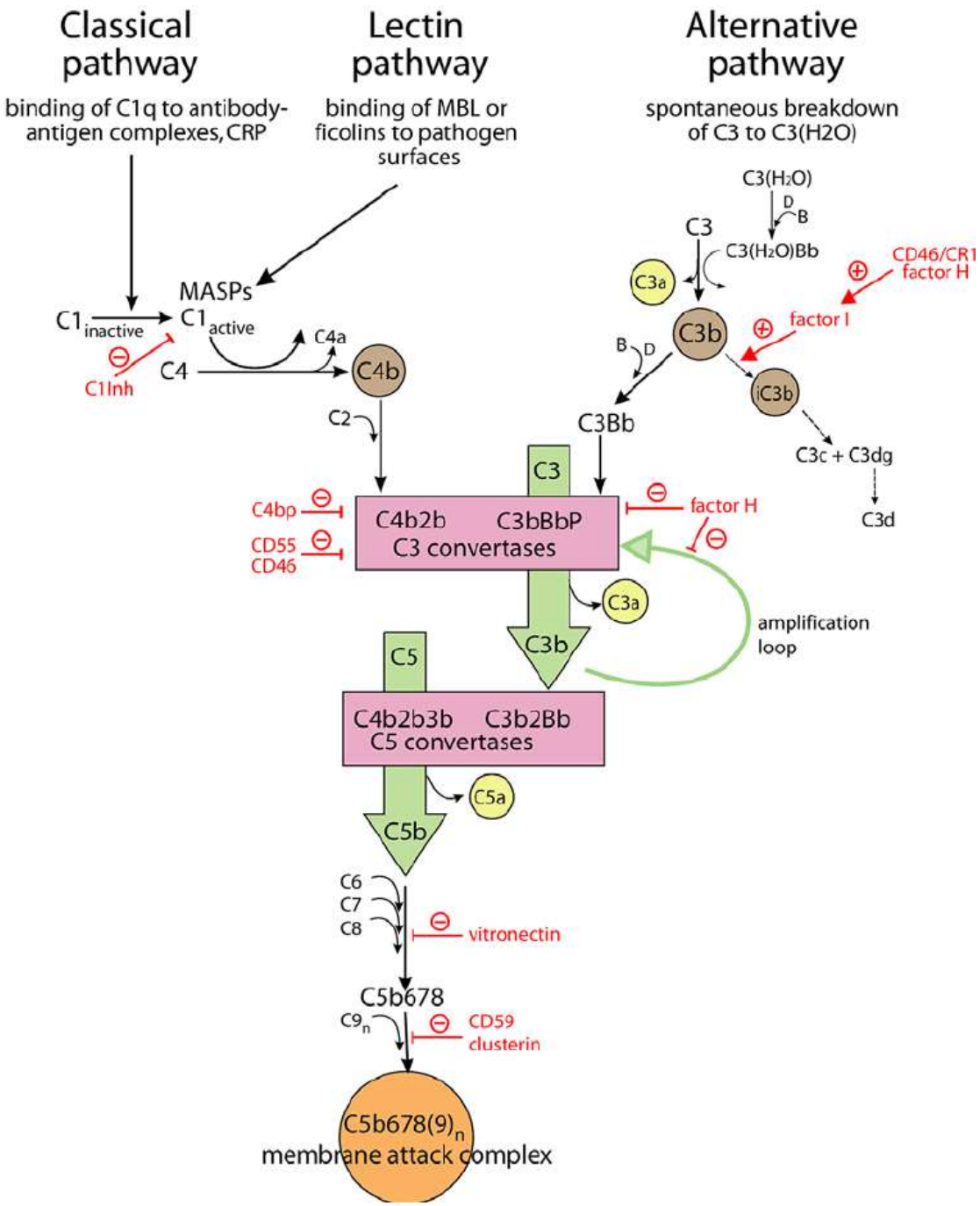


Figure 3 : The Complement Cascade [48]

The complement pathway convertases which mainly include the C3 convertase and the C5 convertase eliminate the pathogen by aiding opsonisation and phagocytosis via the macrophages and the neutrophils.[47]

Anti-inflammatory mechanisms and immunosuppression

The potential harmful effects of the pro-inflammatory response is attenuated by the immune system via the humoral, cellular and the neutrally mediated mechanisms. Tissue repair can be promoted by phagocytes by switching over to the anti-inflammatory phenotype. The regulatory T cells and the myeloid derived suppressor cells will further curtail the inflammation. Via the Neuro-inflammatory reflex the Vagus nerve carries the afferents to the brainstem from where the information is relayed and via the efferent vagus nerve the splenic nerve in the celiac plexus is activated, this results in release of nor-epinephrine in the spleen and acetyl-choline by subset of CD4+ T cells. This acetyl-choline will target the α7 cholinergic receptors which are present over the activated macrophages and leads to the suppression of pro-inflammatory cytokine release.

There is an evidence of immunosuppression among patients who depend on critical care despite surviving through early sepsis this in part is reflected by the decreased expression of KLA-DR present over the myeloid cells. These subset of patients will usually have an ongoing foci of infection despite extensive antimicrobial therapy. Recent post mortem studies on splenocytes in patients who succumbed due to sepsis in ICU have shown strong functional impairments. Apart from splenocytes the lungs also showed similar evidence, both these organs had an increased expression of inhibitory receptor ligands of T cells.[42]

Endothelium and inflammation

Numerous pro-inflammatory and anti-inflammatory mediators are being synthesized by the vascular endothelial cells. Vascular endothelial cells play a vital role in maintaining and regulating the vascular tone, activation of platelets and the leucocytes, they are involved in coagulation cascade and angiogenesis. They also produce various proteins which will alter the permeability of the vessel leading to leakage of fluid and larger molecules like antigen-antibody complexes. Chemotaxis of neutrophils and monocytes occurs by the adhesion of microbial antigen to the endothelial surface, increase in the permeability leads to the migration of neutrophils to the surrounding injured site. Microbial antigens and the endotoxin promote activation of neutrophils and the release of pro-inflammatory mediators.[49]

SEPSIS AND ORGAN DYSFUNCTIONS

The pathologic mechanisms that underlie multiple organ dysfunction in patients with sepsis is only partly understood. Key factor involved is the impaired tissue oxygenation. Multiple other factors like hypotension, decrease in red cell deformability and micro-vascular thrombosis also contribute to decreased oxygen delivery and thus organ dysfunction in sepsis. Subcutaneous edema and body-cavity edema in sepsis patients is mainly attributed to vascular endothelial dysfunction and loss of its barrier integrity.[42,50] Impaired cellular oxygen is also attributed to oxidative stress which causes damage to the mitochondria.[51]

DEFINING ORGAN DYSFUNCTIONS [42]

Acute cardiac dysfunction : In patients with sepsis who do not have any evidence of acute coronary syndrome, pulmonary embolism, dysrrythmia or cardiac tamponad .

- Evidence of left or right ventricular failure
- Elevated ventricular filling pressure
- Low cardiac index (<2.2L/min/m^2)

Acute Respiratory failure:

- Requirement of ventilator support with a FiO_2 of >0.4
- ARDS if need for positive end expiratory pressure is ≥5cmH_20

Acute renal dysfunction:

- Serum creatinine level >2mg% or
- In a patient with prior kidney disease- Doubling of the admission Creatinine

Liver dysfunction :

- Total bilirubin level >2mg% and
- Tansaminases and lactate dehydrogenase levels at least twice the upper limit of normal.

Disseminated intravascular coagulation: evidence of spontaneous hemorrhage from two or more regions along with

- Platelet count of <50,000/cu.mm
- Elevated fibrin degradation products and
- Fibrinogen of <200%.

Neurological dysfunction : Glasgow Coma Scale (GCS) of < 7/15.

CLINICAL MANIFESATIONS AND ETIO-PATHOGENESIS:

ACUTE RENAL FAILURE AND SEPSIS

Acute kidney injury secondary to sepsis has been noted in >50% patients in the ICU. This is associated with an elevated risk of in-hospital mortality by six-to eight fold. Mechanism behind renal injury is poorly understood. Even in the absence of overt hypotension approximately 25% patients are prone to develop renal injury. Beyond mere organ ischemia Current hypothesis includes the combinations of widespread micro-circulatory abnormalities and inflammation. Clinical manifestations include oliguria, elevated blood urea levels and serum creatinine levels frequently requiring renal replacement therapy.[42,51]

RESPIRATORY SYSTEM AND SEPSIS

ARDS is associated mortality rate of 50% to 70% in the United States. The proposed pathology behind this is injury to the alveolar capillary endothelial cells and the type I Pneumocytes attributed to oxidative stress and free radical injury leading to accumulation of edema fluid in the alveoli and the interstitium. ARDS clinically presents as arterial hypoxemia and bilateral infiltrates on chest radiograph of non-cardiac origin within 7 days of sepsis.[52]

ARDS is graded based on the Berlin's scrore: which include. [42]

- Mild ARDS – PaO_2/FiO_2 of 201-300mmHg
- Moderate ARDS - PaO_2/FiO_2 of 101-200mmHg
- Severe ARDS - PaO_2/FiO_2 of <100mmHg

CARDIOVASCULAR SYSTEM IN SEPSIS

Two most frequently involved organs in sepsis are the heart and lungs. Cardiac compromise typically manifests as arterial hypotension. This further leads to organ organ dysfunction as well.[53] Factors responsible for hypotension mainly includes frank hypovolemia, diffuse capillary leakage leading to mal-distribution of blood flow. Decrease in systemic vascular resistance or depressed myocardium. Following initial fluid resuscitation, hypotension still persists requiring vasopressors. Studies have shown a reduction in ejection fraction up to 40% in sepsis.[54] The major molecules which are involved in producing cardiac depression in sepsis are TNF, IL-1β and NO.[55] The cardiac dysfunction findings include:

1. Reduced Ejection fraction
2. Elevated end diastolic and end systolic volumes of ventricles with maintained stroke volume
3. Increased heart rate
4. Decreased systemic vascular resistance

CENTRAL NERVOUS SYSTEM AND SEPSIS

Coma or delirium are the typical presentations of nervous system dysfunction in sepsis. There will be no focal lesions or electro-encephalogram findings usually suggesting a non-focal encephalopathy. The Pro-inflammatory response to sepsis manifests as delirium without any objective evidence of primary nervous system infection. Other neurological manifestations of prolonged sepsis are critical illness polyneuropathy and myopathy. The proposed mechanisms for nervous sytem dysfunction are disseminated micro abscesses via hematogenous route, multiple microscopic infarctions due to coagulation abnormality, oxidative injury and imblanace in neurotransmitters.[56]

SEPSIS INDUCED THROMBOCYTOPENIA

Hemophagocytosis of megakaryocytes and transient bone marrow suppression lead to decrease in platelet count in sepsis. The incidence of which is about 35-44%. A count of ≤100,000 is noted in 12-15% of patients.[57]

THE ENDOCRINE SYSTEM DURING SEPSIS

Elevated sugar levels and increase in insulin resistance has been encountered most commonly in sepsis. There is depressed production of Corticosteroids and vasopressin. Studies have shown optimal sugar control can confer survival benefit in sepsis.[58,59]

ADDITIONAL CLINICAL MANIFESTATIONS

The clinical presentation of the septic response are superimposed on patient's primary illness. Hyperventilation is often an initial feature of the septic response.[59]

Disorientation, delirium and other manifestations of encephalopathy may also develop early especially in the elderly and in individuals with preexisting neurologic impairment. Focal neurologic signs are uncommon, although preexisting focal deficits may become more prominent.[42, 59]

Hypotension and DIC predispose to acro cyanosis and ischemic necrosis of peripheral tissues, most commonly the digits. Cellulitis, pustules, bullae, or hemorrhagic lesions may develop when hematogenous bacteria or fungi seed the skin or underlying soft tissue.[42]

Gastrointestinal manifestations include nausea, gastro-enteritis presenting as emesis and diarrhea. Upper gastrointestinal bleeding secondary to stress ulceration. Obstructive jaundice secondary to cholestasis. Persistent arterial hypotension may lead to acute ischemic hepatitis or bowel necrosis secondary to ischemia.[59]

Differential Diagnosis

The following serious medical conditions may mimic sepsis:

- Cardiogenic shock
- Extensive myocardial infarction
- Saddle Pulmonary Embolism
- Major hemorrhage
- Hypo adrenal crisis
- Acute pancreatitis
- Diabetic ketoacidosis

TREATMENT[59,60]

1. Identification and removal of the septic foci
   - Removal of infected catheters or venous access devices
   - Identification and drainage of abscess
   - Debridement of infected tissue
2. Fluid resuscitation guided by vital signs (including central venous pressure) and urine output
3. Initiate vasoactive agents if needed.
4. Obtain antimicrobial cultures
5. Broad-spectrum antibiotics
6. Supportive management of other symptoms
   a. Oxygen, to keep saturations more than 90 mmHg
   b. Treatment of delirium, nausea, vomiting and pain.
   c. Intravenous Insulin for hyperglycemia
   d. Initiate prophylactic measures for venous thromboembolism and

gastrointestinal hemorrhage

e. Initiate lung protective ventilation strategies

Protocols and guidelines

The SCC guidelines has revised the initial 6 hours bundle for resuscitation and the initial 24 hours Management Bundle following hospital admission and the sepsis diagnosis into hour 1 bundle for immediate management of sepsis.[3,4] Mortality in patients with severe sepsis has decreased when these SSC guidelines or modified protocols have proposed initial goal directed therapy to prevent organ damage. The Spanish study was able to show prospectively how better compliance with the bundles decreased mortality.[61]

SCORING SYSTEM

The first ICU model of disease severity, the Therapeutic Intervention Scoring System (TISS), was proposed in 1974[62].25 years later a number of physiology based ICU scoring systems have developed to aid in assessment of in-hospital mortality rates. Scoring systems essentially consists of two parts:

- A severity score (generally the higher this is the more severe the condition).
- Calculated probability of mortality.[63]

VARIOUS SCORING SYSTEMS [15,64,65,66]

General important scores

- APACHE (Acute Physiology and Chronic Health Evaluation)
- MPM (Mortality Probability Model)
- LODS (Logistic Organ Dysfunction Score)
- SOFA (Sequential Organ Failure Assessment)
- SAPS (Simplified Acute Physiology Score)

- Multiple Organ Dysfunction Score
- QSOFA (Quick Sequential Organ Failure Assessment)

SOFA SCORE

SOFA is the most commonly used organ dysfunction assessment model. Most of the variables considered in these systems are easily available and obtained from critical care settings.

Initially Sepsis related organ failure assessment score, was proposed to estimate the organ dysfunction sepsis patients[67]. Further, it was renamed the sequential organ failure assessment because its utility was not restricted merely to sepsis. The SOFA score is a six-organ dysfunction/failure score measuring organ failure daily. Each organ is graded from 0-4 providing daily score of 0-24 points. SOFA score assessment initially can serve as prognostic indicator. Mean and highest SOFA scores are particularly useful predictors of outcome.[68,69.]

Table 3: SOFA score[70]

Sequential [Sepsis-Related] Organ Failure Assessment (SOFA) Score

System	0	1	2	3	4
Respiration PaO2/FIO2, mmHg (kPa)	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support
Coagulation Platelets, x10³/uL	≥150	<150	<100	<50	<20
Liver Bilirubin, mg/dL (umol/L)	<1.2 (20)	1.2 - 1.9 (20 - 32)	2.0 - 5.9 (33 - 101)	6.0 - 11.9 (102 - 204)	>12.0 (204)
Cardiovascular	MAP ≥70mmHg	MAP <70mmHg	Dopamine <5 or Dobutamine (any dose)	Dopamine 5.1 - 15 or Epinephrine ≤0.1 or Norepinephrine ≤0.1	Dopamine >15 or Epinephrine >0.1 or Norepinephrine >0.1
CNS GCS Score	15	13 - 14	10 -12	6 - 9	<6
Renal Creatinine, mg/dL (umol/L) Urine Output, mL/d	<1.2 (110)	1.2 - 1.9 (110 - 170)	2.0 - 3.4 (171 - 299)	3.5 - 4.9 (300 - 440) <500	>5.0 (440) <200

*Catecholamine Doses = ug/kg/min for at least 1hr

The prognosis in sepsis is also dependent on the patient's underlying health status, development of adverse consequences, organ dysfunctions and prevention of complication.

QSOFA SCORE :

The 2016 sepsis campaign proposed that the utility of SOFA score outside ICU and in areas with limited resources would be impractical, hence it introduced a new scoring system, the Quick SOFA Score, which had 3 clinical variables which can be assessed bedside, without any aid for laboratory or advanced assistance. The score includes 2 vital signs and a brief neurological evaluation. The score consists of 1 point for each of hypotension (SBP ≤100 mm Hg), tachypnea (respiratory rate ≥22/min), and altered mental status, positive score is considered as 2 or 3 points. [15]

Table 4: QSOFA score:[15]

CLINICAL PARAMETER	SCORE
SBP ≤ 100mmHg	1
Respiratory rate ≥ 22/min	1
Altered mental state	1

In developing countries, in areas with limited resources where appropriate laboratory facilities are unavailable, when patients contacts primary care initially, a quick estimation of severity of sepsis and the probable need for intensive care, can aid in early referral/ early intensive care with initial goal directed therapy. The 2018 sepsis hour 1 bundle stressed on early goal oriented therapy which can prevent organ dysfunction and thus have a good prognosis in sepsis . Hence there is need for studies that assess the significance of QSOFA score, which can guide in early identification of severity and prognosis of sepsis especially in developing countries.

REVIEW OF LITERATURE

1. Shannon M et al, conducted a study on prognostic accuracy of the QSOFA score for Mortality in patients with Infection, Thirty-eight studies were clubbed (*n* = 385 333). For mortality the qSOFA had a pooled sensitivity of 60.8% and a pooled specificity of 72.0%. The SIRS had a pooled sensitivity of 88.1% and a pooled specificity of (25.8%).This study concluded qSOFA can be rapidly scored bedside without laboratory assistance, and it will facilitate prompt identification of infection that poses a greater threat to life. If appropriate laboratory tests have not already been undertaken, this may prompt testing to identify biochemical organ dysfunction.[71]

2. S Todi, S Chatterjee and M Bhattacharyya conducted a study at AMRI Hospitals, Kolkata. Total of 1,344 sepsis patients were studied. There were no SIRS in 31.3% and SIRS without organ dysfunction in 51.6%. SIRS with organ dysfunction was found in 230 (17.1%) patients, of which 54 (23.5%) were not due to sepsis and 176 (76.5%) were due to sepsis. The incidence of severe sepsis was 13.1% of all admissions. The mean age of the study population was 54.9 years (SD 17.6), of which 67% were male. ITU mortality of all admissions was 13.9% and that of severe sepsis was 54.1%. [6]

3. Acharya SP, Pradhan B,Marhatta MN of Department of Anaesthesiology, Tribhuban University Teaching Hospital, Maharajgunj, Kathmandu, Nepal in their study Application of SOFA score in assessing outcome in SIRS concluded that the non survivors had high initial, mean and highest SOFA scores when compared to survivors. (p value = 0.002). Delta SOFA was not significantly associated with outcome. The initial SOFA score > 11 predicted a mortality of 90%. Similarly,

mean SOFA score of > 7 predicted a mortality of 73.9% and high SOFA score > 11 predicted a mortality of 87.5Thus mean, high and initial SOFA scores were helpful in predicting between the

survivors and the non-survivors[10].

4. Lauren J et al, Study on Prognostic accuracy of age-adapted SOFA, SIRS, PELOD-2, and qSOFA for predicting in-hospital mortality among patients with sepsis, Of 2594 paediatric ICU admissions due to infection, 151 (5.8%) children died, and 949/2594 (36.6%) patients died. A ≥ 2-point increase in each score was associated with a crude mortality increase from 3.1 to 6.8% for SIRS, from 1.9 to 7.6% for age-adapted SOFA, from 1.7 to 7.3% for PELOD-2, and from 3.9 to 8.1% for qSOFA ($p < 0.001$). The outcome discrimination was significantly higher for SOFA (adjusted AUROC 0.829; 0.791–0.868) and PELOD-2 (0.816; 0.777–0.854) than for qSOFA (0.739; 0.695–0.784) and SIRS (0.710; 0.664–0.756). This study concluded that the predictive value of qSOFA to identify patients with organ dysfunction was poor, and may not be of suficient clinical value to be used routinely as a screening tool for patients within the ICU.[72]

5. Flavio Lopes Ferreira, Daliana Peres Bota study concluded organ dysfunction assessment sequentially during the first few days of ICU admission serve as good predictor of prognosis. The mean and highest SOFA scores are particularly useful predictors of outcome. Independent of the initial score, within 48 hours an increase in SOFA score indicates high mortality of at least 50%[73].

METHODOLOGY

MATERIALS AND METHODS

A prospective observational study titled "PROGNOSTIC ACCURACY OF qSOFA SCORE COMPARED TO SOFA SCORE AMONG PATIENTS WITH SEPSIS" was done at Sri Devraj Urs Medical College attached to R L Jalappa Hospital , Tamaka, Kolar after obtaining the approval from the institutional Ethics Committee.

Study site: This study was conducted in the Department of General Medicine, R.L.Jalappa hospital and research centre.

Study population: This study was conducted in R.L.Jalappa hospital in patients of sepsis who fulfilled inclusion and exclusion criteria.

Study design: The current study was a prospective observational study

Sample Size:

The sample size for the study is estimated based on the difference in proportions in SOFA and Qsofa score in a study by Yutaka U, Hiroshio O, Satoshi G, Shigek K, Daizoh S, Toshihiko M, et al[2] to detect an effect size of 20% with 80% power, 95% confidence interval , the estimated sample size is 96. However 150 patients with sepsis were included in the study. [74]

Sample size estimation formula:

Sample size = $Z_{1-\alpha/2}^2$ p (1-p) / d^2

Here,
$Z_{1-\alpha/2}$ = is standard normal varaite (at 5% type 1 error ($p<0.05$) it is 1.96 and at 1% error
($p<0.01$) it is 2.58). As in majority of studies p values are considered significant below

0.05 hence 1.96 is used in formula
P = expected proportion in population based on previous studies or pilot studies.

d = absolute error or precision.

Sampling method: All the eligible subjects were recruited into the study consecutively by convenient sampling till the sample size is reached.

Study duration: The data collection for the study was done between November 2017 to September 2019 for a period of 2 years.

INCLUSION CRITERIA

- Patients above 18 years of age.
- Patients admitted to medicine department with sepsis. (According SCC-3 guidelines: that is patients with SOFA score of >2)

EXCLUSION CRITERIA

- Patients with pre-existing organ dysfunction prior to infection (chronic kidney disease, decompensated liver disease)
- Patients discontinuing treatment.

Ethical considerations: Study was approved by the institutional ethics committee. Written informed consent was taken from all the study subjects. The risks and benefits involved in the study and the voluntary nature of participation were explained to the

participants before obtaining consent. Confidentiality of the study participants was maintained.

Data collection tools: All the relevant parameters were documented in a structured study proforma.

The study requires investigations such as:

- Complete blood count
- Blood, Urine, Sputum culture
- CRP
- Renal function tests
- Liver function tests
- ABG
- Chest X-ray
- Serological tests

Methodology:

1. Patients admitted to medicine department with sepsis as per sepsis definitions (SOFA score > were taken up for the study.
2. Informed written consent was taken.
3. A detailed history was elicited from the patient or a reliable relative. The duration of onset and progress of the presenting symptoms were documented
4. A complete physical examination was done.
5. After an initial evaluation at admission the patient was followed up till discharge or death or a maximum period of 5 days.
6. The progress of the patient was assessed at regular intervals by the Sequential Organ Failure Assessment score and Quick Sequential Organ Failure Assessment score.
7. The need for supportive management was noted. Which included inotropic support, dialysis, ventilator support and ICU care.
8. The outcome of the patient in terms of morbidity (length of ICU stay, need for ventilator support, inotropic support, and dialysis) and mortality was documented in terms of SOFA score and QSOFA score.

Statistical analysis. [62,63,64,65,74]

Data was entered into Microsoft excel data sheet and was analysed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Chi-square test or Fischer's exact test (for 2x2 tables only) was used as test of significance for qualitative data. Continuous data was represented as mean and standard deviation. Independent t test was used as test of significance to identify the mean difference between two quantitative variables. SOFA and QSOFA score were further analysed using the receiver operating characteristic (ROC) and optimal cut-off points were chosen for the calculation of sensitivity, specificity. A test that predicts an outcome no better than chance has an area under the ROC curve of 0.5. An area under the ROC curve above 0.8 indicated fairly good prediction.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs

P value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel, SPSS version 22 (IBM SPSS Statistics, Somers NY, USA) was used to analyse data

RESULTS

RESULTS

This study was carried out in the period of November 2017 to September 2019 and 150 patients were studied.

