Dissertation on

THE INCIDENCE, ETIOLOGY, RISK FACTORS AND PROGNOSIS AMONG PATIENTS WITH SIRS/SEPSIS SPECTRA ADMITTED TO THE MEDICAL WARDS IN A TERTIARY CARE HOSPITAL

Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations

for the award of the degree of

M.D. BRANCH - I

GENERAL MEDICINE



MADRAS MEDICAL COLLEGE AND GOVERNMENT GENERAL HOSPITAL, CHENNAI – 3

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI, INDIA

APRIL 2011

CERTIFICATE

This is to certify that the dissertation entitled "THE INCIDENCE, ETIOLOGY, RISK FACTORS AND PROGNOSIS AMONG PATIENTS WITH SIRS/SEPSIS SPECTRA ADMITTED TO THE MEDICAL WARDS IN A TERTIARY CARE HOSPITAL" is a bonafide work done by Dr. MOHAMMED SAMEER, M.J., post graduate student, Institute of Internal Medicine, Madras Medical College, Chennai-3 in partial fulfillment of the University Rules and Regulations for the award of MD Branch – I General Medicine, under our guidance and supervision, during the Academic period from April 2008 to April 2011.

Dr. C. RAJENDIRAN, M. D Director & Professor Institute of Internal Medicine Madras Medical College Chennai – 600 003.

Prof. S TITO, M.D.,

Associate Professor Institute of Internal Medicine Madras Medical College Chennai – 600 003.

Dr. J. MOHANASUNDARAM, M.D., Ph.D., DNB D E A N

Madras Medical College

Chennai - 600 003

DECLARATION

I solemnly declare that the dissertation entitled "THE INCIDENCE, ETIOLOGY,RISK FACTORS AND PROGNOSIS AMONG PATIENTS WITH SIRS/SEPSIS SPECTRA ADMITTED TO THE MEDICAL WARDS IN A TERTIARY CARE HOSPITAL" is done by me at Madras Medical College, Chennai-3 during the academic period from April 2008 to April 2011 under the guidance and supervision of **Prof.S.Tito**, M.D., Associate Professor of Medicine, Madras Medical College and Government General Hospital, Chennai.

This dissertation is submitted to The Tamilnadu Dr M.G.R Medical University towards the partial fulfillment of requirements for the award of **M.D. Degree (Branch-I) in General Medicine.**

Place : Chennai

(Dr. MOHAMMED SAMEER.M.J)

Date :

ACKNOWLEDGEMENT

I thank **Prof. J.MOHANASUNDARAM, M.D., Ph.D., DNB** Dean, Madras Medical College, for having permitted me to conduct the study and use the hospital resources in the study.

I express my heartfelt gratitude to **Prof. C.RAJENDIRAN, M.D.,** Director & Professor, Institute of Internal Medicine, for his inspiration, advice, and guidance in making this work complete.

I express my deep gratitude to my chief **Prof. S. TITO, M.D.,** Associate Professor, Institute of Internal Medicine, for his comments, corrections and guidance to complete the study.

I am extremely thankful to Assistant Professors of Medicine Dr. G.SUBBARAGAVALU, M.D., and Dr. C.SRIDHAR, M.D., for their co-operation and guidance.

Last but not the least, I would wish to thank all the patients without whose kind cooperation, this study would not have been possible.

CONTENTS

Sl.No	TITLE	PAGE NO
1.	INTRODUCTION	01
2.	AIMS AND OBJECTIVES	03
3.	REVIEW OF LITERATURE	04
4.	MATERIALS AND METHODS	29
5.	OBSERVATIONS AND RESULTS	34
6.	DISCUSSION	59
7.	CONCLUSION	67
8.	LIMITATIONS	69
	ANNEXURE	
	> ABBREVIATIONS	
	> PROFORMA	
	> MASTER CHART	
	> BIBLIOGRAPHY	
	> ETHICAL COMMITTEE APPROVAL ORDER	

INTRODUCTION

Sepsis is a frequent cause of admission to intensive care units (ICUs)/ Medical wards and one of the leading causes of death among hospitalized patients. With an estimated annual mortality of between 30 and 50 deaths per 100 000 population, this condition ranks in the top 10 causes of death, affects all ages, and occurs in the community, in long term care facilities, and among patients admitted to hospital under the care of any, and every, medical specialty. Over the last 100 years, huge advances have been made in the field of sepsis in terms of pathophysiology, epidemiology, diagnosis, monitoring, and therapeutics. Despite these changes, mortality rates remain unacceptably high and continued progress, particularly in early diagnosis and therapy, is urgently needed.