The subjects were in the age group of 20 to 95 years.

Of 150 patients of sepsis, 87 were male and 63 were females

Table 5: Age distribution of study patients

Age group	Frequency	Percent
20-39yrs	24	16.0
40-59yrs	57	38.0
60-79yrs	50	33.3
80-99yrs	19	12.7
Total	150	100.0

Mean ± SD: 51.66±18.93

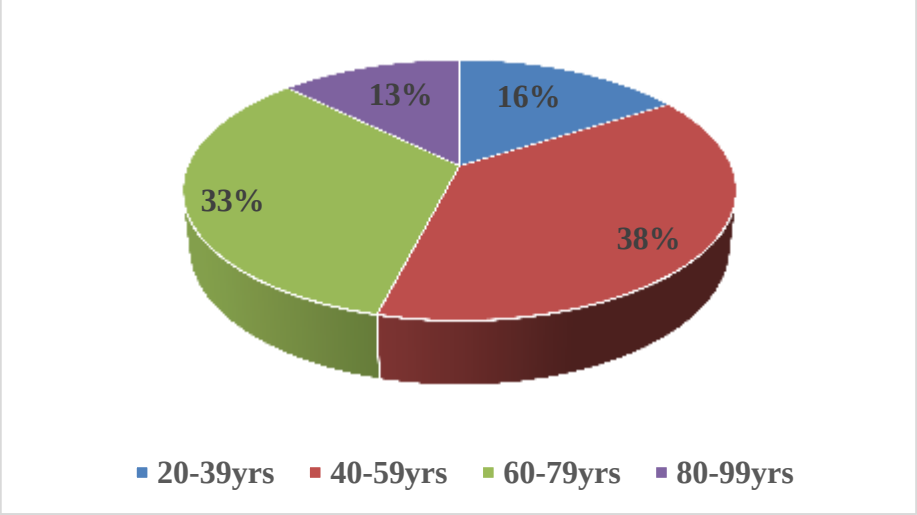


Figure 4: Age distribution of patients studied

Highest numbers of cases were in age group of 40 to 59 years i.e. 57 patients (38%) followed by 60 to 79 years in 50 cases (33.3%).

Table 6: Gender distribution of study participants

Gender	Number of patients	Percentage
Male	87	58.0
Female	63	42.0
Total	150	100

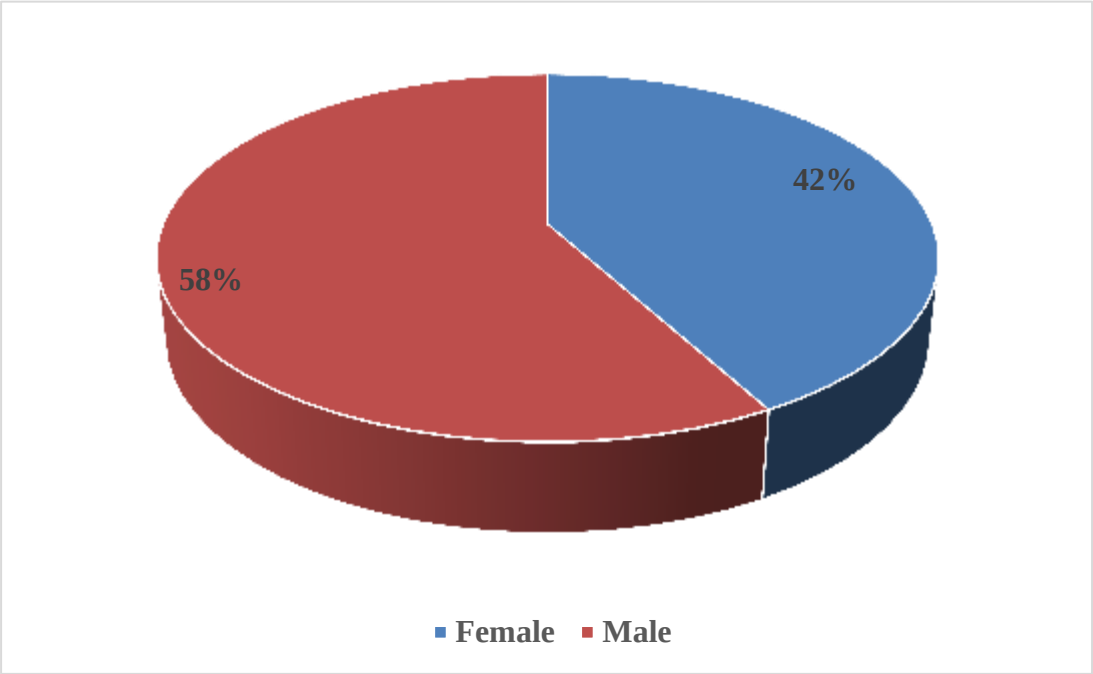


Figure 5: Gender distribution

Out of 150 patients, 87 were males and 63 were females

Table 7a: Clinical symptoms distribution of patients studied

Clinical symptom	Frequency	Percent
Fever	115	76.7
Headache	3	2.0
Cough	63	42.0
Breathlessness	52	34.7
Altered Sensorium	45	30.0
Vomiting	21	14.0
Abdominal Pain	13	8.7
Decreased Urine output	1	0.7

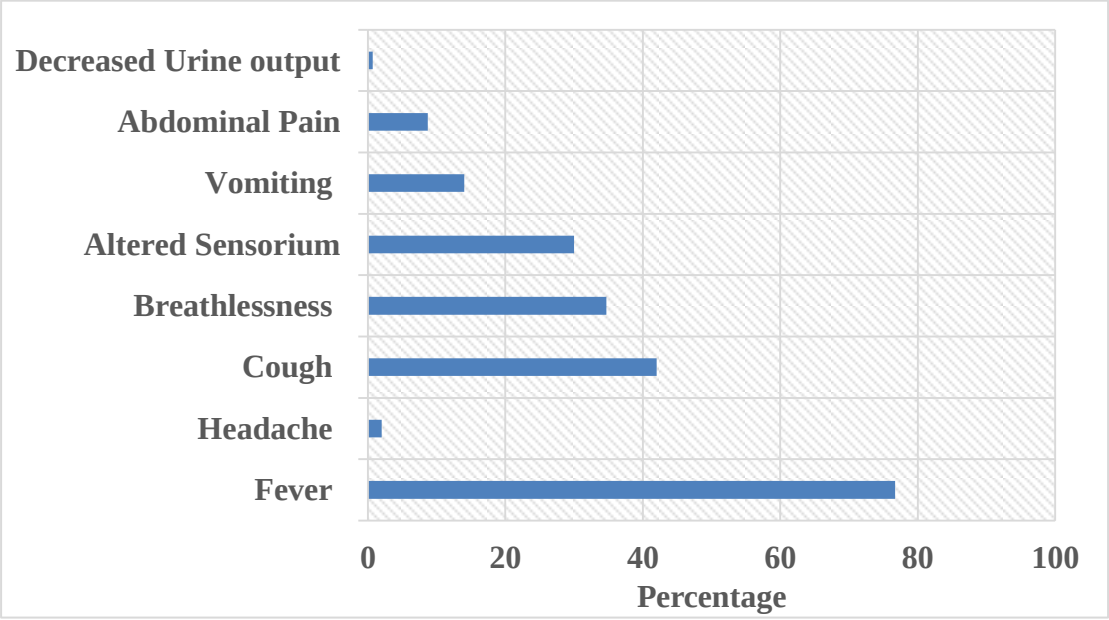


Figure 6a: Symptoms distribution

Table 7b:- Frequency of distribution of other symptoms

	Frequency	Percent
Abdominal distension	1	0.7
Anuria	1	0.7
B/L lower limb swelling	4	2.7
Burning micturition	12	8.0
Facial puffiness+	2	1.3
Haemoptysis	1	0.7
Left sided chest pain	1	0.7
Loose stools	17	11.3
U/L Lower limb swelling	11	7.3
Rashes	1	0.7
Seizures	3	2.0
Wound over right foot	1	0.7

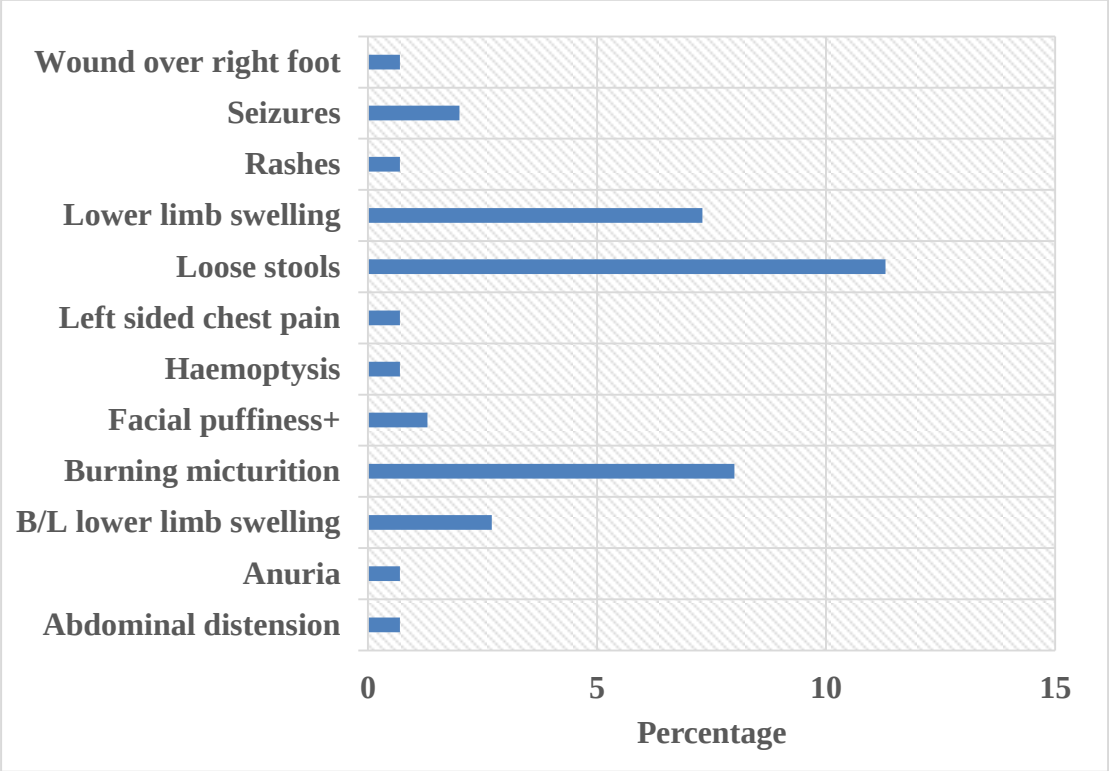


Figure 6b:- Distribution of other symptoms

The commonest symptom in the study was fever which is seen in 76.7% of patients followed by cough, breathlessness, altered sensorium, vomiting, abdominal pain and decreased urine output.

Table 8: Comorbidities distribution of patients studied

Comorbidities	Frequency	Percent
None	22	14.66
Bronchial asthma	7	4.66
COPD	9	6
TB	6	4
Hypertension	49	32.67
DM	84	56
EPILEPSY	3	2.0
IHD	14	9.3
HYPOTHYROIDISM	5	3.3

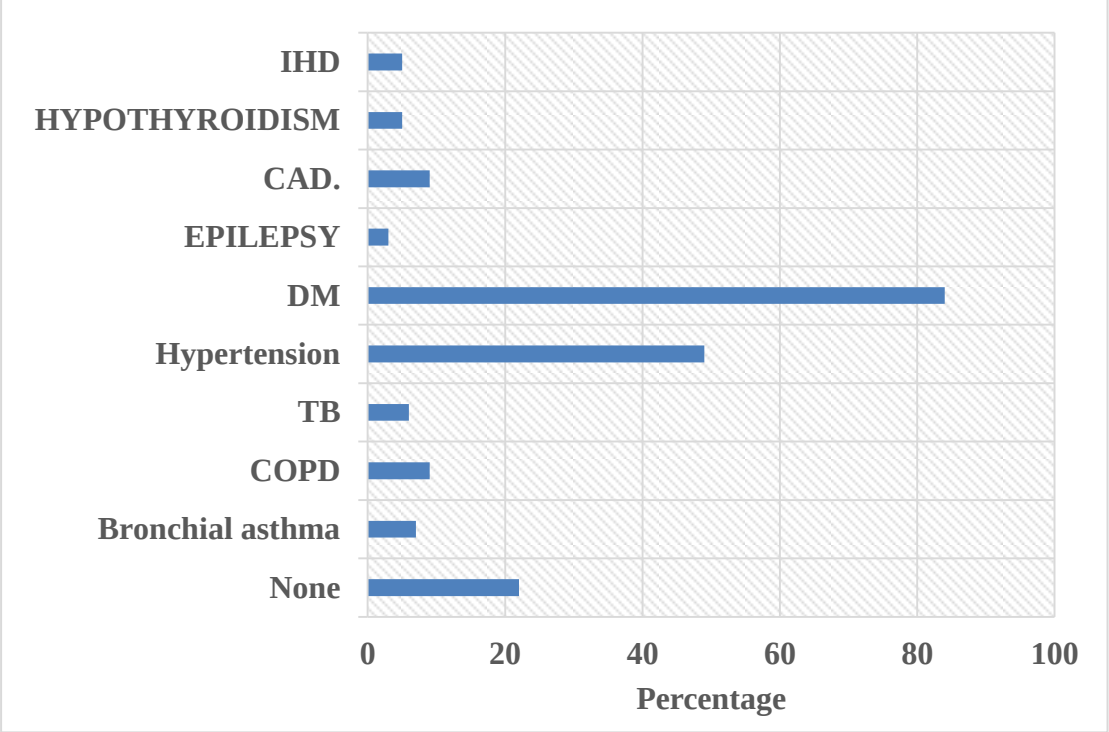


Figure 7: Comorbidities of patients studied

Most common co morbidity was diabetes seen in 56% of study patients. Hypertension was next common seen in 32.67%. 40% of study patients did not have any comorbidities.

Table 9:- Diagnosis of study subjects

DIAGNOSIS	Frequency	Percent
ACUTE GE	15	10.0
Cellulitis	13	8.7
LRTI	71	47.3
LRTI,ARDS	17	11.3
Neuro infection and meningitis	12	8
Urosepsis	22	14.7
Total	150	100.0

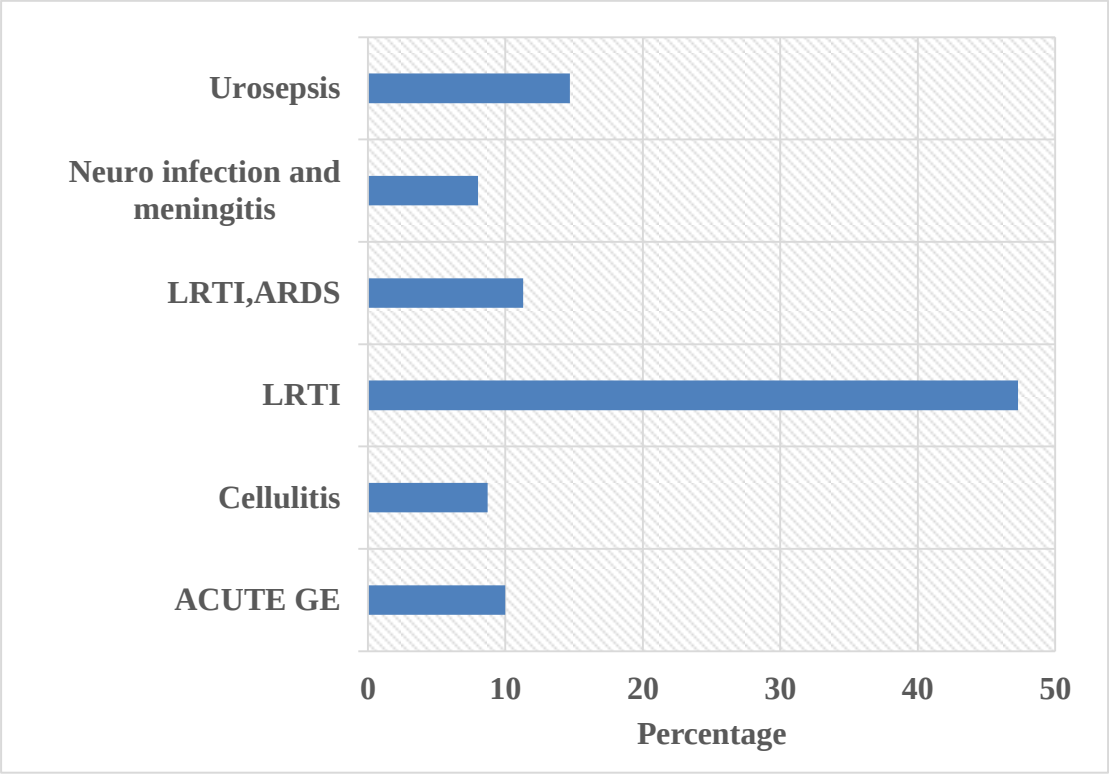


Figure 8:- Distribution of subjects of according to diagnosis

Most common diagnosis was LRTI seen in 71 (47.3%)patients, 17 patients (11.3%) with LRTI developed ARDS, next common diagnosis was urosepsis seen in 22(14.7%) patients.

Table 10:-Requirement of ventilator support

Ventilator Support	Frequency	Percent
No	64	42.7
Yes	86	57.3
Total	150	100.0

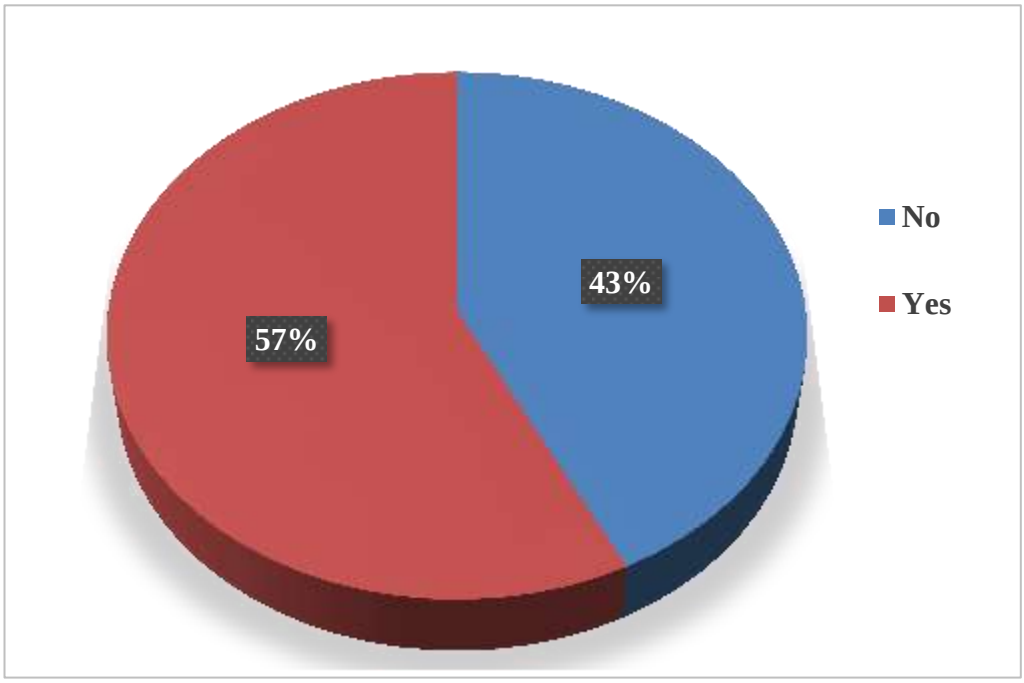


Figure 9:- Distribution of subjects according to ventilator requirement

Among 150 patients, 86 (57.3%) needed ventilator support, 64 (42.7%) did not require any ventilator support.

Table 11:-Requirement of Inotropic support

Inotropic support	Frequency	Percent
No	46	30.7
Yes	104	69.3
Total	150	100.0

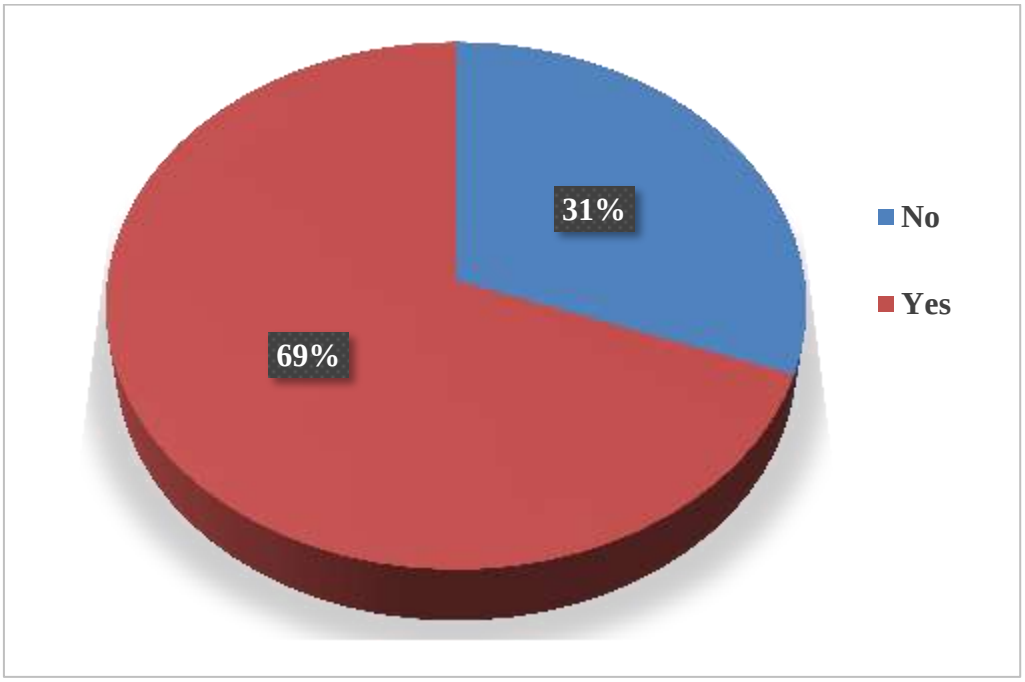


Figure 10:- Distribution of subjects of according to inotropic support

Among 150 study subjects, 104 (69.3%) patients required inotropic support, 46 (30.7%) did not require inotropic support.

Table 12:- Requirement for Haemodialysis

Haemodialysis	Frequency	Percent
No	130	86.6
Yes	20	13.4
Total	150	100.0

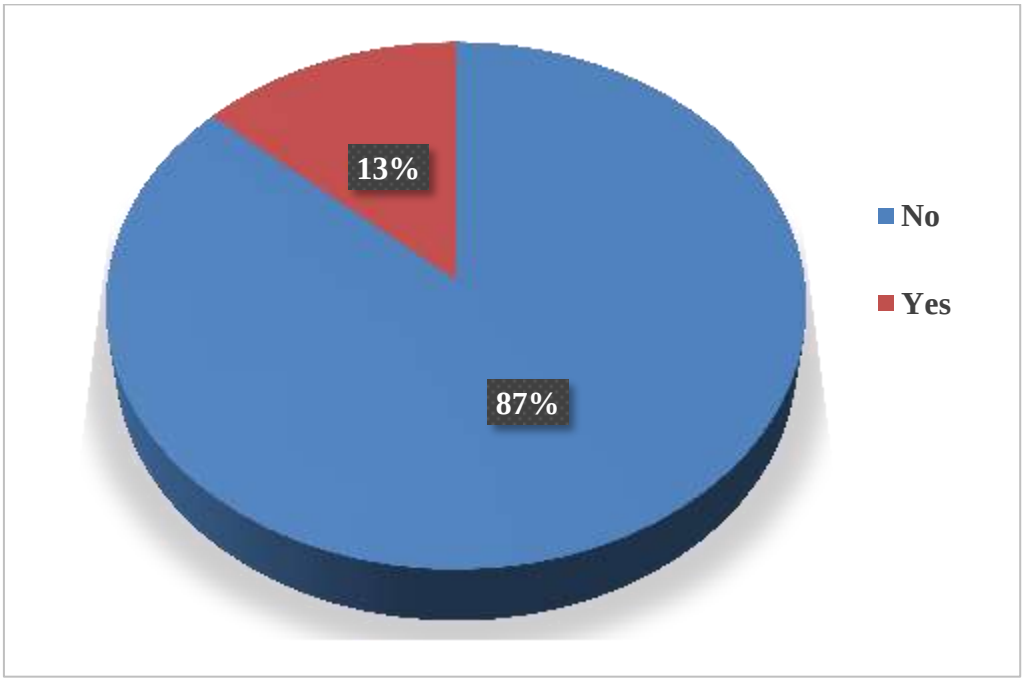


Figure 11:- Distribution of subjects of according to haemodialysis requirement

Among 150 patients, 20 patients required renal replacement therapy, 130 patients did not require renal replacement therapy.