Sepsis encompasses a spectrum of illness that ranges from minor signs and symptoms through to organ dysfunction and shock. The pathophysiology of sepsis arises largely from the response of the host's innate immune system, under the influence of genetic factors. The signs and symptoms of sepsis are influenced by the virulence of the pathogen, the portal of entry, the susceptibility and response of the host, and the temporal evolution of the condition.

Categorization of patients within the subgroups of severity has clinical and prognostic significance and has improved the conduct and interpretation of epidemiologic studies and clinical trials in the field of sepsis. Several risk factors for death of patients with sepsis have been shown and those variables characterizing underlying disease or associated with acute illness and characteristics of infection were associated with mortality.

Sepsis is a clinical diagnosis; microbiological investigations are commonly negative. Powerful molecular biological techniques are likely to make a substantial contribution to the diagnosis of sepsis in the next five to 10 years.

The present study aims to determine the incidence, risk factors, co morbid conditions and infection characteristics for hospital mortality after categorizing them in three stages of severity (SIRS/Sepsis, severe sepsis, and septic shock) and APACHE-II scoring for comparing the outcomes in these patients in a therapy-independent fashion.

AIMS AND OBJECTIVES

- To describe the epidemiology of sepsis syndrome in the tertiary care hospital-medical ward setting.
- The incidence of SIRS/Sepsis, severe sepsis and septic shock in 1000 consecutive patients admitted to the hospital and treated in the medical wards.
- To evaluate and define the patient's characteristics and etiology of SIRS/Sepsis spectra.
- To examine risk factors associated with outcome in SIRS/Sepsis spectra.
- To determine the co morbidities influencing the prognosis of septic patients.

REVIEW OF LITERATURE

The last 100 years have seen great advances in our understanding of sepsis, a term derived from the ancient Greek for rotten flesh and putrefaction. In 2735 BC, Chinese emperor Sheng Nung recorded the use of an herbal remedy to treat fever. Over the next 2 millennia, pandemics of cholera, plague (black death), smallpox, measles, tuberculosis, and gonorrhea spread worldwide, wiping out huge segments of the population.

In 1546, Hieronymus Fracastorius suggested germ theory for infections. In the 1680s, some of the first descriptions of bacteria, Leeuwenhoek's "animalcules" were made, but it was another 200 years before the link between bacteria and infection finally began to be realized by some of the founders of modern microbiology and medicine, including Koch, Pasteur, Semmelweis, and Lister. In 1879, Louis Pasteur identified *Streptococcus* bacteria as the cause of puerperal sepsis. In 1892, Richard Pfeiffer identified the toxin that causes shock in patients. John Pringle, a British army surgeon, proposed the concept of antisepsis for the first time. In the 19th century, antiseptic practices lead to a reduction in mortality from puerperal fever from 13.6% to 1.5% in a Vienna hospital.

In 1928, Alexander Fleming recognized that his bacterial cultures were killed by a blue mold, *Penicillium notatum*. Thus, with the discovery of penicillin, a new era began, with antibiotics used to treat bacterial infections. In 1944 in the United States, Waksman discovered that streptomycin was effective in the treatment of tuberculosis.

Sir William Osler was the first to recognize that "except on few occasions, the patient appears to die from the body's response to infection rather than from the infection." In 1914, Schottmueller¹ wrote, "Septicemia is a state of microbial invasion from a portal of entry into the blood stream which causes sign of illness, changing our modern understanding of the term "sepsis"².

EPIDEMIOLOGY OF SEPSIS/INCIDENCE

Current estimates suggest that that some 750,000 cases of severe sepsis occur annually in the United States, with a mortality rate of around 29%³. The recent Sepsis Occurrence in Acutely III Patients (SOAP) study across Europe reported that more than 35% of intensive care unit (ICU) patients had sepsis at some point during their ICU stay, with a mortality rate of 27%⁴. The rates of sepsis appear to be increasing in hospitals worldwide.



A study conducted in India⁵ showed SIRS with organ dysfunction in 25%, of which 52.77% were due to sepsis. The incidence of severe sepsis was 16.45%. 57.71% were male. ITU mortality of all admissions was 12.08% and that of severe sepsis was 59.26%.

The estimated incidence of patients admitted to ICU with severe sepsis in The Netherlands was 8,643 per year. In 1986 Verbrugh and coworkers, identified 5.4 cases of bacteraemia with clinical symptoms for every 1000 hospital admissions in two general hospitals⁶ and in 1993 Kieft and colleagues⁷, found an incidence of sepsis syndrome in a Dutch university hospital of 1.36% of all hospital admissions. In 1994 the National Institute for Public Health and the Environment reported 4.8 cases of sepsis registered as first or second diagnoses for every 1000 hospital admissions on the basis of National Medical Registration in The

Netherlands⁸. The cross-sectional prevalence of severe sepsis patients in Dutch Intensive Care Units was 29.5%. The most severe sepsis patients were admitted to the ICU due to acute infection⁹.

Sepsis and multiple organ failure are common complications in intensive care unit (ICU) patients and are associated with considerable morbidity and mortality. Sepsis affects some 40% of ICU admissions, severe sepsis occurs in about 30%, and septic shock in 15%. Recent consensus has improved the definition of sepsis and proposed a new classification system based on number of organ dysfunction¹⁰.

Clinically suspected sepsis and confirmed severe sepsis occurred in 9.0 and 6.3 of 100 French ICU admissions, respectively. The 28-day mortality was 56% in patients with severe sepsis, and 60% in those with culture-negative severe sepsis¹¹. The calculated incidence of severe sepsis in adults treated in Australian and New Zealand ICUs is 0.77 (0.76-0.79) per 1000 of population. 26.5% of patients with severe sepsis died in ICU, 32.4% died within 28 days of the diagnosis of severe sepsis and 37.5% died in hospital¹². Prevalence of infections in intensive care units in Mexico were 58.2%¹³.

AGE RELATION

Incidence increased >100-fold with age (0.2/1,000 in children to 26.2/1,000 in those >85 yrs old). Mortality was 28.6% and also increased with age, from 10% in children to 38.4% in those >85 yrs old. Women had lower age-specific incidence and mortality, but the difference in mortality was explained by differences in underlying disease and the site of infection. The incidence was projected to increase by 1.5% per annum¹⁴.

CATEGORIZATION

Since 1992, epidemiological and clinical studies have classified severe infections into three categories: sepsis, severe sepsis and septic shock. Microbiological documentation is not always documented with sepsis. The in-hospital mortality rate increased with severity, from 20% for sepsis to 40% for severe sepsis and 60% for septic shock, but also depended on the origin of infection¹⁵. Clinical characteristics that relate to the severity of sepsis include an abnormal host response to infection, the site and type of infection, the timing and type of antimicrobial therapy, the offending organism, and the development of shock, underlying disease, the patients' chronic health condition, and the number of failed organs¹⁶.

The prevalence of SIRS is very high, affecting one-third of all inhospital patients, and >50% of all ICU patients. The prevalence of infection and bacteraemia increases with the number of SIRS criteria met, and with increasing severity of the septic syndromes. About one-third of patients with SIRS have or evolve to sepsis. Sepsis may occur in approximately 25% of ICU patients, and bacteraemic sepsis in 10%. In such patients, sepsis evolves to severe sepsis in >25% of cases, non-ICU patients. Severe sepsis and septic shock occur in 2%-3% of ward patients and 10%-15% or more ICU patients. 25% of patients with severe sepsis have shock. There is a graded severity from SIRS to sepsis, severe sepsis and septic shock, with an associated 28-d mortality of approximately 10%, 20%, 20%-40%, and 40%-60%, respectively. Mortality rates are similar within each stage, whether infection is documented or not, and microbiological characteristics of infection do not substantially influence outcome, although the source of infection does. About three of four deaths occur during the first months after sepsis¹⁷. About 28% of infections were associated with sepsis, 24% with severe sepsis and 30% with septic shock. Crude hospital mortality rates ranged from 16.9% in non-infected patients to 53.6% in patients with hospital-acquired infections¹⁸.