Table 13:- Distribution of subjects of according to ICU Stay

	Frequency	Percent
<3days	66	44.0
3-6days	72	48.0
>6days	12	8.0
Total	150	100.0

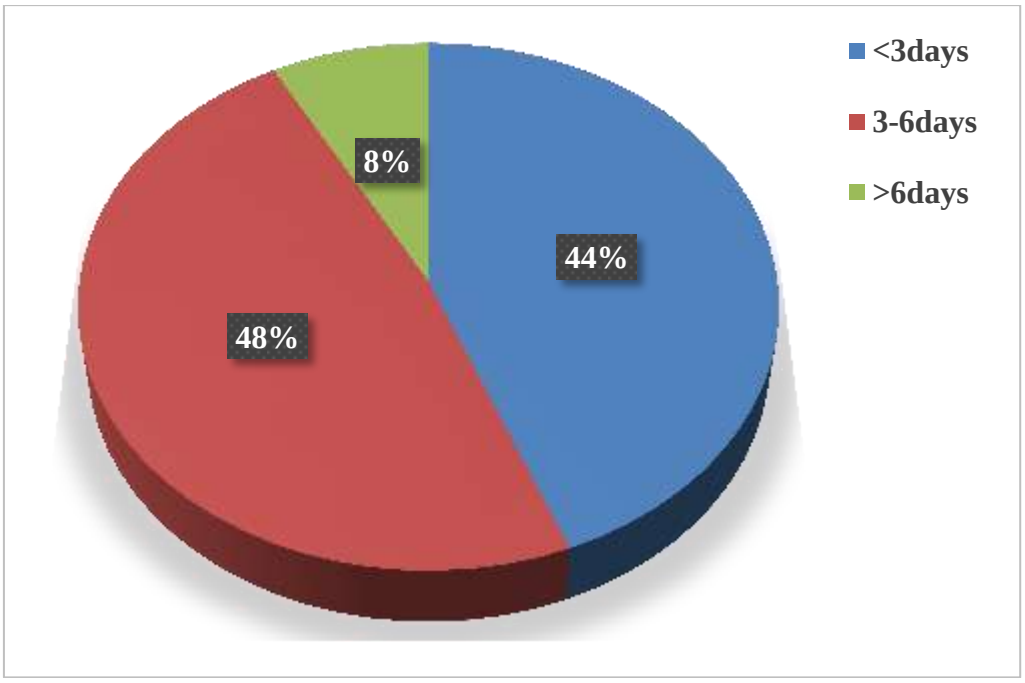


Figure 12:- Distribution of subjects of according to duration of ICU Stay

Among 150 patients, 12(8%) patients required prolonged stay in ICU of more than 6 days, 72(48%) patients stayed for 3-6 days, 66(44%) patients needed less than 3 days.

Table 14:- Initial SOFA score of study subjects

SOFA score	Frequency	Percent
<4	27	18.0
4-8	80	53.3
>8	43	28.7
Total	150	100.0

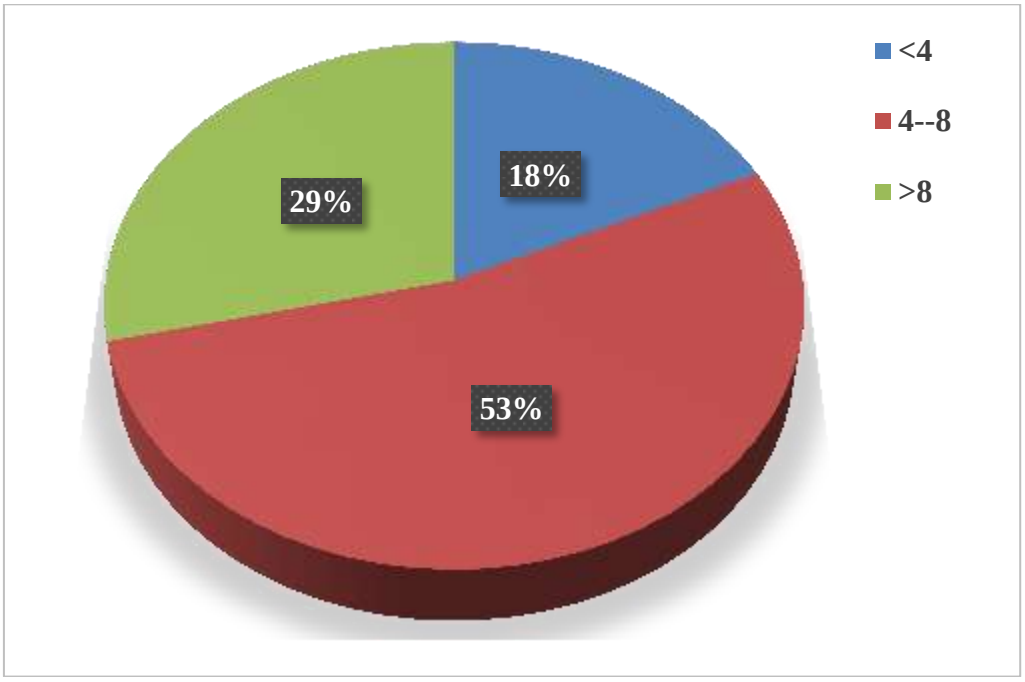


Figure 13:- Initial SOFA score of study subjects

Initial SOFA score of 28.7% of study subjects was more than 8 and 53.3% had a initial SOFA score of 4-8.

Table 15:- Initial QSOFA score of study subjects

QSOFA score	Frequency	Percent
0	5	3.3
1	27	18.0
2	56	37.3
3	62	41.3
Total	150	100.0

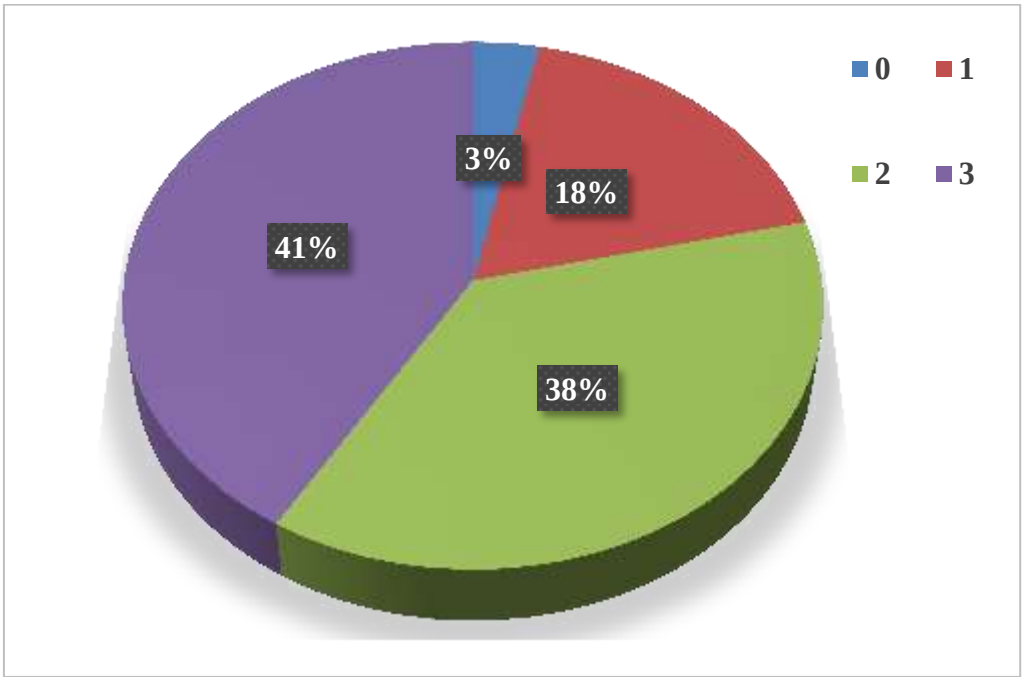


Figure 14:- Distribution of subjects according to initial QSOFA score

Initial QSOFA score of 41% patients was 3, 38% patients had QSOFA score of 2, 18% patients had QSOFA of 1 whereas 3% patients had QSOFA score of 0.

Table 16: Mortality rate

	Frequency	Percent
Survivor	92	61.3
Non -survivor	58	38.7
Total	150	100.0

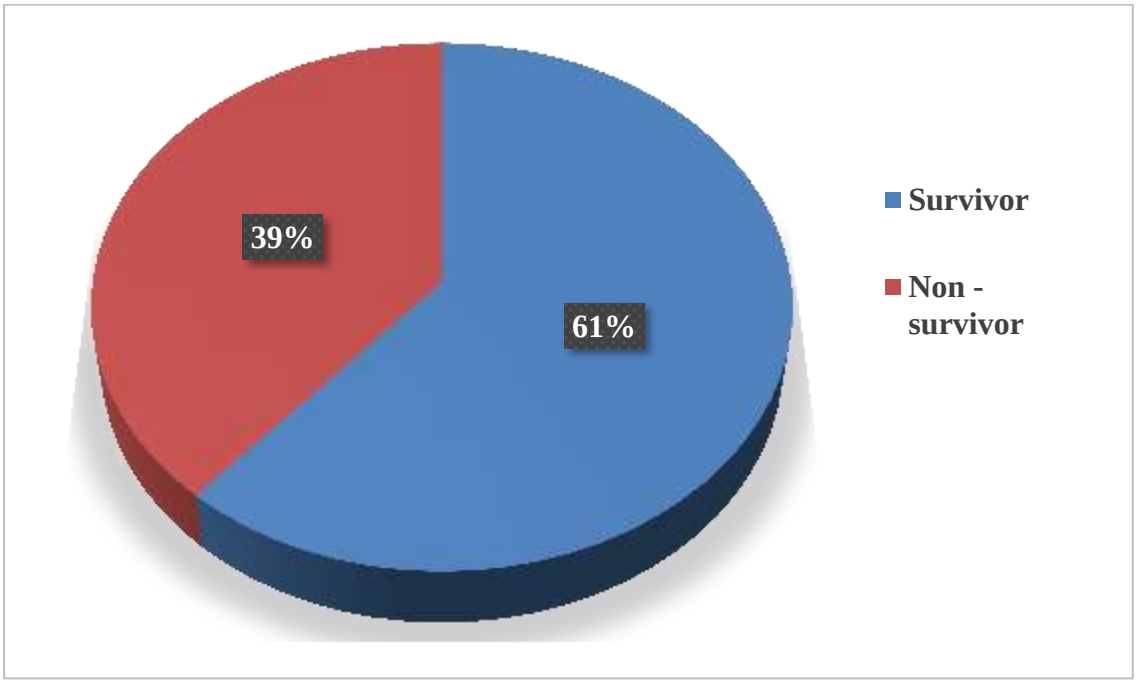


Figure 15: Mortality rate

Mortality rate in the study is 39 %. 92 of 150 patients had survived.

Table 17:- Initial SOFA score and Outcome

Initial SOFA score	Survivor	Non survivor
<4	24(26.1%)	3(5.2%)
4-8	58(63%)	22(37.9%)
>8	10(10.9%)	33(56.9%)
Total	92(100%)	58(100%)

P value <0.001, statistically significant difference found between initial SOFA score and Outcome

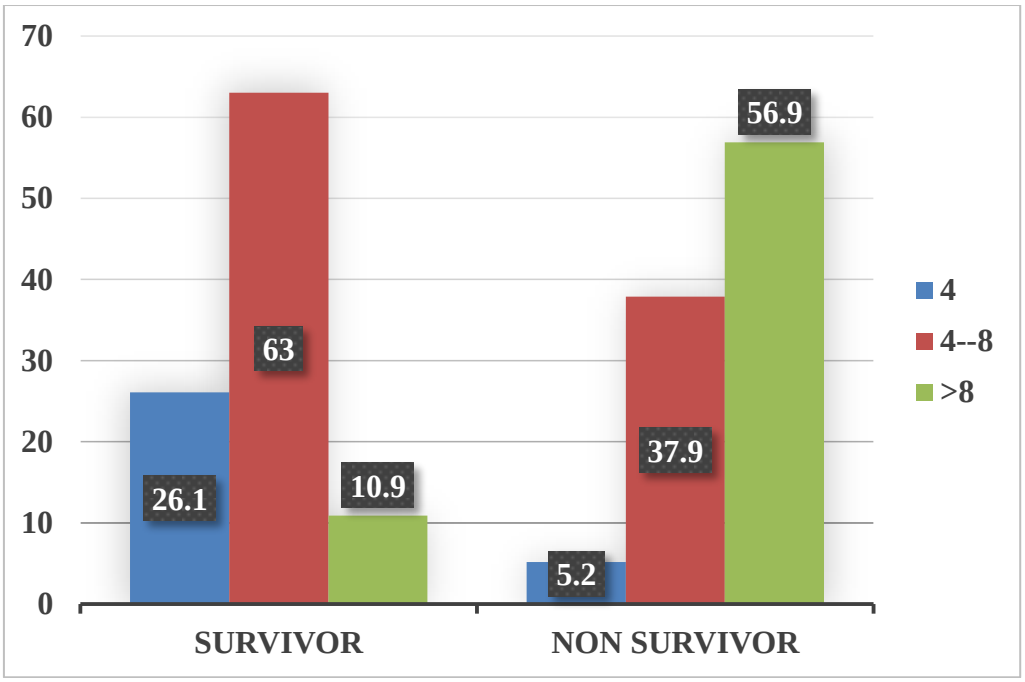


Figure 16:- Initial SOFA score and Outcome

Among Survivors: Initial SOFA score was between 4-8 in 63% patients, <4 in 26.1% patients and more than 8 in 10.9% patients

Among Non Survivors: Initial SOFA score was between 4-8 in 37.9% patients, less than 4 in 5.2% patients and above 8 in 56.9% patients.

Table 18:-Initial QSOFA score and Outcome

QSOFA score	Survivor	Non survivor
0	5(5.4%)	0
1	24(26.1%)	3(5.2%)
2	42(45.7%)	14(24.1%)
3	21(22.8%)	41(70.7%)
Total	92(100%)	58(100%)

P value <0.001, statistically significant difference was found between QSOFA score and Outcome.

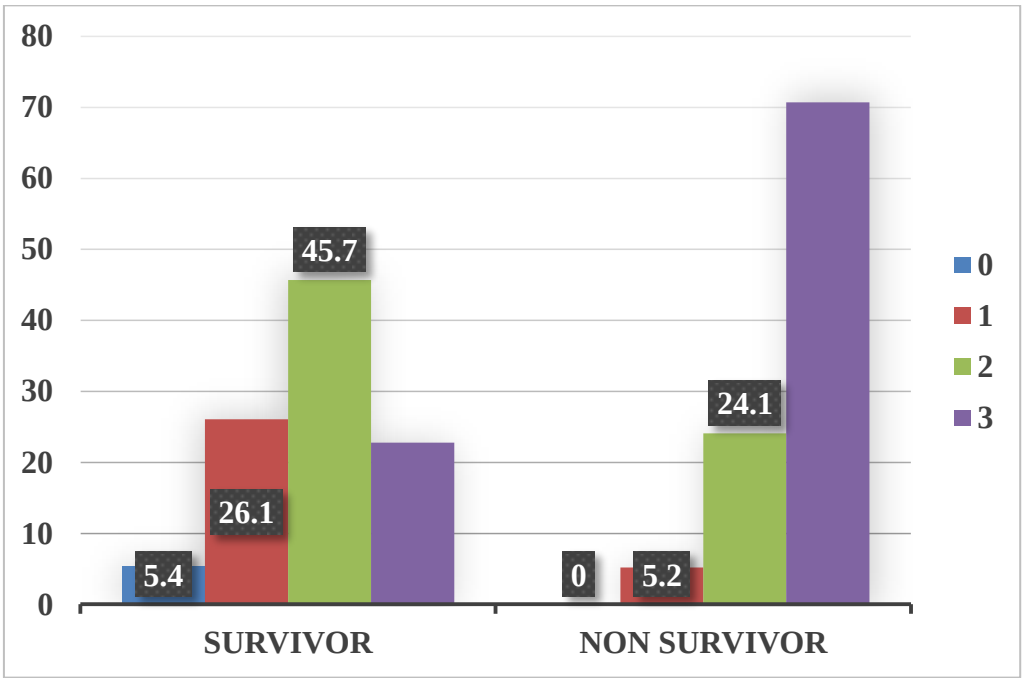


Figure 17:- Initial QSOFA score and Outcome

Among survivors initial QSOFA was 2 in 45.7% patients, 1 in 26.1% patients, 3 in 22.8% patients and 0 in 5.4% patients.

Among Non survivors initial QSOFA score was 3 in 41.7% patients, 2 in 24.1% patients, 1 in 5.25 patients and none with QSOFA score of zero.

Table 19: Comparison of SOFA score among survivors and non-survivors

SOFA score	Survivors		Non Survivors		P value
	Mean	SD	Mean	SD	
Day 1	5.39	2.79	8.64	3.63	<0.001
Day 2	5.51	2.83	9.90	3.72	<0.001
Day 3	4.82	2.80	10.41	4.52	<0.001
Day 4	4.09	2.70	9.28	4.20	<0.001
Day 5	3.22	2.38	10.33	3.58	<0.001

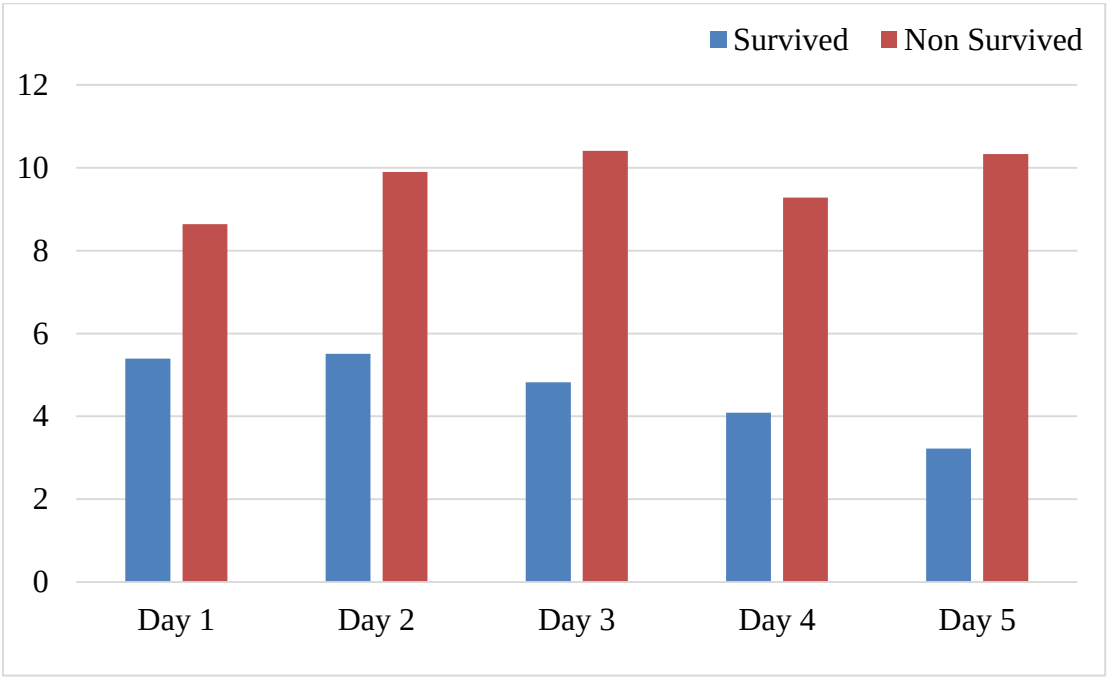


Figure 18: SOFA score among survivors and non-survivors

SOFA scores among non survivors group was higher than survivors group. Significance was highest starting from day 3 and it remained significantly higher till day 5/last day in non-survivors. The mean SOFA score on day 1 among survivors was 5.39 and among non survivors was 8.64, and on day 5 the mean SOFA score in survivors was 3.22 and in non survivors was 10.33. the P value was significant on all 5 days

Table 20: QSOFA score among survivors and non-survivors

QSOFA	Survivors		Non Survivors		P value
	Mean	SD	Mean	SD	
Day 1	1.86	.83	2.66	.58	<0.001
Day 2	1.84	.88	2.74	.54	<0.001
Day 3	1.65	.91	2.70	.61	<0.001
Day 4	1.30	.85	2.61	.85	<0.001
Day 5	0.95	.83	2.75	.45	<0.001

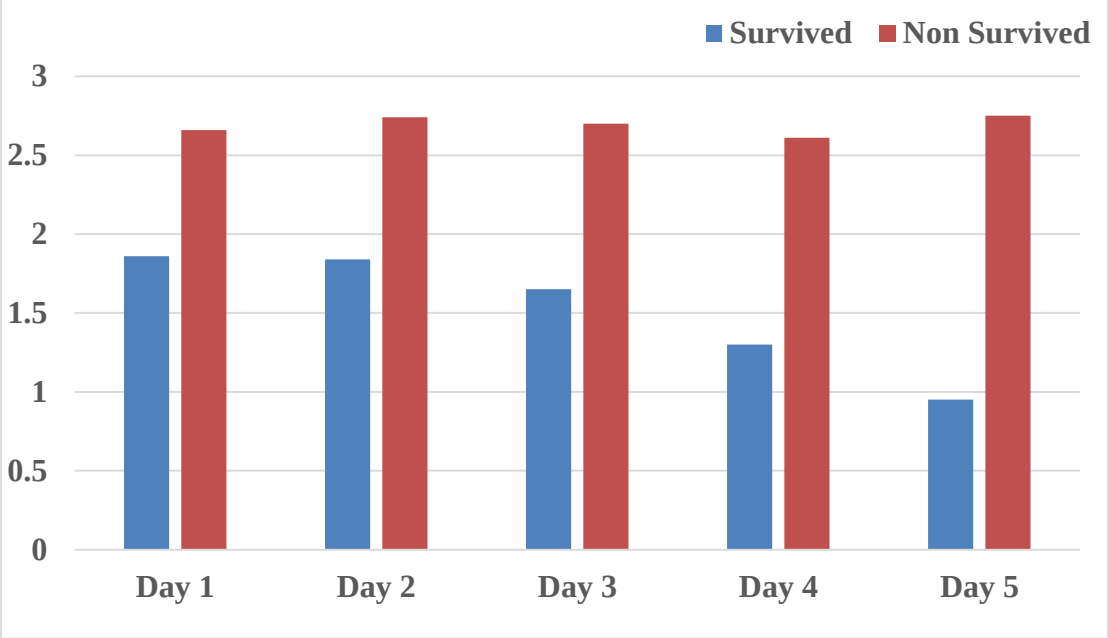


Figure 19: QSOFA score among survivors and non-survivors

QSOFA score among non survivors group was significantly higher than survivors group. Significance was highest starting from day 2 and it remained significantly higher till day 5/last day in non-survivors group. QSOFA score on day 1 in survivors was 1.86 and in non survivors it was 2.66, and on day 5 the mean QSOFA score in survivors was 0.95 and in non survivors was 2.75. the P value was significant on all 5 days

Table 21: Relation of mean initial SOFA score and morbidity.

Morbidity indicators		SOFA		P Value
		Mean	SD	
Ventilator Support	NO	1.72	.52	<0.001
	YES	2.40	.64	
Inotropic support	NO	1.93	.65	0.040
	YES	2.18	.68	
Haemodialysis	NO	2.06	.69	0.037
	YES	2.40	.50	
Length of ICU stay	<3days	2.14	.78	0.805
	3-6days	2.07	.59	
	>6days	2.17	.58	

Mean initial SOFA score in assessing requirement for ventilator support was significant with P value < 0.001. Whereas the mean initial SOFA score in assessing the requirement for inotropic support, haemodialysis and length of ICU stay was statistically not significant.

Table 22: Relation of mean initial QSOFA score and morbidity .

Morbidity indicators		QSOFA		P Value
		Mean	SD	
Ventilator Support	NO	1.81	.91	<0.001
	YES	2.43	.68	
Inotropic support	NO	1.62	.75	<0.001
	YES	2.40	.77	
Renal replacement therapy	NO	2.21	.78	0.127
	YES	1.90	1.12	
Length of ICU stay	<3days	2.26	.79	0.287
	3-6days	2.06	.87	
	>6days	2.33	.89	

The mean initial QSOFA score in assessing requirement for ventilator support and inotropic support was significant statistically with P value < 0.001. whereas initial mean QSOFA score in assessing the requirement for renal replacement therapy and length of ICU stay was statistically insignificant.

Figure 20 :- ROC curve for SOFA score on day 1

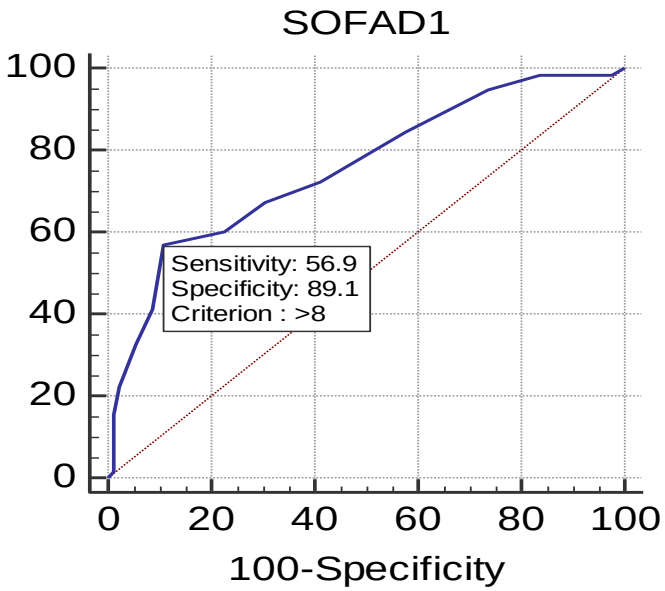


Figure 21:- ROC curve for QSOFA score on day 1

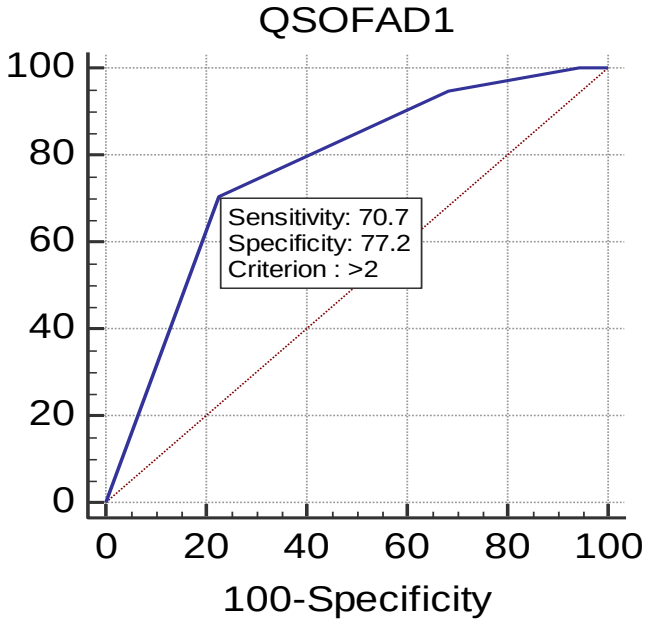


Table 23: Area under the ROC curve of QSOFA and SOFA Score

	QSOFA Score	SOFA Score
Area under the ROC curve (AUC)	0.767	0.757
Standard Error	0.0360	0.0411
95% Confidence interval	0.691 to 0.832	0.681 to 0.824
P Value	<0.0001	<0.0001

An area under the ROC curve above 0.8 indicated fairly good prediction. Area under the ROC curve for both SOFA and QSOFA score was almost similar with 0.767 and 0.757 respectively, suggesting that they are similar in assessing outcome (mortality). SOFA score on day 1 had a sensitivity of 56.9% and specificity of 89.1% in predicting mortality, and QSOFA score on day 1 had a sensitivity of 70.7% and specificity of 77.2% in predicting mortality.

DISCUSSION

DISCUSSION

As per SSC-3 150 patients with sepsis was studied.

The study included 87 males and 63 females win the age group between between 18 years to 95 years. Mean age in the study was 51.66 years. Male preponderance has seen in similar studies in India[7].

Table 24: Age comparison of patients

Age group	Rachel Oommen et al [75]	Abhinandhan et al[76]	Present study
<40 years	29.5%	36%	16%
40-70 years	63%	60%	63%
≥ 70 years	7.5%	4%	21%
Mean age	51.85±15	48.36±17	51.66±18.93

Mean age of study participants in this study is almost similar to Rachel Oommen et al. Mean age in a study by Antonino Mazzone et al in Italy was 73.3[77].

Table 25: Sex comparison of patients studied

Sex	Ferreira FL et al[68]	Abhinandhan et al[76]	Present study
Males	65%	56%	58%
Females	35%	44%	42%

Among 150 patients, 87 were male and 63 females in this study. Marginal male sex predominance was similar as seen other studies done at India and also foreign studies.

Table 26: clinical profile

Clinical feature	Abhinandhan et al[76]	Present study
Fever	100.0	76.7
Cough	26.0	42.0
Breathlessness	32.0	34.7
Altered Sensorium	4.0	30.0
Decreased Urine output	32.0	0.7
Abdominal Pain	32.0	8.7

Most common symptom in our patients was fever, followed by cough, breathlessness, altered sensorium, pain abdomen and reduced urine output . In study conducted by Abhinandhan et al, most common presentation was fever and was present in all patients in their study. Reduced urine output was observed in 29 patients for AKI .Among various organ dysfunctions in sepsis AKI is the most morbid condition since it independently increases of mortality, as well as it increases cost of care.[76,78]

Table 27: Comorbidity comparison of patients studied

Co-Morbid Illness	Dagher et al. (%)[79]	Rachel Oommen et al. (%)[75]	Present study (%)
Diabetes	34	27.5	56
Hypertension	58.8	22.5	32.67
COPD	10.3	4.5	6
IHD	25.8	8	9.3

Most common co-morbidity was diabetes, similar to Rachel Oommen et al. Dagher et al study had more number of hypertensive patients.COPD and IHD in our patients were 6% and 9.3% prevalent respectively.

Table 28: Comparison SOFA score: day 1

SOFA Score	Ferreira FL et al (%)[68]	Hewett et al (%)[80]	Present study (%)
<4	17	35.5	18.0
4-8	35	31.5	53.0
>8	48	33	28.7

Proportion of patients with SOFA score of < 4 on day 1 in our study was similar to the study done by Ferreira FL et al, whereas it was higher in study by Hewett et al. SOFA score between 4-8 was higher in this study when compared to above two studies. In this study SOFA score of >8 on the day of presentation was seen in 28.7% patients suggesting significant multi-organ dysfunction at the time of presentation.

Table 29: Comparison of mortality rate

	Dagher et al[79]	Abhinandhan et al[76]	Rachel Oommen et al[75]	Present study
Mortality	30.9%	36%	34%	38.7.0%

38.7% mortality is noted in this study.in studies done by Abhinandhan et al and Rachel Oommen et al reported a mortality of 36% and 34% respectively, similar to the present study. Mortality in sepsis ranges between 13% and 50% in numerous large clinical trials.

Table 30: Comparison of Cause of sepsis

Cause of sepsis	Antonino et al[81]	Rachel Oommen et al[75]	Present study (%)
Respiratory	26.5	48	58.6
Genitourinary	30.8	20	14.7
Gastrointestinal	-	-	10
Cellulitis	9.1	15	8.7
CNS infection	-	6	8
Others	26	11	-

Respiratory infection was the most common cause of sepsis in the study, 17 patients with pneumonia progressed to ARDS. 22 cases of UTI associated septicemia was observed. 13 patients had cellulitis. 12 had meningitis and 15 patients had gastroenteritis with sepsis.

Organ dysfunction and need for supportive care: In the current study requirement for ventilator support was seen in 86 (57.3%) patients, the mean SOFA score and mean QSOFA score of these patients were 2.4 and 2.43 respectively, both were statistically significant with p value of <0.001. 42.7% patients did not require ventilator support. Requirement for vasopressor therapy was noted in 104 (69.3%) patients among whom the mean SOFA and QSOFA score was 1.93 and 1.62 respectively, mean SOFA score in assessing need for vasopressor therapy was statistically insignificant whereas for mean QSOFA score it was significant statistically with p value of <0.001. Requirement for hemodialysis due to sepsis related AKI was seen in 20 (13.4%), in whom the mean SOFA and QSOFA score was 2.4 and 1.90 respectively, both the mean SOFA and QSOFA in assessing need for hemodialysis was statistically

insignificant with p value of 0.037 and 0.127 respectively. Majority of the patients in the study did not develop AKI.

Based on length of ICU stay patients were divided into three groups, those who required ICU care for < 3 days were 66(44%) patients in those the mean SOFA and QSOFA score was 2.4 and 2.26 respectively. Those who stayed between 3-6 days were 72 (48%) patients, in them the mean SOFA and QSOFA score was 2.07 and 2.06 respectively. 12 (8%) patients stayed for more than 6 days in them the mean SOFA and QSOFA score was 2..17 and 2.33 respectively. For the assessment of duration of ICU stay the p value for both mean SOFA and QSOFA score was statistically insignificant with p values of 0.805 and 0.283 respectively.

Predictors of mortality

In the current study, 58 patients succumbed and 92 patients survived. Among non-survivors the mean age was little high when compared to survivors (54.42 v/s 48.90) which was statistically insignificant (p=0.146).

Table 31: SOFA score as a predictor of mortality

SOFA Score	Present Study		p-value	Abhinandhan et al.[76]		p-value
	Survivors	Non-survivors		Survivors	Non-survivors	
Day 1	5.39±2.79	8.64±3.63	<0.001**	7.94±2.64	10.17±3.45	0.014*
Day 2	5.51±2.83	9.90±3.72	<0.001**	8.28±2.62	11.63±4.33	0.002**
Day 3	4.82±2.80	10.41±4.52	<0.001**	6.84±2.96	13.42±4.06	<0.001**
Day 4	4.09±2.70	9.28±4.20	<0.001**	5.94±3.41	10.78±3.77	0.001**
Day 5	3.22±2.38	10.33±3.58	<0.001**	4.55±3.27	12.25±4.8	<0.001**

In our study, evaluation of SOFA score was done from day of admission to day 5 of hospital stay. SOFA score on day 1 was more among non-survivors when compared to survivors which was significant statistically (p=<0.001).

When compared to study done by Abhinandhan et al, current study had statistically significant correlation on all 5 days, whereas in the study quoted above statistically significant correlation was seen only on day 3 and 5.

Vosylius et al in their study which included 117 patients with sepsis showed that the changes in SOFA score as an indicator of organ involvement was closely related to the outcome in ICU patients with sepsis.[82]

Table 32: Analysis of SOFA score as predictor of mortality

SOFA	Acharya et al (mortality rate)[10]	Ferreira FL et al (mortality rate)[68]	Present study (mortality rate)
SOFA at presentation >11	90%	95%	88%
Mean SOFA >7	73.91%	80% (SOFA >5.1)	87%
Highest SOFA >11	87.5%	85%	85.7

Predictive value of presentation SOFA above 11 was 88% in our study which is comparable other studies done at and Nepal and Belgium mentioned above.

Mean SOFA score of above 7 had 87% mortality predictive value. Highest SOFA score of 11 had 85.7% mortality predictive value.in study by Acharya et al and Ferreira FL et al had a mortality predictive value of 87.5% and 85% respectively with Highest SOFA score of above 11

Table 33: Analysis of QSOFA score as predictor of mortality.

QSOFA at presentation	Rudd, et al (mortality rate)[83]	Present study (mortality rate)
0	3%	0%
1	8%	5.2%
2	16%	24.1%
3	30%	70.7%

In the current study mortality with initial QSOFA score of 0 was 0%, in a study by Rudd, Kristina et al the mortality with QSOFA of 0 was 3%.in their study QSOFA score of 3 was associated with only 30% mortality whereas current study has 70.7% mortality with similar score.

Table 34: Analysis of Area under the ROC curve of QSOFA for mortality

ROC of QSOFA	Rudd et al [83]	Present study
Area under the ROC curve (AUC)	0.69	0.767
95% Confidence interval	0.67-0.71	0.691 to 0.832
P Value	<0.0001	<0.0001

Area under the ROC curve above 0.8 indicated fairly good prediction. Our study had an AUC of 0.767 compared to study by Rudd et al, who had an AUC of 0.69.

CONCLUSION

CONCLUSION

- Sepsis carries a high mortality rate. In our study, it was 38.7%
- LRTI is s most frequent cause for severe sepsis in developing countries like India
- Prompt identification of patients at risk for developing sepsis and classifying them with QSOFA score at bedside with only clinical variables helps in priority care to such patients who are at increased risk.
- QSOFA score and the SOFA score demonstrated fair to good accuracy for predicting in-hospital mortality when implicated to patients with severe sepsis.
- The initial QSOFA score of 1,2 and 3 had 5.2%, 24.1% and 70.7% mortality rate respectively. Initial SOFA score of <4, 4-8 and >8 had mortality rate of 5.2%, 37.9% and 56.9% respectively.
- The mean SOFA score had statistically significant correlation with respect to assessment of ARDS and subsequent ventilator support whereas the mean QSOFA score had a statistically significant relation in predicting need for ventilator support, vasopressor support. Both the scores had statistically insignificant correlation with respect to assessment of AKI and need for haemodialysis and in predicting the probable length of ICU care
- The QSOFA scoring system can aid the physicians in early referral to health care centre, in admitting patients to ICU, monitoring the clinical course, assessing organ dysfunction, prediction of mortality, and for transferring patients out of ICU and hence in proper utilization of ICU resources in developing countries,

SUMMARY

SUMMARY

Sepsis with multi-organ dysfunction syndrome (MODS) is a common cause of Intensive Care Unit (ICU) mortality and morbidity. Sepsis can be reversed, but as sepsis progresses to severe sepsis and septic shock the mortality rate substantially increases.

Multi-organ dysfunction syndrome is well established as the final stage of the continuum. Due to the high mortality associated with sepsis and its complications it is necessary to rapidly diagnose and treat the underlying cause.

Scoring systems for use in the intensive care unit (ICU) have been developed from the past 30 years. They are widely used in the field of critical care medicine. They allow a quantification of the severity of illness and a probability of in-hospital mortality. A well performing prognostic model in sepsis helps to make meaningful decisions regarding early goal directed therapy, anticipate organ dysfunctions and early referral from resource limited settings. The use of these prognostic models helps in providing meaningful information to physicians when discussing patient prognosis with the patient's relatives. Our study used Sequential organ failure assessment (SOFA) score and the Quick Sequential Organ Failure Assessment (QSOFA)score.

The objectives of our study were to assess morbidity and mortality of patients with multi organ dysfunction syndrome in sepsis and to compare the efficacy of a simple bed side estifiable QSOFA score with the widely accepted SOFA score in prognosticating sepsis .

The study was carried out in the period of November 2017 to September 2019 and 150 patients were included in the study. The patients with sepsis as defined by the third international consensus for sepsis :according to surviving sepsis

campaign-3 were included in the study. The detailed history, clinical examination and all the relevant laboratory investigations were done including blood culture. In our study, the conditions were defined according to standard practice and based on relevant literature. All the patients of sepsis admitted to ICU/emergency ward were prognosticated on the basis of and SOFA and QSOFA score. To assess sequential involvement of organ we calculated SOFA score and QSOFA score on every day from day of admission till 5 days/discharge/ In-hospital death. This gave us idea whether involvement of number of organ was increasing or decreasing and if the severity of particular organ was increasing.

We have also analyzed various profiles between two groups, survivor group which include the patients who are successfully discharged after recovery and non-survivor group which include the patients who died.

There were 87 males and 63 females in this cohort. The age of patients varied from 18 years to 95 years. The mean age was 51.66 years. In this study, 58 patients died and 92 patients survived.

Requirement for ventilator support was seen in 86 (57.3%) patients, Requirement for vasopressor therapy was noted in 104 (69.3%) patients, Requirement for hemodialysis due to sepsis related AKI was seen in 20 (13.4%), with respect to ICU care, patients who stayed for < 3 days were 66(44%),between 3-6 days were 72 (48%) patients and 12 (8%) patients stayed for more than 6 days.

The initial QSOFA score of 1,2 and 3 had 5.2%, 24.1% and 70.7% mortality rate respectively. Initial SOFA score of <4, 4-8 and >8 had mortality rate of 5.2%, 37.9% and 56.9% respectively.

Serial measurement of SOFA score during first week is very useful tool in predicting the outcome. The trend of SOFA score was progressively declining in survivors while non-survivors had stable higher score during the first week.

Assessment of QSOFA score at presentation is a useful tool as a sepsis prognosticator and the need for early intensive care.

LIMITATIONS OF THE STUDY

- With a sample size of 150 patients this model requires external validation.
- The time of admission to ICU for each patient is different. Lead time bias is possible.
- Nosocomial complications and socio-economic constraints are difficult to model in studies.
- History of prior antibiotic usage could not be ascertained by history.
- The short term follow up of survivors of sepsis only till hospital discharge was done, hence long term effects of sepsis on survivors could not be established by the current study.

RECOMMENDATIONS:

1. There is strong need for scoring systems for prognostication of sepsis in resource poor settings which uses clinical variables for early identification of patients who require early intensive management for prevention of development of organ dysfunction due to sepsis
2. The accuracy of QSOFA score for prognostication of sepsis patients also needs to be evaluated by further studies, to guide the clinical practice.

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ANNEXURES

ರೋಗಿಯ ತಿಳುವಳಿಕೆ ಸಮ್ಮತಿ ನಮೂನೆ

ಸಂಶೋಧಕರ ಹೆಸರು: ಡಾ. ಹಂಸ ಬಿ ಟಿ

ಸಂಸ್ಥೆಯ ಹೆಸರು: ಆರ್.ಎಲ್ ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ - ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ಗೆಜೋಡಿಸಲಾಗಿದೆ.

ಪಾಲ್ಗೊಳ್ಳುವವರ ಹೆಸರು: ಕ್ರಮ ಸಂಖ್ಯೆ :

ನಾನು ಶ್ರೀ /ಶ್ರೀಮತಿ ನನಗೆ ಆರ್. ಎಲ್. ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ನಡೆಸಲಾಗುತ್ತಿರುವ ಅಧ್ಯಯನ ''ನಿರೀಕ್ಷಿತ ಸೋಂಕಿನ ರೋಗಿಗಳಲ್ಲಿ ರೋಗದ ನಿಖರವಾದ ಮುನ್ನರಿವಿಗೆ QSOFA ಮತ್ತು SOFA ಅಂಕಗಳ ಹೋಲಿಕೆ '' ದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಲ್ಪಡಲಾಗುವುದು ಎಂದು ನನಗೆ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನನ್ನನ್ನು ಆಹ್ವಾನಿಸಲಾಗಿದೆ. ಈ ದಾಖಲೆಯಲ್ಲಿರುವ ಮಾಹಿತಿಯು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಬೇಕೇ ಅಥವಾ ಬೇಡವೇ ಎಂಬುದನ್ನು ನಿರ್ಧರಿಸಲು ನನಗೆ ನೆರವಾಗುವುದು. ಪ್ರಧಾನಸಂಶೋಧಕನೊಂದಿಗೆ ನಾನು ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನನ್ನ ಅನುಮಾನಗಳನ್ನು ಸ್ಪಷ್ಟಪಡಿಸಿಕೂಂಡಿದ್ದೆನೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಂತೆ ನನಗೆ ಸೂಚಿಸಲಾಗಿದೆ ಏಕೆಂದರೆ ನಾನು ಅರ್ಹತಾ ಮಾನದಂಡಗಳನ್ನು ಪೂರೈಸುತ್ತೇನೆ.

ನನ್ನ ರಕ್ತದ ಮಾದರಿಯನ್ನು ಗೊತ್ತುಪಡಿಸಿದ ಪರೀಕ್ಷೆಗಳಿಗೆ ನಿರ್ವಹಿಸಲು ನಾನು ಡಾ.ಹಂಸ ಬಿ ಟಿ ಅವರನ್ನು ವಿನಂತಿಸುತ್ತೇನೆ ಮತ್ತು ಅಧಿಕಾರವನ್ನು ನೀಡುತ್ತೇನೆ.ಕೆಳಗಿನ ನನ್ನ ಸಹಿಯು ಅರ್ಹ ಆರೋಗ್ಯ ವೃತ್ತಿಪರರಿಂದ ಪರೀಕ್ಷೆಯ ಅನುಕೂಲಗಳು,ಅಪಾಯಗಳು ಮತ್ತು ಮಿತಿಗಳನ್ನು ನನ್ನ ತೃಪ್ತಿಗೆ ವಿವರಿಸಲಾಗಿದೆ ಎಂದು ನನ್ನ ಅಂಗೀಕಾರವನ್ನು ರೂಪಿಸುತ್ತದೆ.

ಭಾಗವಹಿಸುವಿಕೆ ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿರುತ್ತದೆ ಮತ್ತು ಮಾದರಿ ಸಂಗ್ರಹಣೆಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಪಾವತಿಯಿಲ್ಲ. ಎಲ್ಲಾ ಪರೀಕ್ಷಾ ಫಲಿತಾಂಶಗಳನ್ನು

ವೈದ್ಯಕೀಯ ಗೌಪ್ಯತೆಯೊಂದಿಗೆ ಪರಿಗಣಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಕಾನೂನಿನ ಅಗತ್ಯವಿದ್ದರೆ ಹೊರತುಪಡಿಸಿ ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ.

ನನ್ನ ಗೌಪ್ಯತೆ ನಿರ್ವಹಿಸಲ್ಪಡುವವರೆಗೆ ವೈದ್ಯಕೀಯ ಪರೀಕ್ಷೆ, ಪರೀಕ್ಷೆಯ ಮೌಲ್ಯಮಾಪನ ಅಥವಾ ಶಿಕ್ಷಣಕ್ಕಾಗಿ ನನ್ನ ಮಾದರಿಯನ್ನು ಬಳಸಲು ನನ್ನ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ.

ನಾನು ಈ ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಲು ಮುಕ್ತವಾಗಿರುತ್ತೇನೆ ಮತ್ತು ಇದು ನನ್ನ ಮುಂದಿನ ಕಾಳಜಿಯನ್ನು ಬದಲಿಸುವುದಿಲ್ಲ ಎಂದು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ರೋಗಿಯ ಮಾಹಿತಿ ಪತ್ರವನ್ನು ನಾನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಪ್ರತಿಯನ್ನು ಸ್ವೀಕರಿಸಿದ್ದೇನೆ.ಈ ದಾಖಲೆಯಲ್ಲಿ ಒದಗಿಸಿದ ಮಾಹಿತಿಯನ್ನು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ ಮತ್ತು ಪರೀಕ್ಷೆ, ಪ್ರಕ್ರಿಯೆ, ಸಂಬಂಧಿಸಿದ ಅಪಾಯ ಮತ್ತು ಪರ್ಯಾಯಗಳ ಬಗ್ಗೆ ನಾನು ಹೊಂದಿರುವ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶ ಕಲ್ಪಿಸಲಾಗಿದೆ .

ಹೆಸರು ಮತ್ತು ಸಹಿ / ಹೆಬ್ಬೆರಳುಗುರುತು ದಿನಾಂಕ:

ಪೋಷಕರ / ಪಾಲಕರ ಹೆಸರು /ಹೆಬ್ಬೆರಳು ಗುರುತು ದಿನಾಂಕ:

ಒಪ್ಪಿಗೆ ತೆಗೆದುಕೊಳ್ಳುವ ವ್ಯಕ್ತಿಯ ಸಹಿ ದಿನಾಂಕ

PATIENT INFORMATION SHEET

ಅಧ್ಯಯನ :"ನಿರೀಕ್ಷಿತ ಸೋಂಕಿನ ರೋಗಿಗಳಲ್ಲಿ ರೋಗದ ನಿಖರವಾದ ಮುನ್ನರಿವಿಗೆ QSOFA ಮತ್ತು SOFA ಅಂಕಗಳ ಹೋಲಿಕೆ "

ಸಂಸ್ಥೆಯ ಹೆಸರು: ಆರ್.ಎಲ್ ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ - ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ಗೆಜೋಡಿಸಲಾಗಿದೆ.

ಭಾಗವಹಿಸಲುಸಮ್ಮತಿ

ನಾನು, ರುಜುಮಾಡಿರುವ,

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಮತ್ತು ಈ ಒಪ್ಪಿಗೆರೂಪದಲ್ಲಿ ಅಂಶಗಳಂತೆ ನನ್ನ ವೈಯಕ್ತಿಕ ಮಾಹಿತಿಯ ಸಂಗ್ರಹಣೆ ಮತ್ತು ಡಿಸ್ಕ್ಲೋಸ ರ್ಅಧಿಕೃತಗೊಳಿಸಲು ಒಪ್ಪುತ್ತೀರಿ.