In the BASES study¹⁹ the overall 28-day mortality rate was 21.8%. The incidence density rates for sepsis, severe sepsis and septic shock were 61.4, 35.6 and 30.0 per 1000 patient-days, respectively. The mortality rate of patients with SIRS, sepsis, severe sepsis and septic shock increased progressively from 24.3% to 34.7%, 47.3% and 52.2%, respectively. For patients with SIRS without infection the mortality rate was 11.3%. The main source of infection was lung/respiratory tract¹⁹.SIRS occurs more frequently and its occurrence ranges from 40% to 70% of all patients.10% of patients in the ICU suffer from sepsis, 6% from severe sepsis and 2-3% from septic shock. The overall prognosis is still poor, despite the recent advances in treatment. The mortality rate of SIRS ranges from 6% to 7% and in septic shock amounts to over 50%²⁰. In England 27.1% of adult intensive care unit admissions met severe sepsis criteria, most were nonsurgical (67%), and the most common organ system dysfunctions were seen in the cardiovascular (88%) and respiratory (81%) systems. 47% died during their hospital stay. Hospital mortality rate ranged from 17% in the 16-19 age group to 64% in those >85 yrs²¹. Sepsis was more common among men than among women. Gram-positive bacteria becoming the predominant pathogens after 1987.

Organ failure contributed cumulatively to mortality, with temporal improvements in survival among patients with fewer than three failing organs²².

SITE OF INFECTION

3-10% of the patients admitted to either medical or surgical ICUs suffer from bacteremia and/or sepsis of different severities. The primary infectious site causing sepsis has changed with time, from the abdomen as primary source before 1990 to the lungs in more recent years²³. Recent studies indicate that pneumonia is the most common infection associated with sepsis today (~ 40%), followed by intra -abdominal infection (20%), catheters and primary bacteremias (15%), and the urinary tract $(10\%)^{24}$. The Incidence of sepsis syndrome has increased, most likely due to the augmented use of Immunosuppressive therapies and invasive diagnostic as well as therapeutic procedures (e.g. catheters etc.). Site of infection was pulmonary in 50.3% and abdominal in 19.3%²⁵. In an International multicentre study the Respiratory, digestive, urinary tracts, and primary bloodstream infections represented about 80% of all sites. Hospitalacquired and ICU-acquired infections are documented more frequently microbiologically than community-acquired infections. Infections are documented microbiologically in community-acquired infections in 55%²⁶. In particular, abdominal sepsis exhibits the highest mortality rate with

 $72\%^{27}$. In Mexico Community-acquired infection was 23.9%. The most frequently reported infections were pneumonia (39.7%), urinary tract infections (20.5%), wound infection (13.3%), and bacteremia (7.3%)²⁸.

ETIOLOGY/MICROBIOLOGY

The microbiology of severe sepsis and septic shock has also altered over time. Although in the past gram-negative organisms were most commonly implicated, increasingly gram-positive organisms are isolated, such that roughly similar numbers of gram-positive and gram-negative organisms are now associated with sepsis. Sepsis can also be caused by a fungal or parasitic infection, and in about one-third of patients, no infectious agent is identified²⁹, usually either because sampling is impossible (e.g., some patients have community-acquired lung infection without sputum production) or because culture remains negative in patients who are already receiving antimicrobial drugs.



In the Indian study Culture positivity was found in 61.6%. The lung was the predominant source of sepsis (57.45%). Gram-negative organisms were responsible for 72.45% of cases and Gram-positive for 13.13%. The rest were parasitic, viral and fungal infection. Severe sepsis was commoner in Indian ITUs³. ITU mortality was higher compared with western literature. Gram-positive infections were less common although incidences of parasitic and viral infection were higher than in the West.

Only three of four patients presenting with clinically suspected severe sepsis have documented infection. However, patients with clinically suspected sepsis but without microbiological documentation and patients with documented infection share common risk factors and are at similarly high risk of death. In addition to the severity of illness score, acute organ failures and the characteristics of underlying diseases account for stratification of patients and outcome³⁰.

SEASONAL INCIDENCE

The incidence and mortality of sepsis and severe sepsis are seasonal and consistently highest during the winter, predominantly related to respiratory sepsis. Seasonal changes in sepsis incidence vary according to geographic region²³. The mechanisms underlying these differences require further investigation. Understanding seasonal or regional variations may improve knowledge of sepsis epidemiology and pathophysiology and could affect healthcare planning and resource allocation.