ಅಧ್ಯಯನ ಮತ್ತು ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಸಂಗ್ರಹಿಸಿದ ಮತ್ತು ಬಹಿರಂಗ ನಡೆಯಲಿದೆ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯ ಪ್ರಕೃತಿಯಲ್ಲಿ ನನ್ನ ಒಳಗೊಳ್ಳುವಿಕೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯಗಳನ್ನು ಮತ್ತು ಲಾಭಗಳನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ನಾನು ವಿವಿಧ ಈ ಅಧ್ಯಯನದ ಅಂಶಗಳು ಮತ್ತು ನನ್ನ ಪ್ರಶ್ನೆ ಗಳಿಗೆ ನನ್ನ ತೃಪ್ತಿಕರ ಉತ್ತರಗಳನ್ನು ಮಾಡಲಾಗಿದೆ ಸಂಬಂಧಿಸಿದ ಪ್ರಶ್ನೆಗಳನ್ನುಕೇಳಲು ಅವಕಾಶ ಹೊಂದಿದ್ದರು.

ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಈ ಅಧ್ಯಯನದಿಂದ ಹಿಂತೆಗೆದುಕೊಳ್ಳುವಂತೆ ಮತ್ತು ಈ ನನ್ನ ಮುಂದಿನ ಆರೈಕೆ ಬದಲಾಗುವುದಿಲ್ಲ ಉಚಿತ ಉಳಿಯಲು ಎಂದು ಅರ್ಥ.

ವಿಷಯದಹೆಸರುಮತ್ತುಸಹಿ / ಹೆಬ್ಬೆಟ್ಟಿನಗುರುತುದಿನಾಂಕ:

ಪೋಷಕ / ಪೋಷಕರು ಹೆಸರು ಮತ್ತು ಸಹಿದಿನಾಂಕ:

ಒಪ್ಪಿಗೆಪಡೆದ ವ್ಯಕ್ತಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ ದಿನಾಂಕ:

INFORMED CONSENT FORM

Name of the investigator: DR. HAMSA B T

Name of the organisation: R L JALAPPA HOSPITAL AND RESEARCH CENTRE ATTACHED TO SRI DEVARAJ URS MEDICAL COLLEGE

Name of the participant: SI no:

I Mr./Mrs. have been explained in my own understandable language, that I will be included in a study which is "PROGNOSTIC ACCURACY OF QSOFA SCORE COMPARED TO SOFA SCORE AMONG PATIENTS WITH SEPSIS" being conducted in RL JALAPPA HOSPITAL.

I have been invited to take part in this research study. The information in this document is meant to help me decide whether or not to take part. I have clarified my doubts regarding this study with the principal investigator.

I have been asked to participate in this study because I satisfy the eligibility criteria .

I request and authorise Dr. Hamsa B T to perform the designated tests for my blood sample. My signature below constitutes my acknowledgement that the benefits, risks and limitations of this testing have been explained to my satisfaction by a qualified health professional.

Participation is totally voluntary and there would be no payment for sample collection. All test results are treated with medical confidentiality and will not be disclosed to any outsider except if it is required by the law.

I give my consent to allow my sample to be used for medical research, test validation or education as long as my privacy is maintained.

I understand that I remain free to withdraw from this study at any time and this will not change my future care.

I have read and received a copy of patient information sheet. I understand the information provided in this document and I have had the opportunity to ask questions I might have about the testing, the procedure, the associated risk and alternatives.

Subject name and signature/ thumb impression

Date:

Parent’s/ guardian’s name/ thumb impression

Date:

Signature of the person taking consent

Date:

PATIENT INFORMATION SHEET

Study title: "PROGNOSTIC ACCURACY OF QSOFA SCORE COMPARED TO SOFA SCORE AMONG PATIENTS WITH SEPSIS"

Study site: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar

Details : patients aged above 18 years with sepsis admitted to medicine department will be included in the study

Patients with sepsis will be assessed for prognosis based on sofa and qsofa scores, using routine investigations, the two scores at the end of the study will be compared.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in this study we will do routine investigations daily and assess the prognosis. This information collected will be used for dissertation and publication only.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For any further clarification you can contact the study investigator:

Dr. HAMSA B T
Mobile no: 9686276756
E-mail id: hamsareddy12@gmail.com

IEC CERTIFICATE

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH

SDUAHER

SRI DEVARAJ URS MEDICAL COLLEGE

Tamaka, Kolar

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No. SDUMC/KLR/IEC/ 05 /2017-18 Date: 29-11-2017

CERTIFICATE

This is to certify that the ethics committee of Sri Devaraj Urs Medical College, Kolar in its meeting conducted on **29-11-2017** has unanimously approved the synopsis for the dissertation entitled **"Prognostic accuracy of qsofa score compared to sofa score among patients with sepsis"** to be submitted to Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka, by **Dr.Hamsa B T,** Postgraduate student in the department of **General Medicine** at Sri Devaraj Urs Medical College, Kolar.

Member Secretary
Institutional Ethics Committee
SDUMC, Tamaka Kolar

Member Secretary
Institutional Ethics Committee
Sri Devaraj Urs Medical College
Tamaka, Kolar

Chairman
Institutional Ethics Committee
SDUMC, Tamaka Kolar

CHAIRMAN
Institutional Ethics Committee
Sri Devaraj Urs Medical College
Tamaka, Kolar

PROFORMA FOR DATA COLLECTION

Name of the patient:
Age: Sex:
Date of admission: IP Number:
Address:
History of symptoms at presentation with duration:

ON EXAMINATION:

	Day 1	Day 2	Day 3	Day 4	Day 5
Blood pressure					
Pulse					
Respiratory rate					
Spo2					
GCS					

RESPIRATORY SYSTEM:

CARDIOVASCULAR SYSTEM:

ABDOMEN EXAMINATION:

CNS:

DIAGNOSIS:

INVESTIGATIONS:

DATE	DAY1	DAY2	DAY3	DAY4	DAY5
HAEMOGLOBIN (GM %)					
TOTAL LEUKOCYTE COUNT					
PLATELET COUNT					
BLOOD UREA					
S.CREATININE					
BILIRUBIN					

CHEXT XRAY:
ECG:
CRP:
USG ABDOMEN AND PELVIS:
SEROLOGY:

CULTURES:

OTHER INVESTIGATIONS:

SUPPORTIVE CARE:
RENAL REPLACEMENT THERAPY:
VENTILATORY SUPPORT:
IONOTROPIC SUPPORT:

OUTCOME: DISCHARGE: ☐ DEATH: ☐

DAILY PROGRESS OF THE PATIENT:

SOFA SCORE DAY	DAY 1	DAY 2	DAY 3	DAY 4	DAY 5
RESPIRATORY					
COAGULATION					
HEPATIC					
RENAL					
CARDIOVASCULAR					
CNS					
TOTAL					

QSOFA SCORE DAY	DAY 1	DAY 2	DAY 3	DAY 4	DAY 5
RESPIRATION					
ALTERED MENTATION					
SYSTOLIC BLOOD PRESSURE					
TOTAL					

SIGNATURE OF GUIDE

	UHID Number	Age(years)	Sex	Fever	Headache	Cough	Breathlessness	Altered Sensorium	Vomiting	Jaundice	Decreased Urine o	Abdominal Pain	Others	Co-Morbidities	Temperature(F)	Pulse(bpm)	B.P(mmhg)					RR (CPM)					Pallor,Icterus,Cya	RS	CVS	CNS	Hepatomegaly	Splenomegaly	HB%	ECG	ChestX-Ray
																	D1	D2	D3	D4	D5	D1	D2	D3	D4	D5									
1	530539	90	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM. HTN	99	100	100/70	110/70	100/80	106/80	104/74	30	26	26	24	20	PALLOR	B/L BRONCHIAL BS	S1 S2 NORMAL	CONSCIOUS ORIENTED	NO	NO	12	SYNUS RHITHM	B/L LL NHO
2	532564	67	F	YES	NO	NO	NO	YES	NO	NO	NO	NO		T2DM. HTN	100	120	80/50	80/50	9060	100/7	100/74	34	30	26	28	24	PALLOR	B/L N VESCULAR BREATH SOUNDS	S1 S2 NORMAL	ALTERED SENSORIUM.NECK STIFFNES PRESENT	NO	NO	9	SINUS TACHYCARDIA	NORMAL
3	532706	75	M	YES	NO	YES	NO	YES	NO	NO	NO	NO		T2DM,HTM	101	120	90/60	90/69	100/70	98/64	80/60	34	30	28	24	22	CYANOSIS	B/L BASAL CREPITATIONS	S1 S2 NORMAL	ALTERED SENSORIUM. NO MENINGEAL SIGNS	NO	NO	11.4	SINUS TACHYCARDIA. P PULMONALE	B/L LOWER ZONE NHO+
4	540240	43	M	YES	NO	YES	NO	YES	NO	NO	NO	NO		T2DM,HTM	99	130	70/60	90/60	80/50	100/60	10064	34	30	26	28	20		B//L CREPITATIONS +IN ISA	S1 S2 NORMAL	ALTERED SENSORIUM.NO MENINGEAL SIGNS	NO	NO	14.5	NORMAL. RAD	B/L LOWER ZONE NHO+
5	540306	78	F	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM,HTM	100	120	100/70	100/84	110/70	100/80	110/70	30	26	24	22	20	PALLOR	B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS , ORIENTED,NO FOCAL NEUROLOGICAL DEFICITS	NO	NO	9.3	SINUS RHYTHM. LVH	B/L LL NHO+
6	497190	28	M	YES	NO	YES	NO	NO	NO	NO	NO			T2DM,HTM	101	134	90/60	94/60	80/60	90/64	100/60	36	30	26	24	22		B/L CREPITATIONS + IN LUNG FIELDS	S1 S2 NORMAL	CONSCIOUS ,ORIENTED,NO FOCAL DEFICITS			10.5	SINUS TACHYCARDIA	B/L MID AND LL NHO +
7	541622	57	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		B,ASTHMA.HTN	102	100	100/70	100/60	104/70	106/80	106/84	30	20	22	24	20	CYANOSIS	B/L ISA IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS,RESTLESS & UNSTABLE NO FOCAL NEUROGICAL DEFICITS	NO		12.9	SINUS TACHYCARDIA +	B/L LL NHO+
8	543029	50	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM,HTM	100	120	100/60	104/60	110/60	108/64	100/68	35	36	30	28	24		B/L CREPITATIONS IN LUNG FIELDS	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	9.2	SINUS RHYTHM	B/L LL NHO +
9	438519	80	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM	99	110	100/70	110/70	108/60	110/70	100/60	30	34	30	28	20		B/L INFLA AXILLARY AREA CREPITTION +	S1 S2 NORMAL	CONSCIOUS, ORIENTED			10.7	SINUS RHYTHM	B/L LL NHO +
10	543086	50	F	NO	NO	NO	YES	YES	NO	NO	NO	NO		T2DM. HTN	98	130	90/60	80/40				30	34					ISA ,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS,RESTLESS & DISORIENTED NO FOCAL DEFICITS	NO	NO	6.4	SINUS TACHYCARDIA +	LL NHO +
11	543576	85	F	YES	NO	NO	NO	YES	NO	NO	NO	NO			100	120	110/70	100/72	110/70	110/60	100/70	30	26	20	18	18		B/L NVBS +	S1 S NORMAL	SEMI CONSCIOUS,NOT ORIENTED O TIME ,PLACE,PERSON	NO		11.5	SINUS RHYTHM	NORMAL
12	542129	40	M	YES	NO	NO	NO	YES	NO	NO	NO	NO		HTN	101	120	100/60	80/50	90/50	70/40	70/40	30	28	26	24	24		B/L NVBS +	S1 S2 NORMAL	ALTERED SENSORIUM+, NO FOCAL DEFICITS, NECK STIFFNESS+, KERNIG'S SIGN+	NO	NO	11.9	SINUS RHYTHM	NORMAL
13	546323	35	M	YES	NO	NO	NO	YES	NO	NO	NO	NO		B,ASTHMA	101	100	90/60	100/60	94/60	100/60	140/70	26	28	22	18	18		B/L RONCHI +	S1 S2 NORMAL	ALTERED SENSORIUM+, NO FOCAL DEFICITS, KERNIG'S SIGN+	NO	NO	14.6	NORMAL	NORMAL
14	543604	81	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM. HTN	99	120	90/60	80/60	100/60	110/60	100/70	30	26	20	20	20		B/L ISA CREPTS +	S1 S2 NORMAL	CONSCIOUS , ORIENTED,NO FOCAL NEUROLOGICAL DEFICITS	NO	NO	15.2	SINUS RHYTHM	NORMAL
15	546581	68	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		COPD. HTN	101	120	90/60	100/60	110/60	100/70	100/80	30	28	26	28	26		B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	16.1	SINUS TACHYCARDIA	B/L LL NHO+
16	547931	80	F	YES	NO	YES	NO	NO	NO	NO	NO	NO		CAD. T2DM.HTN	101	100	90/60	90/60	100/60	110/60	110/70	28	30	26	20	18		B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS, RESTLESS	NO	NO	11.9	NORMAL	LEFT LL NHO +
17	549820	67	F	YES	NO	NO	NO	NO	NO	NO	NO	NO	LEFT L L SWELLING	CAD. T2DM.HTN	99	120	90/60	80/50	100/60	100/70	110/60	28	30	26	24	20		B/L NVBS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.5	RBBB	NORMAL
18	549901	72	M	NO	NO	YES	NO	YES	NO	NO	NO	NO		T2DM.HTN	98	90	90/60	90/50	100/60	100/70	110/60	30	26	26	20	18		RIGHT ISA, IAA CREPTS+	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	13.8	NORMAL	RIGHT LL NHO+
19	550422	68	M	YES	NO	NO	NO	YES	NO	NO	NO	NO		B.ASTHMA	101	100	90/50	80/50	90/60	100/70	110/74	30	28	26	20	18		B/L DIFFUSE CREPTS+	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	16.8	NORMAL	NORMAL
20	550779	50	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		HTN	101	100	90/60	80/50	90/60	100/60	110/70	30	26	26	20	18		LEFT ISA, IAA CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	16.8	LVH	LEFT LL NHO+
21	552620	55	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		HTN	102	120	70/40	80/50	80/50			36	30	28				B/L DIFFUSE CREPTS+	S1 S2 NORMAL	ALTERED SENSORIUM+ , NO FOCAL DEFICITS	NO	NO	7.2	NORMAL	B/L LL NHO+
22	546654	77	M	NO	NO	YES	NO	NO	NO	NO	NO	NO		HYPOTHYROIDISM	98	120	90/50	90/60	90/60	100/60	110/60	36	34	30	34	36		B/L DIFFUSE CREPTS+	S1 S2 NORMAL	ALTERED SENSORIUM+ , NO FOCAL DEFICITS	NO	NO	8.4	SINUS TACHYCARDIA	LEFT LL NHO+
23	552639	65	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM.HTN	99	130	80/50					34						B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS, RESTLESS	NO	NO	7	NORMAL	B/L LL NHO+
24	553048	51	M	NO	NO	YES	NO	YES	NO	NO	NO	NO			101	120	80/50	90/60				34	36					B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	8	POOR R WAVE PROGRESSION	B/L LL NHO+
25	553330	70	F	YES	NO	NO	NO	NO	NO	NO	NO	YES	BURNING MICTURITION	T2DM.HTN	102	100	90/60	90/60	90/64	100/60	100/60	12	14	16	16	14		B/L NVBS +	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	11.2	NORMAL	NORMAL
26	540383	63	M	YES	NO	NO	YES	NO	NO	NO	NO	NO		HTN	101	100	90/60	84/60	96/60	100/60		30	30	28	28			B/L RHONCHI+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	10.9	RBBB	EMPHYSEMATOUS
27	535523	45	F	YES	NO	YES	NO	NO	NO	NO	NO	NO		HYPOTHYROIDISM	101	100	80/50	80/50	100/60	96/60	100/60	28	26	28	28	26		B/L BRONCHIAL BS	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	12.3	NORMAL	B/L CAVITIES+
28	556296	35	F	NO	NO	NO	YES	YES	NO	NO	NO	NO		B,ASTHMA	98	120	60/40					34						B/L DIFFUSE CREPTS+	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	11.7	LVH	B/L NHO +
29	556363	45	F	NO	NO	NO	YES	NO	NO	NO	NO	NO	SEIZURES	EPILEPSY	98	120	90/60					34						B/L COARSE CREPTS+	S1 S2 NORMAL	UNCONSCIOUS, POORLY RESPONDING TO COMMANDS	NO	NO	12	SINUS TACHYCARDIA	RIGHT LL NHO+
30	557297	82	F	YES	NO	NO	NO	YES	NO	NO	NO	YES	BURNING MICTURITION	T2DM.HTN.CAD	99	120	80/50					30						B/L CLEAR	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	10	LVH	NORMAL
31	557290	85	M	YES	NO	NO	NO	NO	YES	NO	NO	YES	BURNING MICTURITION	T2DM. HTN	100	100	90/60	90/60				30	28					B/L CLEAR	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	12	LVH	NORMAL
32	559470	40	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM	100	100	90/60	90/64	90/60	92/60	94/64	30	34	30	26	30		B/L ISA CREPTS +	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	16.9	POOR R WAVE PROGRESSION	B/L LL NHO+
33	560247	30	F	YES	NO	YES	YES	NO	NO	NO	NO	NO			100	100	80/60	90/62	94/60	100/60	110/60	30	32	30	28	26		B/L BASAL CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	4.9	SINUS TACHYCARDIA	LEFT LOWER ZONE NHO+
34	560243	70	M	YES	NO	NO	NO	YES	NO	NO	NO	YES	BURNING MICTURITION	T2DM,HTN	101	112	80/50	80/50				30	28					B/L CLEAR	S1 S2 NORMAL	ALTERED SENSORIUM, B/L PLANTAR: FLEXOR	NO	NO	13.3	SINUS TACHYCARDIA	NORMAL
35	560744	65	F	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM,HTN	102	120	100/60	100/60	90/60	80/50	90/60	30	34	30	28	30		B/L DIFFUSE RHONCHI+, CREPTS+	S1 S2 NORMAL	ALTERED SENSORIUM, RESTLESS & IRRITABLE	NO	NO	7.9	SINUS TACHYCARDIA	B/L DIFFUSE NHO+
36	561500	63	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM,HTN	102	120	140/70	130/80	140/90			36	34	30				B/L DIFFUSE RHONCHI+	S1 S2 NORMAL	CONSCIOUS, RESTLESS & IRRITABLE	NO	NO	14.1	NORMAL	B/L LOWER ZONE NHO+
37	728727	44	F	YES	NO	NO	NO	YES	NO	NO	NO	YES	BURNING MICTURITION	T2DM	101	120	80/50	84/60	90/60	100/70	110/60	30	28	26	22	20		B/L NVBS +	S1 S2 NORMAL	ALTERED SENSORIUM, B/L PLANTAR: FLEXOR	NO	NO	14.4	NORMAL	NORMAL
38	561951	68	F	NO	NO	YES	YES	NO	NO	NO	NO	NO		T2DM.HTN	98	120	110/60	110/74	110/70	120/80	124/70	36	30	28	24	20		B/L DIFFUSE RHONCHI+	S1 S2 NORMAL	RESTLESS, DROWSY	NO	NO	10.5	SINUS TACHYCARDIA	B/L LOWER ZONE NHO+
39	561980	35	F	YES	NO	NO	NO	YES	NO	NO	NO	NO			101	120	100/60	90/60	90/64	100/60	110/70	22	20	18	16	16		B/L NVBS +	S1 S2 NORMAL	DROWSY, NECK STIFFNESS+	NO	NO	13.9	NORMAL	NORMAL
40	561808	60	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM	98	120	90/60	100/60	110/70	110/80		36	34	30	20			B/L DIFFUSE RHONCHI+	S1 S2 NORMAL	CONSCIOUS, RESTLESS, IRRITABLE	NO	NO	13.3	SINUS RHYTHM	B/L LOWER ZONE NHO+
41	660167	75	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM.HTN.CAD	101	104	100/60	100/64	110/70	110/76	104/76	26	26	28	24	20		B/L BASAL FINE CREPTS+	S1 S2 NORMAL	DROWSY, RESPONDING TO COMMANDS	NO	NO	14.4	SINUS RHYTHM	B/L LOWER ZONE NHO+
42	658731	55	M	YES	NO	YES	YES	NO	NO	NO	NO	NO			100	120	150/90	160/80	150/90	140/90	140/90	36	34	30	26	24		B/L ISA CREPTS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	11.3	SINUS TACHYCARDIA	B/L LOWER ZONE NHO+
43	660188	85	F	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM	101	120	160/80	150/90				30	32					B/L DIFFUSE RHONCHI+	S1 S2 NORMAL	DROWSY, POORLY RESPONDING TO COMMANDS, B/L PLANTAR-FLEXOR, NO MENINGEAL SIGNS	NO	NO	10.6	LOW VOLTAGE COMPLEXES	LEFT UPPER AND LOWER ZONE NHO+
44	658875	30	M	YES	NO	YES	NO	NO	NO	NO	NO	NO			102	90	80/50	90/60	100/60	100/60	100/60	26	30	30	28	26		B/L BASAL FINE CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.7	SINUS TACHYCARDIA	B/L LUNG ZONE DIFFUSE INFILTRATES+
45	659821	53	F	NO	NO	YES	YES	NO	NO	NO	NO	NO		T2DM	98	90	100/60	100/70	110/60	110/70	100/60	26	24	26	22	20		B/L BASAL FINE CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	10.2	SINUS RHYTHM	B/L LOWER ZONE NHO+
46	658878	50	M	NO	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	T2DM,HTN	98	100	80/50	90/60	90/60	100/60	100/60	26	24	22	20	20		B/L CLEAR	S1 S2 NORMAL	DROWSY, POORLY RESPONDING TO COMMANDS, B/L PLANTAR-FLEXOR	NO	NO	11.6	LOW VOLTAGE COMPLEXES	NORMAL
47	659806	82	F	NO	NO	NO	NO	YES	NO	NO	NO	YES		HTN	99	92	170/40	160/100	150/100	150/10	150/90	24	22	24	22	20		B/L CLEAR	S1 S2 NORMAL	DROWSY, POORLY RESPONDING TO COMMANDS, B/L PLANTAR-FLEXOR, NO MENINGEAL SIGNS	NO	NO	12.6	SINUS RHYTHM	NORMAL
48	658483	20	F	NO	NO	YES	YES	NO	NO	NO	NO	NO			98	100	100/70	100/60	110/60	110/60	110/70	36	34	36	34	30		B/L BRONCHIAL BS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	14	SINUS RHYTHM	B/L UPPER ZONE INFILTRATES+
49	726062	67	M	YES	NO	NO	NO	NO	NO	NO	NO	NO	LEFT LOWER LIMB SWELLING	T2DM	99	84	90/60	100/60	100/60	110/60	100/70	20	20	22	24	20		B/L CLEAR	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	11.7	SINUS RHYTHM	NORMAL
50	719748	60	M	NO	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	B,ASTHMA	98	86	110/70	110/70	100/60	114/74	110/70	26	24	26	22	24		B/L CLEAR	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	9.8	NORMAL	NORMAL
51	657021	20	F	NO	NO	YES	YES	NO	NO	NO	NO	NO		T2DM,HTN	98	76	120/80	120/70	120/74	126/74	124/74	32	30	30	28	26		B/L ISA CREPTS +	S1 S2 NORMAL	CONSCIOUS, RESTLESS	NO	NO	14.7	SINUS RHYTHM	B/L LOWER ZONE NHO+
52	662895	51	M	YES	NO	NO	NO	NO	NO	NO	NO	NO	B/L LOWER LIMB SWELLING	T2DM,HTN	99	100	118/70	128/76	140/80	130/80	140/80	18	16	16	14	12		B/L NVBS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	11	NORMAL	NORMAL
53	665620	55	M	YES	NO	NO	YES	NO	NO	NO	NO	NO	B/L LOWER LIMB SWELLING	T2DM	101	98	90/60	108/60	110/60	100/60	104/60	24	28	28	28	26		B/L DIFFUSE RHONCHI+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	14	POOR R WAVE PROGRESSION	
54	665542	50	M	YES	NO	NO	NO	YES	NO	NO	NO	NO		T2DM	100	90	170/10	150/90	150/100	140/90	130/80	30	28	26	26	24		B/L CLEAR	S1 S2 NORMAL	ALTERED SENSORIUM, MENINGEAL SIGNS+	NO	NO	17	SINUS RHYTHM	NORMAL

	UrineRoutine	Blood/ Urine/ sputum C/S	Other investigations(If any)	GCS					Total leucocyte count(T/mm)					Platelet Count(L/cu.mm)					Serum Creatinine(mg/dl)					Serum bilirubin(mg/dl)					VentillatorSupport	iontropic support	Renal replacement	duration of icu stay	survived	SOFAscore					QSOFA- Score					DIAGNOSIS
				D1	D2	D3	D4	D5	D1	D2	D3	D4	D5	D1	D2	D3	D4	D5	D1	D2	D3	D4	D5	D1	D2	D3	D4	D5						D1	D2	D3	D4	D5	D1	D2	D3	D4	D5	
1	NORMAL	NO GROWTH	SPUTUM AFB-NEGATIVE	15	15	15	15	15	42T	24T	22.9T	12.29T	10.2T	204	164	159	168	146	3.2	2.4	1.6	1.5	1.2	2	2	2	2	2	2 D			7	YES	6	6	4	4	3	2	2	2	1	0	LRTI
2	NORMAL	NO GROWTH	CSF ANALYSIS: UNREMARKABLE	10	10	13	14	15	29.6	24.8	19.2	25.2	13.3	132	84	83	82	67	3.1	3	2.1	1.5	1.1	1.4	1.2	1	0.6	0.6	2D	4D	NO	2	YES	10	11	9	7	4	3	3	3	3	3	NEUROINFECTION
3	NORMAL	ET ASPIRATES C/S KLEBRILLA PNEMONIA,ACINOBACTER SPECIES	SPUTUM AFB-NEGATIVE	10	10	10	10	10	9.3	8.39	8.47	6.9	10	59T	44T	42T	49T	50T	0.8	0.7	0.8	1.1	1.2	0.9	0.9	0.9	0.9	0.9	5D	5D	NO	7	NO	7	8	8	9	11	3	3	3	3	3	LRTI
4	NORMAL	BLOOD C/S ,ET TUBE C/S -NO GROWTH	SPUTUM AFB-NEGATIVE	13	13	14	14	15	19.5	22	25.5	24	24	280	282	275	260	262	1	0.8	0.7	0.8	0.9	1.3	1.3	1.3	1.3	1.3	2D	3D	NO	5	YES	4	5	5	5	3	3	3	3	3	1	LRTI
5	NORMAL	ET ASPIRATES C/S NO GROWTH	SPUTUM AFB-NEGATIVE	13	13	14	14	15	20.8	21.4	21.4	16.6	16.8	459	476	482	558	500	0.8	0.6	0.6	0.6	0.6	1.2	1.2	1.2	1.2	1.2	3D	NO	NO	6	YES	2	2	2	1	0	2	2	2	2	0	LRTI
6	NORMAL	C/S ACENETOBCTER SPECIES	SPUTUM AFB-NEGATIVE	1	1	1	1	0	43.8	26.9	33.4	21.5	19.1	367	304	239	187	167	1.5	0.8	0.9	0.7	0.8	1.3	1.3	1.3	1.2	1.2	7D	6D	NO	5	NO	6	6	6	5	5	3	3	3	3	3	LRTI
7	NORMAL	ET ASPIRTES C/S CANDIDA ALBICANS ,ACINOBACTER SPECIES	SPUTUM AFB-NEGATIVE	12	12	13	13	13	11.2	14.6	11.7	7.5	5.15	99	100	79	74	79	2.5	2.7	2.6	2.2	1.7	3.6	3.4	3	2.3	2.3	3D	NO	NO	7	YES	10	10	8	7	6	2	2	2	2	1	LRTI
8	NORMAL	NO GROWTH	SPUTUM AFB-NEGATIVE	15	15	15	15	15	18.78	18	16	16	12	256	256	248	250	248	10.8	8	8.6	7	6.4	1.2	2	2	2	2	NO	NO	3 C	10	YES	5	6	6	5	5	1	1	1	1	1	LRTI
9	NORMAL	NO GROWTH	SPUTUM AFB-NEGATIVE	15	15	15	15	15	23.7	23.3	22	20	20	508	450	400	400	400	1.7	2	2	2	1.6	1.2	1.2	1.2	1	1	NO	NO		12	YES	4	4	4	3	2	1	1	1	1	0	LRTI
10	NORMAL		SPUTUM AFB-NEGATIVE	10	12				29	16.3				368	303									11.3	11				2D	NO		2	NO	12	15				3	3				LRTI,ARDS
11	NORMAL	URINE C/S- E COLI		12	12	14	14	15	13.9	8.19	9	10	10	194	202	200	200	200	1.7	1	1	0.8	0.7	0.6	0.6	0.6	0.6	0.6	NO	NO		5	YES	4	3	1	1	0	3	2	1	0	0	UROSEPSIS
12	PROTEIN 1+		CRP-POSITIVE	11	11	11	11	11	4.75	10.5	16.6	17	18	333	301	300	302	289	0.8	0.6	0.8	0.8	0.8	1.5	1.5	1.5	2	2	NO	NO	NO		NO	4	5	8	8	8	3	3	3	3	3	UROSEPSIS
13	NORMAL	ET ASPIRATE C/S KLEBSIELLA PNEUMONIA, ACINOBACTER		9	8	9	9	9	19.9	13	12	12	11	243	203	200	203	200	20	0.8	0.6	0.5	0.8	1.4	1.4	1.4	1.4	1.4	3D	2D	NO	7	YES	8	7	8	8	6	3	3	3	2	2	LRTI
14	NORMAL	ET ASPIRATE C/S KLEBSIELLA OXYTOCA	CRP-POSITIVE	13	12	13	13	15	3.05	4	4.5	4.6	4.8	164	200	230	220	200	1.8	1.2	1.4	1.4	1.4	0.8	0.8	0.8	0.8	0.8	4D	3D	NO	6	YES	6	7	4	4	1	3	3	3	2	1	LRTI
15	NORMAL	ET ASPIRATE C/S ACINETOBACTER SPECIES	CRP-POSITIVE	14	14	14	15	15	15.9	14	14	12	10	218	200	200	250	300	0.9	0.6	0.8	0.6	0.6	1	1	1	1	1	3D	2D	NO	7	YES	5	6	4	2	1	3	3	3	1	1	LRTI
16	PUS CELLS 3	NO GROWTH	CRP-POSITIVE	14	14	14	15	15	19.19	15.24	14	14	12	300	303	300	300	256	0.5	1.5	2	2	2	2	2	2.6	2	2	NO	2D	NO	8	YES	7	7	8	5	5	3	3	3	1	1	LRTI
17	NORMAL	NO GROWTH	CRP-POSITIVE	14	14	15	15	15	10.7	9.3	13.8	8.8	9	175	112	161	135	130	1.5	1.3	1	0.8	0.8	2	2	2	2	2	NO	3D	NO	8	YES	6	7	4	4	3	3	3	1	1	0	CELLULITIS
18	PROTEIN 1+	SPUTUM- ACINETOBACTER, KLEBSIELLA		13	14	14	14	15	15.6	17.6	17	20	20	163	184	170	170	150	4.4	5.2	5.8	3.3	2	0.6	0.6	0.6	0.6	0.8	NO	2D	2CY	5	YES	6	7	6	5	1	3	3	3	2	1	LRTI
19	NORMAL	BLOOD C/S- NO GROWTH		13	14	14	15	15	20.9	20.3	24	22	20	179	223	250	250	180	1.9	2.3	2.4	2.4	3	2	2	2.8	2.8	2	NO	3D	NO	3	YES	6	7	7	5	4	3	3	3	1	0	LRTI
20	NORMAL	NO GROWTH		14	13	15	15	15	20.9	20.3	20	22	20	179	223	240	200	200	1.3	1.2	3.2	3	3	2	2	2	2	2	NO	2D	NO	2	YES	6	6	6	4	4	3	3	2	0	0	LRTI
21	NORMAL	NO GROWTH		13	10	10			13.5	11.5	20			322	352	350			1.6	1.6	1.8			3	3	3			3D	3D	NO	3	NO	11	11	10	8	8	3	3	3	3	3	LRTI.ARDS
22		ET ASPIRATE C/S KLEBSIELLA PNEUMONIA , ACINETOBACTER SPECIES		10	10	13	13	14	21.2	22	22	22	22	402	400	350	350	350	7	7.8	6.6	6.5	7.8	2	2	2	2	2	4D	3D	NO	6	NO	11	11	10	8	8	3	3	3	3	3	LRTI.ARDS
23	NORMAL	NO GROWTH		10					30					150					1.2					1.3					1D	1D	NO	1	NO	9					3					LRTI.ARDS
24	NORMAL		CRP-POSITIVE	13	10				22					120					3					2					2D	NO	NO	2	NO	13	13				3	3				LRTI.ARDS
25	NORMAL	URINE C/S: E.COLI	CRP-POSITIVE	13	13	15	15	15	8.95	11.4	12.5	20	18	151	249	315	300	375	0.7	0.7	0.6	0.6	0.7	1	1	1	1	1	NO	3D	NO	3	YES	2	2	1	0	0	2	2	0	0	0	UROSEPSIS
26	PROTEIN 1+			14	15	15	15		14	16	18	12		226	200	201	204		1	1.4	1.3	1.2		1.2	1.3	1.3	1.3		4D	4D	NO	4	NO	4	4	4	2		3	2	1	1		LRTI.ARDS
27	PUS CELLS 3	NO GROWTH	SPUTUM AFB-NEGATIVE	14	14	10	10	10	15.59	16	20	26	37.3	306	300	305	350	375	0.8	1	1.4	1.7	1.7	9.5	10	10	11.7	11	4D	2D	NO	6	NO	6	7	9	9	9	3	3	3	2	2	LRTI.ARDS
28	NORMAL	NO GROWTH		12					17.9					295					0.7					0.7					1D	1D		1	NO	6					3					LRTI.ARDS
29	NORMAL	ET ASPIRATES C/S PSEUDOMONAS	CRP-POSITIVE	10					30					187					1					2					1D	1D	NO	1	NO	9					3					LRTI
30	PROTEIN 2+	URINE C/S- ACINETOBACTER	CRP-POSITIVE	10					22					100					3					2					1D	1D	NO	1	NO	12					3					UROSEPSIS
31	NORMAL		CRP-POSITIVE	10	10				20	22				100	80				1.2	1.8				1.2	1.2				2D	2D	NO	2	NO	10	11				3	3				UROSEPSIS
32	NORMAL		CRP-POSITIVE	12	12	12	10	10	7.9	8.2	9	10	12	153	155	100	96	113	1	0.8	0.6	0.5	0.5	2.5	2.3	2.2	2	1.8	6D	6D	NO	6	NO	12	13	14	16	17	3	3	3	3	3	LRTI.ARDS
33	NORMAL	ET C/S: ACINETOBACTER SPECIES		15	15	15	15	15	23.3	24.07	26	20	18	417	314	300	256	200	9.7	9.5	6.6	5.5	5.8	0.3	0.6	0.7	0.7	0.7	4D	3D	NO	5	YES	7	7	6	5	4	2	2	2	1	1	LRTI
34	NORMAL		CRP-POSITIVE	12	10				8.49	10.4				174	191				1.2	1.5				2	2				1D	2D	NO	2	NO	8	9				3	3				UROSEPSIS
35	NORMAL		CRP-POSITIVE	12	12	12	12	10	4.4	6.6	8	6.14	8.6	94	86	150	104	109	1	2.1	2.6	1.7	1.4	0.7	0.7	0.7	0.7	0.7	5D	3D	NO	5	NO	5	8	8	9	9	2	2	3	3	3	LRTI.ARDS
36		BLOOD C/S: NO GROWTH, ET ASPIRATE C/S:NO GROWTH, SPUTUM C/S: ACINETOBACTER SPECIES	CRP-POSITIVE	15	13	12			19.3	20	18			205	150	153			1.1	1.2	0.7			0.5	0.7	1.1			3D	3D	NO	3	NO	1	4	5			1	2	2			LRTI
37	NORMAL	SPUTUM C/S: NO GROWTH, URINE C/S: E.COLI	CRP-POSITIVE	13	13	13	14	15	26.9	20	18	16	12	181	222	145	140	160	0.9	0.9	0.6	0.7	0.7	1.2	1	1	1	0.8	NO	4D	NO	4	YES	5	4	4	3	1	3	3	3	1	0	UROSEPSIS
38	NORMAL		CRP-POSITIVE	13	14	13	13	12	17.7	18	16.5	15	14	494	519	486	356	358	0.5	0.5	0.6	0.6	0.5	1.2	1.3	1.2	1	1.2	2D	NO	NO	3	YES	5	4	3	1	3	2	2	2	2	1	LRTI
39	NORMAL		CRP-NEGATIVE, CSF ANALYSIS: PREDOMINANT NEUTROPHILS	12	12	12	13	14	19.2	18.2	16	12	7.12	433	400	402	356	371	0.6	0.6	0.6	0.8	0.8	1.6	1.8	1.6	1.6	1.2	NO	NO	NO	3	YES	3	4	4	2	2	1	2	2	1	1	MENINGITIS
40	NORMAL		CRP-POSITIVE	13	14	15	15		12.5	16	12	10		186	512	500	482		0.7	0.7	0.8	0.8		1.2	1.2	1.4	1		4D	2D	NO	4	NO	5	5	2	0		3	3	2	0		LRTI
41	NORMAL			13	13	14	15	15	20	18.5	16	14	13	237	305	300	250	256	2.8	2.4	4.8	6.4	3.5	1.3	1.6	1.5	1.2	1.2	NO	2D	2C	2	YES	5	5	6	5	4	2	2	2	1	0	LRTI
42	NORMAL		CRP-POSITIVE	15	15	13	12	12	8.8	11	14	10	16	407	393	386	345	333	0.9	0.8	0.8	0.7	0.6	1	1	1	1.2	1	5D	5D	NO	7	YES	1	2	3	5	5	1	1	2	2	2	LRTI.ARDS
43	NORMAL		CRP-POSITIVE	10	10				8.9	16				292	200				0.3	1.2				2	3				2D	2D	NO	2	NO	7	9				2	2				LRTI
44	NORMAL		CRP-POSITIVE , HIV, HBSAG- NEGATIVE	15	15	15	15	15	7.9	7.59	4.5	5	6.2	44	47	88	90	100	1.2	1.4	1.4	1.6	1.6	6.4	6.4	4.7	5.2	4.6	3D	NO	NO	5	YES	9	9	6	5	5	2	2	1	1	1	LRTI
45	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	15.01	14	13.6	13	12	362	300	352	300	342	1.7	1.6	1.7	1.6	1.2	1.6	1.5	1.5	1.4	1.2	2D	2D	NO	4	YES	4	4	3	3	2	1	1	1	1	1	LRTI
46	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	12	12	12	12	13	10.5	12	14	15	15	188	91	90	80	56	1.5	1	1.1	1.4	1.2	1.4	1.4	1.4	1.2	1	NO	3D	NO	3	YES	6	7	4	7	6	3	3	2	1	1	ACUTE GE
47	PUS CELLS- 10-15/HPF	URINE C/S: E.COLI	CRP-POSITIVE	12	12	13	12	12	20.18	12	10	9	8	365	354	350	200	256	1.4	1	1.2	1.2	1	1.1	1.2	1.2	1.2	1.2	NO	2D	NO	2	YES	3	3	3	4	3	2	1	2	1	1	UROSEPSIS
48	NORMAL		CRP-POSITIVE	15	15	15	15	15	9	7	8	8.5	8	313	311	316	350	348	0.4	0.5	0.5	0.6	0.6	1	1	1	1	1	3D	NO	NO	4	YES	2	2	1	1	0	1	1	1	1	1	LRTI.ARDS
49	PROTEIN 2+		CRP-POSITIVE	15	15	15	15	15	10.8	10.15	10	9	8	215	222	200	256	200	1.8	1.6	1.4	1.3	1.2	1.4	1.4	1.2	1.2	1.2	NO	2D	NO	2	YES	4	3	2	2	2	1	1	0	0	0	CELLULITIS
50	NORMAL		CRP-POSITIVE	15	15	15	15	15	7.6	8	10	12	12	183	150	132	122	100	1.5	1.6	1.8	1.4	1.2	1.4	1.2	1.2	1	1	NO	2D	NO	2	YES	2	3	3	2	3	0	0	0	0	0	ACUTE GE
51	NORMAL		CRP-POSITIVE	14	15	15	15	15	20.4	18.6	16.8	13.6	12	466	316	350	312	300	0.8	0.8	0.6	0.6	0.5	1.2	1.2	1.2	1.2	1	NO	NO	NO	2	YES	3	2	1	1	0	2	1	1	1	1	LRTI
52	PUS CELLS 8	URINE C/S- NEGATIVE	CRP- NEGATIVE	15	15	15	15	15	26	24	22	20	18	150	100	120	134	156	10.2	8.6	8.9	7.6	6.4	10.8	9.6	8.6	7.2	3.2	NO	NO	4C	4	YES	8	8	8	8	6	0	0	0	0	0	CELLULITIS
53	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	16.99	14.82	13.6	11.48	10.28	257	312	368	412	326	1.3	1.9	1.4	1.2	0.9	0.4	0.3	0.3	0.2	0.2	NO	2D	NO	2	YES	4	3	2	1	0	2	2	1	1	1	LRTI
54	NORMAL		CSF ANALYSIS: 10CELLS- 60%NEUTROPHILS, 40%LYMPHOCYTES	10	10	12	12	14	19	18	17	17	16	342	308	300	300	308	1	0.9	1	1.2	1.2	3.6	3.6	2.8	2.8	2.6	1D	NO	NO	2	YES	6	5	5	5	4	2	2	2	2	2	MENINGITIS

55	663496	47	F	NO	NO	YES	YES	NO	NO	NO	NO	NO		HYPOTHYROIDISM	98	90	150/10	146/90	140/90	136/84	130/80	20	20	18	18	20		B/L RHONCHI+, CERPTS + IN ISA	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	8.8	RBBB	B/L LOWER ZONE NHO+	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	15.89	14.81	10.2	12.05	11.59	431	297	321	293	321	4	3.6	2.8	2.9	4	0.4	0.4	0.3	0.3	0.3	1D	NO	NO	3	YES	4	4	2	2	3	2	2	1	1	1	LRTI
56	664374	62	F	YES	NO	NO	YES	NO	NO	NO	YES	NO	BURNING MICTURITION	T2DM.HTN	101	74	120/70	124/76	120/80	124/80	120/86	18	20	18	18	16		B/L ISA CREPTS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	4.7	SINUS RHYTHM	LEFT NHO+	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	8.67	8.57	9.23	9.51	9.17	445	423	438	456	448	3.5	4.1	3.8	3.6	3.8	0.3	0.3	0.2	0.4	0.4	NO	NO	NO	2	YES	3	3	3	3	3	2	2	1	1	1	LRTI
57	663815	95	F	NO	NO	NO	YES	YES	NO	NO	NO	NO		T2DM,HTN	98	100	200/10	160/100	160/100	150/90	150/90	28	28	26	24	24		B/L COARSE CREPITATIONS+	S1 S2 NORMAL	ALTERED SENSORIUM, NO MENINGEAL SIGNS	NO	NO	12.7	LVH+	RIGHT LL NHO+	NORMAL		CRP-POSITIVE	12	12	12	13	14	11	12	14	11	11	307	300	308	350	300	0.7	0.7	0.8	0.8	0.8	1.2	1.2	1.2	1.2	1.2	NO	NO	NO	2	YES	4	4	4	2	2	2	2	2	2	2	LRTI
58	665170	58	F	YES	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	T2DM	101	100	80/50	84/60	90/60	100/60	100/60	20	20	22	18	16		B/L CLEAR	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	11.6	SINUS TACHYCARDIA	NORMAL	NORMAL		CRP-POSITIVE	15	15	15	15	15	12.2	14	14	14.2	11	200	150	100	100	132	1.6	1.8	1.9	1.4	1.2	2	2	1.2	1.2	1	NO	3D	NO	3	YES	5	5	4	3	2	1	1	1	0	0	ACUTE GE
59	666395	87	F	NO	NO	YES	YES	NO	NO	NO	NO	NO		T2DM	98	86	200/11	200/100	180/100	170/90	174/96	26	28	30	28	22		B/L COARSE CREPITATIONS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	11.8	SINUS RHYTHM	LEFT LOWER ZONE NHO+	NORMAL	NO GROWTH	CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	13	13	13	15	14.5	13.8	13	12.6	12	210	256	212	216	200	1.3	1.5	2.8	3.2	3	1.2	1.2	1	0.8	0.8	2D	NO	NO	3	YES	2	3	3	3	2	1	2	2	2	0	LRTI
60	667367	85	F	YES	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	T2DM.HTN.COPD	99	100	80/60	80/60	90/60	90/60	90/60	20	20	18	20	18		B/L NVBS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	7.5	NORMAL	NORMAL	NORMAL		CRP-POSITIVE	15	15	15	15	15	4.97	5.1	5.9	6.2	7	288	208	151	123	162	2.7	2.5	2.3	2	1.8	1.2	1.2	1	1	1	NO	3D	NO	3	YES	5	5	3	3	1	1	1	1	1	0	ACUTE GE
61	664604	80	F	YES	NO	NO	NO	NO	NO	NO	NO	NO	RASHES	T2DM. COPD	101	100	130/80	80/50	80/60	90/60	100/60	26	28	24	22	20		B/L NVBS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.2	NORMAL	NORMAL	NORMAL		CRP-POSITIVE	15	14	14	14	15	23.8	30.2	18.4	12.3	10.2	341	366	287	287	266	0.7	2.7	1.8	1.6	0.8	0.9	1.2	1.2	1	0.9	NO	3D	NO	3	YES	1	5	4	2	0	1	3	3	2	0	UROSEPSIS
62	665163	70	F	YES	NO	YES	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	T2DM. CAD	101	102	60 SYS	80/60	84/60	90/60	100/60	36	34	30	28	24		B/L DIFFUSE RHONCHI+	S1 S2 NORMAL	ALTERED SENSORIUM, NO MENINGEAL SIGNS	YES	NO	10.7	ST DEPRESSION IN V4-V6	B/L LOWER ZONE NHO+	NORMAL		CRP-POSITIVE	13	13	13	14	15	35	23	20	17	9	435	238	170	140	120	1.8	2.3	1.7	1.6	1.6	0.5	0.5	0.6	0.8	0.8	2D	3D	NO	3	YES	8	6	5	4	2	3	3	3	3	1	LRTI
63	661265	29	M	NO	NO	YES	YES	YES	NO	NO	NO	NO			98	100	110/80	110/70	110/84	114/70	110/70	30	26	28	26	26		RIGHT ISA, IAA CREPTS+	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	13.7	SINUS RHYTHM	RIGHT LL NHO+	NORMAL		CRP-POSITIVE	12	13	14	15	15	11.2	11.6	12	13.6	13	260	280	264	284	320	0.9	0.9	1	1	0.9	1	1	1	1	1	NO	NO	NO	1	YES	2	1	1	0	0	2	2	2	1	1	LRTI
64	661226	65	F	YES	NO	NO	NO	YES	YES	NO	NO	NO	LOOSE STOOLS	T2DM	100	110	100/80	100/70	106/70	110/70	100/74	20	22	22	20	22		B/L CLEAR	S1 S2 NORMAL	DROWSY, PHOTOPHOBIA+, NECK STIFFNESS+	NO	NO	8.5	SINUS RHYTHM	NORMAL	NORMAL		CRP-POSITIVE	13	13	14	14	15	6.8	10	12.6	12.5	11	53	60	65	70	90	1.1	2.8	4.8	6.5	4.5	1.4	1.2	1.2	1.2	1	NO	NO	2C	2	YES	4	5	6	8	5	2	2	2	0	0	MENINGITIS
65	660897	30	F	YES	NO	YES	NO	NO	NO	NO	NO	NO			100	120	80/50	90/60	94/60	90/60	100/60	30	26	26	24	22		B/L BASAL CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	10.9	POOR R WAVE PROGRESSION	RIGHT LL NHO+	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	12	11.6	11.5	12	12	107	90	84	120	150	0.8	0.8	0.8	0.9	0.9	1	1	1	1	1	NO	4D	NO	4	YES	3	4	3	2	1	2	2	2	2	2	LRTI
66	729907	53	M	YES	NO	NO	NO	NO	NO	NO	NO	NO	RIGHT LOWER LIMB SWELLING	T2DM	101	100	130/70	134/74	140/80	140/86	140/84	24	26	22	20	20		B/L CLEAR	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.5	SINUS RHYTHM	NORMAL	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	8.1	12.8	9.6	10.1	11.5	342	389	368	437	400	16	15.4	14.7	12	10.6	0.9	1	1.2	1.2	0.9	NO	NO	6C	3	YES	4	4	4	4	4	0	0	0	0	0	CELLULITIS
67	636842	25	F	NO	NO	NO	YES	NO	NO	NO	NO	NO	SEIZURES	EPILEPSY	98	90	116/80	80/60				24	28					B/L COARSE CREPITATIONS+	S1 S2 NORMAL	DROWSY, MOVING ALL 4 LIMBS, B/L PLANTAR-MUTE	NO	NO	8	RBBB	RIGHT MID AND LOWER ZONE NHO+	NORMAL		CRP-POSITIVE	8	8				20	20				628	600				0.5	0.5				1	1				2D	1D	NO	2	NO	5	8				2	3				LRTI
68	636437	58	F	YES	NO	NO	YES	NO	NO	NO	NO	NO	B/L LOWER LIMB SWELLING	T2DM	100	90	70 SYS	100/60	100/60	110/60	110/70	30	30	28	26	24		B/L BASAL CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	13.9	LOW VOLTAGE COMPLEXES	NORMAL	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	19.59	19.87	19.8	18	16.8	186	138	136	140	160	1.7	0.8	0.6	0.8	1	0.9	0.9	0.9	0.9	0.9	2D	4D	NO	4	YES	5	4	3	2	0	2	2	2	1	1	CELLULITIS
69	634895	80	M	NO	NO	YES	NO	NO	YES	NO	NO	NO		T2DM, IHD	98	118	130/80	120/70	120/84	110/70	100/70	30	26	20	20	16		B/L BASAL CREPTS+	S1 S2 NORMAL	ALTERED SENSORIUM, B/L PLANTAR: MUTE	NO	NO	8.5	SINUS TACHYCARDIA	B/L MID AND LOWER ZONE NHO+	PROTEIN 1+		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	9	10	10	10	10	16.84	16.02	10.9	11.9	9	471	375	345	424	421	1	1.1	1.4	1.4	1.3	1.6	1.6	1.6	1.6	1.6	5D	NO	NO	6	YES	5	4	5	5	5	2	2	2	2	2	LRTI
70	635492	49	F	YES	NO	NO	NO	NO	NO	NO	NO	NO	BURNING MICTURITION	T2DM	99	120	90/50	100/64	100/64	110/70	130/86	30	26	28	24	20		B/L NVBS +	S1 S2 NORMAL	CONSCIOUS, RESTLESS & IRRITABLE	NO	NO	10.5	SINUS TACHYCARDIA	NORMAL	PUS CELLS 6		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	14	12	12	10	14	13.53	15.27	14.1	13.1	12.47	120	150	138	196	209	1.1	1.6	0.8	0.8	0.6	1.4	1.6	1.6	1.6	1.6	NO	3D	NO	3	YES	4	6	5	4	3	3	3	3	3	3	UROSEPSIS
71	636341	50	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM.HTN	100	120	120/80	110/70	100/60	100/64	110/70	36	30	26	24	24		RIGHT ISA, IAA CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	YES	YES	10.7	SINUS RHYTHM	RIGHT LL NHO+	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	13	14	15	15	15	7.25	8.19	10	12	14	70T	75T	56T	70	100	2.4	3.2	3.6	3.8	4.2	5.5	5	4.8	4.6	3.2	3D	NO	NO	4	YES	9	9	7	6	6	1	1	0	0	0	LRTI
72	635051	45	M	YES	NO	NO	YES	NO	NO	NO	NO	NO	ABDOMINAL DISTENSION+		100	94	90/60	100/60	100/70	104/70	120/80	26	24	26	26	28		DECREASED BREATH SOUNDS IN RIGHT ISA, IAA. B/L COARSE CREPTITAIONS IN BASAL AREAS	S1 S2 NORMAL	CONSCIOUS, ORIENTED	YES	YES	13.2	LOW VOLTAGE COMPLEXES IN LIMB LEADS	B/L MID AND LOWER ZONE NHO+	NORMAL	BLOOD C/S AND SPUTUM C/S-NEGATIVE	CRP- POSITIVE, NEGATIVE FOR MALARIA, LEPTOSPIRA, DENGUE	15	15	15	15	15	9.34	10.9	15	11.8	10	60T	50T	86T	97T	87T	1	1.2	1.1	1	0.8	5	5	7	7	5	NO	3D	NO	3	YES	8	9	7	7	7	3	3	3	3	3	LRTI
73	634661	60	F	YES	NO	NO	NO	YES	YES	NO	NO	NO		T2DM.HTN	101	90	134/80	132/80	126/86	134/80	120/80	22	24	26	24	26		B/L NVBS +	S1 S2 NORMAL	DROWSY, MOVING ALL 4 LIMBS, B/L PLANTAR-FLEXOR	NO	YES	11.1	SINUS RHYTHM	NORMAL	NORMAL		CRP-POSITIVE, NEGATIVE FOR MALARIA, DENGUE	12	13	13	15	15	11.47	14.24	11.1	15.81	16.1	260	269	217	255	224	10.7	8.7	9.5	7.9	5	0.8	0.8	0.8	0.8	0.8	3D	NO	4C	4	YES	7	7	6	5	4	2	2	2	2	2	LRTI
74	648607	55	F	YES	NO	NO	NO	NO	YES	NO	NO	NO		HYPOTHYROIDISM	102	102	70/60	80/50	90/50	90/60	100/60	30	34	30	30	26		LEFT ISA CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	9.4	SINUS TACHYCARDIA	LEFT LOWER ZONE NHO+	NORMAL		CRP-POSITIVE	15	15	15	15	15	16	20	17	16	12	162	101	100	120	136	2.7	1.4	0.9	1.2	1.4	6	6	5.2	5	4.8	NO	4D	NO	4	YES	7	7	5	5	4	2	2	2	2	1	LRTI
75	649110	80	F	YES	NO	NO	NO	YES	NO	NO	NO	NO	LOOSE STOOLS	T2DM, HTN	99	120	130/70	140/80	130/70	120/80	130/70	30	26	24	26	30		B/L NVBS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED, NECK STIFFNESS+	NO	NO	11.1	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSTIVE	12	12	14	14	15	10.45	9.84	9.32	10	11.5	8	20	46	50	64	1.1	1.5	1.4	1.6	1.5	8	8	7.2	7	7.2	2D	NO	NO	3	YES	11	12	10	9	7	2	2	2	2	1	ACUTE GE
76	648719	80	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM.CAD	99	120	110/70	100/60	110/74	108/70	100/70	30	32	36	30	28		B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS, RESTLESS, IRRITABLE	NO	NO	11.9	RBBB	RIGHT LL NHO+	NORMAL		CRP-POSITIVE	14	14	15	15	15	29.3	26	22	23	18	44	50	66	98	140	1.1	1.2	1.1	1	1.2	2.6	2.6	2.6	2	2	NO	NO	NO	2	YES	6	7	5	4	4	2	2	1	1	1	LRTI
77	648041	65	F	YES	NO	NO	YES	NO	NO	NO	NO	NO		T2DM. CAD	100	90	80/60	80/50	90/60	80/50	100/60	26	24	26	28	30		LEFT ISA, IAA CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.2	POOR R WAVE PROGRESSION	LEFT LOWER ZONE NHO+	NORMAL		CRP- POSITIVE	14	15	15	15	15	6.39	6.89	5.95	4.66	5.02	80	108	111	104	120	0.7	0.8	0.7	0.6	0.5	2.7	2.6	1.8	1	1.8	NO	4D	NO	4	YES	7	5	4	2	2	3	3	3	3	3	LRTI
78	648166	35	M	YES	YES	NO	NO	NO	NO	NO	NO	NO		T2DM	101	96	100/60	104/62	110/70	110/74	120/70	30	28	26	24	22		B/L NVBS +	S1 S2 NORMAL	CONSCIOUS, RESTLESS, NECK STIFFNESS+, KERNIG`S SIGN +	NO	NO	14.6	SINUS RHYTHM	NORMAL	NORMAL			15	15	15	15	15	8.2	15.07	16	16.36	18.1	99	107	90	86	90	4.3	3.9	3.6	3	3.4	3.8	3.8	3.6	2.6	2.4	NO	4D	NO	4	YES	8	7	8	6	7	1	1	1	1	1	MENINGITIS
79	648748	60	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM, HTN	102	90	110/70	90/60	80/50	100/60	110/70	30	26	26	30	26		RIGHT ISA CREPTS+	S1 S2 NORMAL	CONSCIOUS, RESTLESS, IRRITABLE	NO	NO	16.3	SINUS TACHYCARDIA	RIGHT MID AND LOWER ZONE NHO+	NORMAL		CRP- POSITIVE	14	14	14	15	15	8.24	12	12	10	8.24	27	30	45	60	90	1.1	1.2	1.4	1.4	1	4.8	4.8	4.8	4	4	NO	4D	NO	4	YES	8	10	10	8	6	2	3	3	2	1	LRTI
80	648324	55	M	YES	NO	NO	NO	NO	NO	NO	NO	NO	FACIAL PUFFINESS+	HTN	101	110	170/10	150/100	140/100	150/90	150/90	26	22	24	24	26		B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	10	SINUS RHYTHM	B/L LOWER ZONE INFLITRATES+	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	14.4	16.04	22.7	20	18	50	74	102	100	150	4.9	6.5	6.7	5.7	5	1.6	1.6	2	1.8	1.8	NO	NO	6C	4	YES	8	7	7	7	6	1	0	1	1	1	LRTI
81	645554	65	M	NO	NO	NO	NO	NO	NO	NO	NO	NO	WOUND OVER RIGHT FOOT	T2DM, IHD	98	90	140/10	130/80	140/90	130/84	126/80	26	24	30	26	22		B/L NVBS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	11.2	SINUS RHYTHM	NORMAL	NORMAL		CRP-POSITIVE	15	15	15	15	15	9.32	10.4	11.4	10.6	12	346	368	460	350	268	0.9	1.1	1.3	1.3	1.4	2	2	2	1.8	1.6	NO	NO	NO	2	YES	2	2	3	2	2	1	1	1	1	0	CELLULITIS
82	647672	73	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM.HTN	100	80	150/10	140/100	150/90	140/10	150/90	26	28	30	26	24		RIGHT ISA, IAA CREPTS+	S1 S2 NORMAL	DROWSY, NO MENINGEAL SIGNS	NO	NO	15.1	POOR R WAVE PROGRESSION	RIGHT LL NHO+	NORMAL			12	12	10	10	12	6.8	7	7.4	9	10	126	118	100	140	132	2	1.6	1.8	2	1.6	1.2	1.4	1.6	1.4	1.2	NO	NO	NO	2	YES	6	5	6	6	5	2	2	2	2	2	LRTI
83	645163	80	M	YES	NO	NO	YES	NO	NO	NO	NO	NO		T2DM	100	90	90/50	80/60	80/50	100/60	100/70	30	28	26	24	26		B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	15.6	SINUS RHYTHM	B/L LL NHO+	NORMAL		CRP-POSITIVE	15	15	15	15	15	11.79	10.52	18.5	21.6	15.8	76	62	97	196	243	4.7	4.9	4.4	4.4	5.9	6.6	7	8.2	8	7.6	2D	5D	NO	5	YES	12	11	10	7	7	2	2	2	2	1	LRTI
84	646768	65	F	YES	NO	NO	NO	YES	NO	NO	NO	NO		T2DM, HTN	101	90	100/60	102/64	100/60	108/64	110/70	26	22	24	26	24		B/L NVBS +	S1 S2 NORMAL	ALTERED SENSORIUM, NECK STIIFNESS+, KERNIG`S SIGN +	NO	NO	10.8	SINUS TACHYCARDIA	NORMAL	NORMAL		CRP-POSITIVE, CSF ANALYSIS: CELLS CONT-10, PREDOMINANTLY	12	12	11	12	12	9	12	16	14	12	47	56	62	78	102	2.1	3.2	2.8	2.6	1.6	1.2	1.2	1.2	1	1	2D	NO	NO	3	YES	10	8	7	6	5	2	1	2	2	2	MENINGITIS
85	643280	50	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM	102	90	120/80	110/70	116/74	110/76	120/70	26	28	26	20	22		B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.7	SINUS RHYTHM	B/L LL NHO+	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	27.8	26	19.7	19	18	288	254	250	260	280	0.8	0.8	1	1.2	1.2	1.2	1.2	1	1	1.2	NO	NO	NO	2	YES	2	2	0	1	2	1	1	1	1	1	LRTI
86	646296	76	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM, HTN	100	100	120/80	124/82	110/70	116/74	120/80	30	32	26	24	22		RIGHT ISA CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	14	NORMAL	RIGHT LL NHO+	NORMAL	NEGATIVE	CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	16	14	14	12	10	100	95	90	100	126	1.2	1.2	1.2	1.4	1.6	1.2	1.2	1	1	1	NO	NO	NO	1	YES	5	6	4	3	3	1	1	1	1	1	LRTI
87	646401	56	M	YES	NO	NO	NO	YES	NO	NO	NO	NO	BURNING MICTURITION	T2DM	101	130	130/80	120/70	110/70	100/70	100/74	32	30	26	24	24		B/L NVBS +	S1 S2 NORMAL	DROWSY, RESPONDING TO COMMANDS	NO	NO	10.4	SINUS TACHYCARDIA	NORMAL	PUS CELLS 20.	URINE C/S: E.COLI	CRP-POSITIVE, HIV, HBSAG-NEGATIVE	12	12	13	13	14	19.4	20.43	18	16	14	326	342	340	342	340	0.3	0.3	0.4	0.8	0.7	1.2	1.2	1.2	1.2	1.2	NO	1D	NO	1	YES	3	3	2	2	2	2	2	2	2	2	UROSEPSIS
88	646252	53	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM, HTN	100	90	160/10	120/70	120/70	110/70	110/70	26	24	24	22	20		B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	14.1	NORMAL	B/L LL NHO+	PUS CELLS 7	NO GROWTH	CRP-POSITIVE, HIV, HBSAG-NEGATIVE	14	14	15	15	15	14.37	16	14	12	10	274	270	260	268	270	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	NO	NO	NO	1	YES	2	1	0	0	0	2	2	1	0	0	LRTI
89	665638	36	M	NO	NO	NO	YES	YES	NO	NO	NO	NO		B,ASTHMA	98	112	100/60	100/60	90/60			30	34	36				LEFT LUNG FILEDS CREPTS+	S1 S2 NORMAL	DROWSY, NO MENINGEAL SIGNS	NO	NO	9	SINUS TACHYCARDIA	LEFT LUNG DIFFUSE NHO+	NORMAL		CRP-POSITIVE	14	13	12			20	22	20			100	96	95			4.6	4.8	5.2			4.8	5	5.2			2D	2D	NO	2	NO	10	12	15			2	2	2			LRTI
90	732034	70	F	YES	NO	NO	NO	NO	NO	NO	NO	NO	FACIAL PUFFINESS+	HTN	102	100	120/79	110/80	160/100	130/80	120/76	26	22	24	24	26		B/L ISA CREPITATIONS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	N	10	LOW VOLTAGE COMPLEXES	B/L LL NHO+	NORMAL			15	15	15	15	15	14.3	16.6	22.7	20	18	50	74	105	100	150	4.9	6.5	6.7	5.7	5	1.6	1.6	2	1.8	1.8	NO	NO	6C	4	YES	8	7	7	7	6	1	0	1	1	1	LRTI
91	735316	35	M	YES	YES	NO	NO	YES	NO	NO	NO	NO			100	90	15/90	146/80	130/70	134/74	140/80	24	26	24	20	18		B/L NVBS+	S1 S2 NORMAL	KERNIGS SIGN +	NO	N	13	NORMAL	NORMAL	NORMAL			12	12	13	13	14	5.47	13.1	11.8	18.9	14.1	98	100	88	34	33	1.2	2.9	2	1.4	1	0.8	0.8	1.2	1	0.8	NO	NO	NO	2	YES	5	6	6	5	4	2	2	2	1	1	MENINGITIS
92	647252	60	F	YES	NO	NO	NO	YES	NO	NO	NO	NO		T2DM, HTN	99	100	90/60	90/60	90/64			30	28	26				B/L NVBS+	S1 S2 NORMAL	DROWSY, NECK STIFFNESS+	NO	NO	12.6	LVH	NORMAL	NORMAL		CRP- POSITIVE, CSF ANALYSIS: CELLS-8-NEUTROPHILS	12	10	10			28	36	34			50	48	32			1.8	1.6	1.8			3.2	2.8	2.9			2D	3D	NO	3	NO	12	14	15			3	3	3			MENINGITIS
93	649085	55	M	NO	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	HTN	98	120	80/50					34						B/L BASAL CREPTS+	S1 S2 NORMAL	RESTLESS, IRRITABLE	NO	NO	12.5	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSITIVE	14					27.3					260					1.5					0.9					1D	1D	1C	1	NO	9					3					ACUTE GE
94	728121	73	M	YES	NO	NO	NO	NO	NO	NO	NO	NO	LOWER LIMB SWELLING	DM	99	140	80/50					36						B/L NVBS+	S1 S2 NORMAL	DROWSY	NO	NO	12.6	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSITIVE	12					32					30					1.6					2.1					1D	1D	NO	1	NO	14					3					LRTI.ARDS
95	716249	94	M	NO	NO	NO	NO	YES	NO	NO	NO	YES		HTN	99	92	170/40	160/100	150/100	150/10	150/90	24	22	24	22	20		B/L CLEAR	S1 S2 NORMAL	DROWSY, POORLY RESPONDING TO COMMANDS, B/L PLANTAR-FLEXOR, NO MENINGEAL SIGNS	NO	NO	12.6	SINUS RHYTHM	NORMAL	PUS CELLS- 10-15/HPF	URINE C/S: E.COLI	CRP-POSITIVE	12	12	13	12	12	20.18	12	10	9	8	365	354	350	200	256	1.4	1	1.2	1.2	1	1.1	1.2	1.2	1.2	1.2	NO	2D	NO	2	YES	3	3	3	4	3	2	1	2	1	1	UROSEPSIS
96	652296	45	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		HTN	100	130	110/70	114/76	120/70			36	32	34				B/L DIFFUSE CRACKLES+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.8	RBBB	B/L NHO+ (L>R)	NORMAL	NO GROWTH	CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15			21	20	16			150	126	134			1.2	1.4	1.6			1.2	1.8	1			3D	2D	NO	3	NO	4	4	3			1	1	1			LRTI
97	717976	96	M	YES	NO	NO	NO	NO	NO	NO	NO	YES	BURNING MICTURITION	T2DM.HTN	102	100	90/60	90/60	90/64	100/60	100/60	12	14	16	16	14		B/L NVBS +	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	11.2	NORMAL	NORMAL	PUS CELLS 14	URINE C/S: E.COLI	CRP-POSITIVE	13	13	15	15	15	8.95	11.4	12.5	20	18	151	249	315	300	375	0.7	0.7	0.6	0.6	0.7	1	1	1	1	1	NO	3D	NO	3	YES	2	2	1	0	0	2	2	0	0	0	UROSEPSIS
98	653327	55	F	YES	NO	YES	YES	YES	NO	NO	NO	NO		T2DM.HTN	101	108	90/70	100/60				32	30					B/L BRONCHIAL BREATH SOUNDS+	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	12.1	SINUS TACHYCARDIA	B/L NHO+	NORMAL	NO GROWTH	CRP-POSITIVE, HIV, HBSAG-NEGATIVE	13	12				12	14				198	123				0.6	0.8				0.8	0.8				2D	2D	NO	2	NO	4	8				3	3				LRTI
99	653585	65	F	YES	NO	NO	NO	YES	NO	NO	NO	YES		T2DM	100	120	80/50	90/60	90/64	100/60	100/64	34	30	28	26	26		B/L NVBS+	S1 S2 NORMAL	ALTERED SENSORIUM, NO MENINGEAL SIGNS	NO	NO	6.3	RBBB	NORMAL	PUS CELLS 10	URINE C/S : E.COLI	CRP-POSITIVE, HIV, HBSAG-NEGATIVE	12	12	13	13	13	7.7	7.59	7.9	21.3	22.2	139	101	76	66	37	3.3	3.5	2.4	3.6	2.7	26.5	23.9	20.8	15.4	7.4	4D	6D	4C	6	NO	14	16	16	14	14	3	3	3	3	3	UROSEPSIS
100	655316	45	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		HYPOTHYROIDISM	100	98	100/70	100/60				36	32					B/L COARSE CRACKLES+	S1 S2 NORMAL	RESTLESS, IRRITABLE	YES	NO	12.9	SINUS RHYTHM	B/L PATCHY NHO+	NORMAL		CRP- POSITIVE	12	13				18	21				179	160				2.2	2				6.7	6.6				2D	2D	NO	2	NO	11	9				3	3				LRTI.ARDS
101	666217	55	M	YES	NO	NO	NO	YES	NO	NO	NO	YES		T2DM	101	140	70/40					30						B/L NVBS+	S1 S2 NORMAL	ALTERED SENSORIUM, NO MENINGEAL SIGNS	NO	NO	14.7			NORMAL			12					16					235					0.7					2.1					1D	1D	NO	1	NO	10					3					UROSEPSIS
102	667377	21	M	YES	NO	YES	YES	NO	NO	NO	NO	NO			102	130	100/60	90/60	80/50	80/50		46	42	40	30			B/L CRACKLES+	S1 S2 NORMAL	ALTERED SENSORIUM, RESTLESS, IRRITABLE	NO	NO	14.8	SINUS TACHYCARDIA	B/L DIFFUSE NHO+	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	13	12	10	10		10	12.17	19.1	14.7		102	140	155	150		0.8	1	0.9	1		0.7	0.8	0.9	1		4D	3D	NO	4	NO	5	7	8	9		2	3	3	3		LRTI
103	668102	45	M	NO	NO	YES	YES	YES	NO	NO	NO	NO		T2DM	98	140	100/60	94/60				42	46					B/L DIFFUSE CRACKLES+	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	14.7	SINUS TACHYCARDIA	B/L DIFFUSE NHO+	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	12	10				6.5	5.2				45	29				1.2	1.4				1.6	2.4				2D	1D	NO	2	NO	10	13				2	3				LRTI.ARDS
104	668166	70	M	YES	YES	NO	NO	YES	NO	NO	NO	NO		COPD	101	90	160/90	150/80	140/80	150/10	140/90	24	26	24	20	18		B/L NVBS+	S1 S2 NORMAL	ALTERED SENSORIUM, KERNIG`S SIGN +	NO	NO	13.1	NORMAL	NORMAL	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE, CSF ANALYSIS:CELL COUNT-6-NEUTROPHILS, SUGARS 20, PROTEIN 40	12	12	13	13	14	5.47	13.1	11.8	18.9	14.1	98	100	88	34	33	1.2	2.9	2	1.4	1	0.8	0.8	1.2	1	0.8	NO	NO	NO	2	YES	5	6	6	5	4	2	2	2	1	1	MENINGITIS
105	724010	77	M	YES	NO	NO	YES	NO	NO	NO	NO	NO	B/L LOWER LIMB SWELLING	T2DM	100	90	70 SYS	100/60	100/60	110/60	110/70	30	30	28	26	24		B/L BASAL CREPTS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	13.9	LOW VOLTAGE COMPLEXES	NORMAL	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	19.59	19.87	19.8	18	16.8	186	138	136	140	160	1.7	0.8	0.6	0.8	1	0.9	0.9	0.9	0.9	0.9	2D	4D	NO	4	YES	5	4	3	2	0	2	2	2	1	1	CELLULITIS
106	669004	65	M	NO	NO	YES	NO	YES	NO	NO	NO	NO	LEFT SIDED CHEST PAIN	T2DM, HTN. COPD	99	120	50 SYS	70/50	90/40	90/60	100/60	30	34	30	30	28		LEFT ISA CRACKLES+	S1 S2 NORMAL	ALTERED SENSORIUM, NO MENINGEAL SIGNS	YES	NO	6.5	NORMAL	LEFT LL NHO+	NORMAL	NO GROWTH	CRP-POSITIVE, HIV/HBSAG-NEGATIVE	10	12	12	12	14	7.28	12.5	10.9	21.6	18	140	101	76	66	80	3.3	3.5	2.4	3.6	3	26.5	24.8	23.9	20	16.2	3D	4D	2C	4	YES	16	17	16	16	11	3	3	3	3	3	LRTI
107	671500	69	M	NO	NO	YES	YES	NO	NO	NO	NO	NO		PTB. COPD	99	90	110/60	90/60	90/64	100/60	110/64	36	40	42	36	30		B/L CRACKLES + IN ALL LUNG FIELDS	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	11.2	P PULMONALE+, PAD+	RIGHT UL,LL NHO+	NORMAL	SPUTUM AFB +	CRP-POSITIVE, HIV/HBSAG-NEGATIVE	15	15	15	15	15	6.46	5.49	6.98	7.2	7.4	161	141	156	160	172	0.9	0.9	0.9	0.9	0.9	1	1.2	1.2	1	1	NO	2D	NO	2	YES	2	6	4	2	0	1	2	2	2	1	LRTI
108	671460	35	M	YES	NO	NO	NO	NO	NO	NO	NO	NO	LEFT LOWER LIMB SWELLING	T2DM	101	100	100/60	90/60	80/50	90/60	100/60	28	26	22	20	18		B/L NVBS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	9	SINUS RHYTHM	NORMAL	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	15	15	15	15	15	26	35	30	28	26	191	107	98	102	116	2.6	2	1.8	1.6	1.4	2.4	2.2	2.6	1.8	1.8	NO	3D	NO	3	YES	8	8	8	4	3	2	2	1	1	0	CELLULITIS
109	671278	38	F	YES	NO	NO	NO	YES	NO	NO	NO	NO			102	130	90/60	80/50				38	36					B/L NVBS+	S1 S2 NORMAL	DROWSY, SIGNS OF MENINGEAL IRRITATION +	NO	NO	12.8	NORMAL	NORMAL	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE, CSF ANALYSIS: CELLS-8 NEUTROPHILS	12	11				18.8	24				50	38				1.2	1.8				4.5	4.5				2D	2D	NO	2	NO	14	16				3	3				MENINGITIS
110	671935	72	M	YES	NO	NO	NO	YES	YES	NO	NO	NO		T2DM. CAD	101	80	110/70	90/50	80/50			36	40	35				B/L NVBS+	S1 S2 NORMAL	DROWSY, B/L PLANTA- FLEXOR	NO	NO	11.5	SINUS TACHYCARDIA	NORMAL	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	12	10	10			6.14	10.2	12.6			27	36	26			6.8	5.9	6.2			7.2	7.2	7.2			2D	2D	1C	2	NO	14	16	18			2	3	3			UROSEPSIS
111	671880	65	M	YES	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	COPD	101	110	30 SYS	90/60	90/60	100/60	110/60	18	20	20	18	18		B/L NVBS+	S1 S2 NORMAL	DROWSY, RESPONDING TO COMMANDS	NO	NO	10	SINUS RHYTHM	NORMAL	NORMAL	NO GROWTH	CRP-POSITIVE, HIV/HBSAG-NEGATIVE	14	15	15	15	15	18	16	12	11	10.6	190	150	132	118	96	1.4	1.4	1.6	1.8	1.4	4	4.6	5.2	4.8	3.4	NO	3D	NO	3	YES	8	8	8	6	6	2	1	1	1	0	ACUTE GE
112	671945	40	F	YES	NO	NO	YES	NO	NO	NO	NO	NO		T2DM	102	60	70/50					40						B/L BASAL CREPTS+	S1 S2 NORMAL	DROWSY, RESPONDING TO COMMANDS, B/L PUPIL-REACTIVE	NO	NO	4.3	SINUS BRADYCARDIA, VPC +	B/L LOWER ZONE NHO+	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	13					8.6					188					0.6					2.3					1D	1D	NO	1	NO	9					3					LRTI
113	671947	81	M	YES	NO	NO	NO	YES	NO	NO	NO	NO	BURNING MICTURITION	T2DM	101	90	80/50	90/60	100/60	110/64	112/68	40	42	40	38	36		B/L NVBS+	S1 S2 NORMAL	DROWSY, POORLY RESPONDING TO COMMANDS	NO	NO	14	NORMAL	NORMAL	PUS CELLS 16	URINE C/S- E.COLI	CRP-POSITIVE	12	12	13	13	15	11.5	13.5	18.3	20.3	22.4	189	160	150	138	112	1.8	1.6	1.2	1.1	0.8	1.2	1.2	1.2	1.1	1	5D	4D	NO	5	NO	7	9	9	10	9	3	3	3	3	2	UROSEPSIS
114	672488	70	M	YES	NO	NO	NO	YES	NO	NO	NO	NO		T2DM	101	120	80/50	90/60				36	34					B/L NVBS+	S1 S2 NORMAL	DROWSY	NO	NO	10.6	SINUS RHYTHM	NORMAL	PUS CELLS 10-12	URINE C/S- E.COLI	CRP-POSITIVE	12	10				12.9	22.4				50	81				0.9	1.4				0.9	1.2				2D	2D	NO	2	NO	11	14				3	3				UROSEPSIS
115	672794	63	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM, HYPOTHYROIDISM	102	120	100/60	100/64	80/60			46	30	32				B/L DIFFUSE CRACKLES+	S1 S2 NORMAL	RESTLESS, IRRITABLE	NO	NO	10.5	LVH, LOW VOLTAGE COMPLEXES	B/L DIFFUSE NHO+	NORMAL	ET ASPIRATE - ACINETOBACTER	CRP-POSITIVE	14	12	12			16.8	26.5	28.4			268	294	200			2.9	4.4	4.8			2.1	2.6	2.4			1D	1D	NO	1	NO	8	11	13			2	2	3			LRTI
116	673122	32	M	YES	NO	YES	YES	NO	NO	NO	NO	NO			99	120	100/64	90/60	80/50	80/54	80/50	40	30	30	28	30		B/L DIFFUSE CRACKLES+	S1 S2 NORMAL	DROWSY, RESTLESS	NO	NO	16.9	SINUS TACHYCARDIA	B/L DIFFUSE NHO+	NORMAL	ET ASPIRATE - KLEBSIELLA PNEUMONIAE	CRP-POSITIVE	13	13	12	12	10	5.8	8.4	15.4	19.9	22	137	147	170	188	160	0.7	0.7	3.7	5.2	5.7	0.5	1.6	2.1	2.6	3.2	5D	4D	1C	5	NO	5	7	13	15	16	2	3	3	3	3	LRTI
117	673240	36	F	YES	NO	NO	YES	NO	NO	NO	NO	NO		HYPOTHYROIDISM	99	80	110/80	100/80	110/70	110/60	100/70	32	30	28	26	26		B/L ISA CREPITATIONS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	16.3	SINUS RHYTHM, LOW VOLTAGE COMPLEXES	B/L LOWER ZONE NHO+	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	15	15	15	15	15	11.7	12.5	14.6	16.2	14.8	314	262	276	256	286	0.5	0.5	0.4	0.4	0.4	2	2	2	1.8	1.8	2D	NO	NO	3	YES	5	5	4	2	2	1	1	1	1	1	LRTI

118	673528	50	F	YES	NO	NO	NO	YES	NO	NO	NO	NO	BURNING MICTURITION	T2DM, IHD	99	120	80/50	70/40				30	28					B/L NVBS+	S1 S2 NORMAL	DROWSY	YES	NO	9.4	LVH	NORMAL	PUS CELLS 16-18	URINE C/S- E.COLI	CRP-POSITIVE	12	10				11.2	6.5				61	83				0.7	0.6				0.8	0.8				2D	2D	NO	2	NO	11	12				3	3				UROSEPSIS
119	402090	73	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		COPD.T2DM.CAD	100	100	140/80	136/80	140/82	136/80	140/80	40	36	36	38	30		LEFT ISA CRACKLES+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	14.9	SINUS TACHYCARDIA	LEFT LL NHO+	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	14	15	15	15	15	9.12	9.16	10.2	11.2	10.8	250	236	240	256	286	1.1	1.3	1.4	1.2	1.1	1.4	1.4	1.2	1.2	0.8	2D	NO	NO	3	YES	5	4	3	3	1	2	1	1	1	1	LRTI
120	673706	50	M	NO	NO	NO	NO	NO	NO	NO	NO	NO		T2DM	98	110	60/40	80/50	90/50			24	24	20				B/L DIFFUSE CREPTS+	S1 S2 NORMAL	UNCONSCIOUS, B/L PUPIL - SLUGGISHLY REACTIVE, B/L PLANTAR- FLEXOR	NO	NO	11.1	SINUS RHYTHM	RIGHT LL NHO+			CRP-POSITIVE, HIV/HBSAG-NEGATIVE	8	8	8			26.1	29.5	32			261	121	84			2.2	4.2	5.8			0.2	0.2	0.8			3D	3D	2C	3	NO	11	15	17			3	3	3			LRTI
121	729241	95	M	YES	NO	YES	NO	NO	NO	NO	NO	NO		T2DM.HTN	99	130	80/50					34						B/L ISA,IAA CREPITATIONS +	S1 S2 NORMAL	CONSCIOUS, RESTLESS	NO	NO	7	NORMAL	B/L LL NHO+	NORMAL	NO GROWTH		10					30					150					1.2					1.3					1D	1D	NO	1	NO	9					3					LRTI.ARDS
122	656067	50	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM, HYPOTHYROIDISM	99	110	140/80					40						B/L BASAL CRACKLES+	S1 S2 NORMAL	RESTLESS, IRRITABLE	NO	NO	14.2	SINUS TACHYCARDIA	B/L BASAL NHO+	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	14					12.8					250					0.8					1.8					1D	1D	NO	1	NO	3					2					LRTI
123	655765	50	F	YES	NO	NO	NO	NO	NO	NO	NO	NO	LEFT LOWER LIMB SWELLING	DM, IHD	100	120	90/60	84/60	94/64	90/60	100/60	30	28	26	20	20		B/L NVBS+	S1 S2 NORMAL	CONSCIOUS, RESTLESS	NO	NO	12.6	LVH	NORMAL	NORMAL		CRP- POSITIVE	14	13	13	13	13	26	32	28	30	26	100	96	84	64	48	2.1	1.8	1.6	1.8	1.6	2.8	2.2	1.8	1.6	1.2	NO	7D	NO	7	NO	9	10	9	9	10	3	3	3	2	2	CELLULITIS
124	729627	48	M	YES	NO	NO	NO	NO	NO	NO	NO	NO	LOWER LIMB SWELLING	DM	99	140	80/50					36						B/L NVBS+	S1 S2 NORMAL	DROWSY	NO	NO	12.6	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSITIVE	12					32					30					1.6					2.1					1D	1D	NO	1	NO	14					3					LRTI.ARDS
125	656572	36	M	YES	NO	NO	NO	YES	NO	NO	NO	NO			101	120	90/60	80/54	80/60	90/64		30	28	26	28			B/L NVBS+	S1 S2 NORMAL	DROWSY, NECK STIFFNESS+	NO	NO	13.2	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSITIVE, CSF ANALYSIS: 6CELLS, NEUTROPHILS	12	10	10	10		26	22	28	24		59	50	42	26		1.2	1.1	1	1.6		4.8	4.6	3.2	2.8		4D	4D	NO	4	NO	10	11	12	14		3	3	3	3		MENINGITIS
126	656588	55	F	YES	NO	NO	NO	NO	NO	NO	NO	NO	LEFT LOWER LIMB SWELLING	DM, IHD	102	120	90/60	84/60				30	28					B/L NVBS+	S1 S2 NORMAL	CONSCIOUS, RESTLESS	NO	NO	12.6	LVH	NORMAL	NORMAL		CRP- POSITIVE	14	13				26	32				100	96				2.1	1.8				2.8	2.2				NO	2D	NO	2	NO	9	10				3	3				CELLULITIS
127	720938	34	F	YES	NO	NO	NO	YES	NO	NO	NO	NO	LOOSE STOOLS	T2DM, HTN	99	120	130/70	140/80	130/70	120/80	130/70	30	26	24	26	30		B/L NVBS +	S1 S2 NORMAL	CONSCIOUS, ORIENTED, NECK STIFFNESS+	NO	NO	11.1	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSTIVE	12	12	14	14	15	10.45	9.84	9.32	10	11.5	8	20	46	50	64	1.1	1.5	1.4	1.6	1.5	8	8	7.2	7	7.2	2D	NO	NO	3	YES	11	12	10	9	7	2	2	2	2	1	ACUTE GE
128	658400	70	M	YES	NO	NO	NO	YES	NO	NO	NO	YES		T2DM, HTN	101	140	70/50					36						B/L NVBS+	S1 S2 NORMAL	DROWSY, NO MENINGEAL SIGNS	NO	NO	13.2	LVH +	NORMAL	NORMAL		CRP- POSITIVE	10					26					100					2.8					2.1					1D	1D	NO	1	NO	15					3					UROSEPSIS
129	728638	65	M	YES	NO	YES	YES	NO	NO	NO	NO	NO			90	100	140/80					40						B/L BASAL CRACKLES+	S1 S2 NORMAL	RESTLESS, IRRITABLE	NO	NO	14.2	SINUS TACHYCARDIA	B/L BASAL NHO+	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	14					12.8					250					0.8					1.8					1D	1D	NO	1	NO	3					2					LRTI
130	659750	58	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		HTN	102	130	110/70	114/76				36	32					B/L DIFFUSE CRACKLES +	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.8	RBBB	B/L NHO (L>R)	NORMAL	NO GROWTH	CRP- POSITIVE	15	15				21	20				150	126				1.2	1.4				1.2	1.8				2D	NO	NO	2	NO	4	4				1	1				LRTI
131	668182	60	M	YES	NO	NO	NO	NO	NO	NO	NO	NO	LOWER LIMB SWELLING	DM	99	140	80/50					36						B/L NVBS+	S1 S2 NORMAL	DROWSY	NO	NO	12.6	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSITIVE	12					32					30					1.6					2.1					1D	1D	NO	1	NO	14					3					CELLULITIS
132	676460	39	M	YES	NO	YES	YES	NO	NO	NO	NO	NO			101	98	100/70	100/60	110/74	110/60	120/70	36	32	30	28	26		B/L COARSE CRACKLES+	S1 S2 NORMAL	RESTLESS, IRRITABLE	YES	NO	12.9	POOR R WAVE PROGRESSION	B/L PATCHY NHO+	NORMAL		CRP- POSITIVE	12	13	13	12	12	18	21	10.9	14.36	12	179	160	151	138	100	2.2	2	2.9	1.6	1.4	6.7	6.6	5.8	5.8	4.9	7D	2D	NO	8	YES	11	9	9	10	9	3	3	2	2	2	LRTI
133	730428	58	M	YES	NO	NO	NO	YES	NO	NO	NO	NO	BURNING MICTURITION	T2DM	101	130	130/80	120/70	110/70	100/70	100/74	32	30	26	24	24		B/L NVBS +	S1 S2 NORMAL	DROWSY, RESPONDING TO COMMANDS	NO	NO	10.4	SINUS TACHYCARDIA	NORMAL	PUS CELLS 12-14	URINE C/S: E.COLI	CRP-POSITIVE, HIV, HBSAG-NEGATIVE	12	12	13	13	14	19.4	20.43	18	16	14	326	342	340	342	340	0.3	0.3	0.4	0.8	0.7	1.2	1.2	1.2	1.2	1.2	NO	1D	NO	1	YES	3	3	2	2	2	2	2	2	2	2	UROSEPSIS
134	677680	37	F	YES	NO	NO	NO	NO	NO	NO	NO	NO	HEMOPTYSIS	TB	99	90	100/60	102/70	100/64	90/60		34	30	32	28			B/L CRACKLES +	S1 S2 NORMAL	DROWSY, NO SIGNS OF MENINGEAL SIGNS	NO	NO	10.5	RBBB	NORMAL	NORMAL		CRP- POSITIVE	13	12	10	10		12	14	16	14		93	55	40	32		2	1.5	1.8	1.6		1.6	2.2	2.1	1.8		4D	1D	NO	4	NO	7	10	12	13		2	2	2	3		LRTI
135	730521	48	F	NO	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	T2DM. HYPOTHYROIDISM	98	120	80/50					34						B/L BASAL CRACKLES+	S1 S2 NORMAL	RESTLESS, IRRITABLE	NO	NO	12.5	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSITIVE	14					27.3					260					1.5					0.9					1D	1D	1C	1	NO	9					3					ACUTE GE
136	667379	56	M	YES	NO	YES	YES	NO	NO	NO	NO	NO			102	130	100/60	90/60	80/50	80/50		46	42	40	30			B/L CRACKLES+	S1 S2 NORMAL	ALTERED SENSORIUM, RESTLESS, IRRITABLE	NO	NO	14.8	SINUS TACHYCARDIA	B/L DIFFUSE NHO+	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	13	12	10	10		10	12.17	19.1	14.7		102	140	155	150		0.8	1	0.9	1		0.7	0.8	0.9	1		4D	3D	NO	4	NO	5	7	8	9		2	3	3	3		LRTI
137	678419	32	M	NO	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS		98	100	100/60	80/50	96/60	90/60	104/60	30	28	26	24	24		B/L NVBS+	S1 S2 NORMAL	RESTLESS, IRRITABLE	NO	NO	15.7	LVH	NORMAL	NORMAL		CRP- POSITIVE	15	14	14	14	15	10.8	12	16	14	10	210	200	168	172	160	0.9	0.8	0.9	1	1.8	1.6	1.2	1.4	1.6	1.8	NO	3D	NO	3	YES	2	5	6	4	2	1	3	3	3	1	ACUTE GE
138	678400	30	M	NO	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS		98	120	80/50	90/50	100/64	110/64	110/70	30	32	30	28	26		B/L NVBS+	S1 S2 NORMAL	CONSCIOUS, RESTLESS, IRRITABLE	NO	NO	15.5	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSITIVE	14	14	15	15	15	12	14	12	11	10	205	200	160	150	148	0.9	0.9	0.8	1	0.8	2.8	2.6	3.2	2.8	1.8	NO	2D	NO	2	YES	7	5	3	3	2	3	3	1	1	1	ACUTE GE
139	728163	24	F	NO	NO	NO	YES	NO	NO	NO	NO	NO	SEIZURES	EPILEPSY	98	90	116/80	80/60				24	28					B/L COARSE CREPITATIONS+	S1 S2 NORMAL	DROWSY, MOVING ALL 4 LIMBS, B/L PLANTAR-MUTE	NO	NO	8	POOR R WAVE PROGRESSION	RIGHT MID AND LOWER ZONE NHO+	NORMAL		CRP-POSITIVE	8	8				20	20				628	600				0.5	0.5				1	1				2D	1D	NO	2	NO	5	8				2	3				LRTI
140	678401	30	F	NO	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS		98	110	80/40	80/60	90/60	100/60	100/64	20	22	20	18	16		B/L NVBS+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.3	SINUS RHYTHM	NORMAL	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	14	15	15	15	15	10.17	10.6	12.4	12.8	9.6	352	350	256	258	322	0.6	0.8	0.8	0.6	0.7	0.9	0.8	0.8	0.8	0.8	NO	4D	NO	4	YES	4	4	3	2	1	2	1	1	1	0	ACUTE GE
141	728592	65	F	YES	NO	NO	NO	NO	NO	NO	NO	NO	RIGHT LOWER LIMB SWELLING	T2DM	101	100	130/70	134/74	140/80	140/86	140/84	24	26	22	20	20		B/L CLEAR	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.5	SINUS RHYTHM	NORMAL	NORMAL		CRP-POSITIVE, HIV, HBSAG-NEGATIVE	15	15	15	15	15	8.1	12.8	9.6	10.1	11.5	342	389	368	437	400	16	15.4	14.7	12	10.6	0.9	1	1.2	1.2	0.9	NO	NO	6C	3	YES	4	4	4	4	4	0	0	0	0	0	CELLULITIS
142	678572	58	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		HTN	101	130	110/70	114/76	120/70	130/80	126/70	36	32	34	28	26		B/L DIFFUSE CRACKLES+	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	12.8	RBBB	B/L NHO+ (L>R)	NORMAL	NO GROWTH	CRP-POSITIVE, HIV/HBSAG-NEGATIVE	15	15	15	15	15	21	20	16	12	10	150	126	134	148	160	1.2	1.4	1.6	1.2	1	1.2	1.8	1	0.8	0.6	NO	NO	NO	2	YES	4	4	3	2	0	1	1	1	1	1	LRTI
143	678515	44	M	YES	NO	YES	YES	NO	NO	NO	NO	NO		T2DM,HTN	100	110	140/80	130/80	120/70	120/80	124/80	40	36	36	38	32		B/L BASAL CRACKLES+	S1 S2 NORMAL	RESTLESS, IRRITABLE	NO	NO	14.2	SINUS TACHYCARDIA	B/L BASAL NHO+	NORMAL		CRP-POSITIVE, HIV/HBSAG-NEGATIVE	14	13	13	13	13	12.8	14	16	12.6	10.8	250	260	254	286	320	0.8	0.8	0.6	0.8	0.6	1.8	1.8	2.1	1.6	1.2	NO	NO	NO	1	YES	3	3	4	2	2	2	2	2	2	2	LRTI
144	728593	65	M	NO	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	HTN	98	120	80/50					34						B/L BASAL CREPTS+	S1 S2 NORMAL	RESTLESS, IRRITABLE	NO	NO	12.5	SINUS RHYTHM	NORMAL	NORMAL		CRP- POSITIVE	14					27.3					260					1.5					0.9					1D	1D	1C	1	NO	9					3					ACUTE GE
145	678531	55	M	YES	NO	YES	YES	YES	NO	NO	NO	NO		T2DM.HYPOTHYROIDISM	102	108	90/70	100/60	94/60			32	30	28				B/L BRONCHIAL BREATH SOUNDS+	S1 S2 NORMAL	ALTERED SENSORIUM	NO	NO	12.1	SINUS TACHYCARDIA	B/L NHO+	NORMAL	NO GROWTH	CRP- POSITIVE, HIV/HBSAG - NEGATIVE	13	12	10			12	14	16			198	123	100			0.6	0.8	0.8			0.8	0.8	1.2			3D	3D	NO	3	NO	4	8	10			3	3	3			LRTI
146	678570	52	M	NO	NO	NO	NO	NO	YES	NO	NO	YES	ANURIA	T2DM	98	80	150/100	140/100	140/96	146/80	140/100	30	32	34	28	26		B/L NVBS+	S1 S2 NORMAL	CONSCIOU, ORIENTED	NO	NO	14.1	IHD	CARDIOMEGALY+	NORMAL		CRP- POSITIVE	15	13	12	12	12	25	26	31	24	18	26	16	20	34	56	3.3	5.2	6.4	4.8	3.8	3.5	4.2	3.8	3.3	2.8	NO	NO	4C	3	YES	8	12	13	11	9	1	2	2	2	2	UROSEPSIS
147	679296	53	F	YES	NO	NO	NO	NO	YES	NO	NO	YES	LOOSE STOOLS	DM, HYPOTHYROIDISM	99	112	84/50	90/60	100/60	104/64	110/70	38	36	30	24	20		B/L NVBS+	S1 S2 NORMAL	RESTLESS	NO	NO	10.4	LOW VOLTAGE COMPLEXES+	CARDIOMEGALY+	NORMAL		CRP- POSITIVE	14	15	15	15	15	28	31.1	28	26	18	301	272	150	134	160	1.4	1.8	2.1	2.2	1.8	2.1	2.4	2.2	1.8	1.6	NO	2D	NO	2	YES	7	5	5	4	3	3	2	1	1	1	ACUTE GE
148	679474	48	M	YES	NO	NO	NO	YES	NO	NO	NO	NO		COPD.HTN	99	110	80/60	90/50	80/60			30	32	34				B/L NVBS+	S1 S2 NORMAL	DROWSY, B/L PUPIL- SLUGGISHLY REACTIVE, NECK STIFFNESS+	YES	NO	10.3	SINUS TACHYCARDIA	NORMAL	NORMAL		CRP- POSITIVE, DENGUE -POSITIVE	12	10	10			22	26	28			17	15	20			6	7	8.6			8	7.2	6.4			1D	3D	1C	3	NO	16	18	19			3	3	3			MENINGITIS
149	658938	51	M	YES	NO	NO	NO	NO	NO	NO	NO	NO	LEFT LOWER LIMB SWELLING	T2DM	99	84	90/60	100/60	100/60	110/60	100/70	20	20	22	24	20		B/L CLEAR	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	11.7	SINUS RHYTHM	NORMAL	NORMAL		CRP-POSITIVE	15	15	15	15	15	10.8	10.15	10	9	8	215	222	200	256	200	1.8	1.6	1.4	1.3	1.2	1.4	1.4	1.2	1.2	1.2	NO	2D	NO	2	YES	4	3	2	2	2	1	1	0	0	0	CELLULITIS
150	660170	60	F	NO	NO	NO	NO	NO	YES	NO	NO	NO	LOOSE STOOLS	B,ASTHMA	98	86	110/70	110/70	100/60	114/74	110/70	26	24	26	22	24		B/L CLEAR	S1 S2 NORMAL	CONSCIOUS, ORIENTED	NO	NO	9.8	NORMAL	NORMAL	NORMAL		CRP-POSITIVE	15	15	15	15	15	7.6	8	10	12	12	183	150	132	122	100	1.5	1.6	1.8	1.4	1.2	1.4	1.2	1.2	1	1	NO	2D	NO	2	YES	2	3	3	2	3	0	0	0	0	0	ACUTE GE