RISK FACTORS

Risk factors for severe sepsis and septic shock³¹

- 1. Extremes of age (<10 y and >70 y)
- Primary diseases (Liver cirrhosis, Alcoholism, Diabetes mellitus, Cardiopulmonary diseases, Solid malignancy, Hematologic malignancy, Immunosuppression, Neutropenia, Immunosuppressive therapy, Corticosteroid therapy, Intravenous drug abuse, Complement deficiencies, Asplenia)

- 3. Major surgery, trauma, burns.
- Invasive procedures (Catheters, Intravascular devices, Prosthetic devices, Hemodialysis and peritoneal dialysis catheters, Endotracheal tubes)
- 5. Prior antibiotic treatment
- 6. Other factors Childbirth, abortion, and malnutrition

Several risk factors for severe sepsis have been identified in previous studies, including age, sex, comorbidities, and causative pathogen^{32,33}. Major determinants of both early (<3 days) and secondary deaths in the whole cohort were the Acute Physiology Score and the number of acute organ system failures. Other risk factors for early death included a low arterial blood pH (<7.33) (P < .001) and shock (P = .03), the admission category (P < .001), a rapidly or ultimately fatal underlying disease (P < .001), a preexisting liver (P = .01) or cardiovascular (P = .002) insufficiency, hypothermia (P = .02), thrombocytopenia (P = .01), and multiple sources of infection (P = .02). In patients with documented sepsis, bacteremia was associated with early mortality (P = .03)³⁴.

Other independent mortality risk factors in a French study were mechanical ventilation (OR = 4.97), chronic alcoholism (OR = 3.38), age >65 years (OR = 2.65), prothrombin ratio <40% (OR = 2.37), and PaO_2/FiO_2 ratio <150 (OR = 1.91). These six mortality risk factors allow screening immediately septic shock patients with a high mortality risk³⁵. The major determinants of outcome, both short-term and long-term, of patients with sepsis are the severity of underlying diseases and co morbidities, the presence of shock and organ failures at onset of sepsis or evolving thereafter³⁶. Multivariate regression analysis have also showed the following risk factors for acquired infections: neurologic failure as a primary cause of admission, number of therapeutic and/or diagnostic interventions during the preceding week, peripherally administered infusion of hyperosmolar solutions, sedative usage in the preceding week. The administration of antimicrobial treatment if there was an infection decreased the risk of death³⁷.

DEFINITIONS

An American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference held in North-brook was in August1991 with the goal of agreeing on a set of definitions that should be applied to patients with sepsis and its sequelae. Definitions for severe sepsis, septic shock, hypotension, and multiple organ dysfunction syndrome were also offered. The current definitions are from The Sepsis definitions conference held in 2001, sponsored by the SCCM, the ACCP, the American Thoracic Society, the European Society of Intensive Care Medicine, and the Surgical Infection Society³⁸.

The term sepsis in popular usage implies a clinical response arising from infection. It is apparent that a similar, or even identical, response can arise in the absence of infection. Systemic inflammatory response syndrome (SIRS) describes this inflammatory process, independent of its cause.



Figure:1 The Interrelationship between systemic inflammatory response syndrome (SIRS), Sepsis and infection BACTEREMIA: The presence of visible bacteria in the blood.

INFECTION: Microbial phenomenon characterized by an inflammatory response to the presence of micro-organisms or the invasion of normally sterile host tissue by those organisms.

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS): The

systemic inflammatory response to a variety of severe clinical insults. The

response is manifested by two or more of the following conditions:

- Temperature >38°C or <36°C
- Heart rate >90 beats per minute
- Respiratory rate >20 beats per minute or PaCO2 <32 mmHg

- WBC count >12,000/cu.mm, <4000/cu.mm or >10% immature band forms.
- Hyperglycemia (Plasma glucose > 120 mg/dl or 7.7 mmol/l) without DM

SEPSIS: The systemic response to infection, manifested by two or more of the above criteria's as a result of infection, documented microbiologically or strongly clinically suspected.

MULTIPLE ORGAN DYSFUNCTION SYNDROME (MODS): Presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention.

SEVERE SEPSIS: Sepsis associated with organ dysfunction, hypoperfusion or hypotension. Hypoperfusion and perfusion abnormalities may include, but are not limited to lactic acidosis, oliguria, or an acute alteration in mental status.

SEPSISI INDUCED HYPOTENSION: SBP <90 mmHg or a reduction of >40mmHg from baseline in the absence of other causes of hypotension.

SEPTIC SHOCK: Sepsis induced hypotension despite adequate fluid resuscitation along with the presence of perfusion abnormalities defined above. Patients receiving vasopressors or inotropes may not be hypotensive at the time the perfusion abnormalities are measured.

MODS may be described as being either primary or secondary. MODS develop by two relatively distinct, but not mutually exclusive, pathways. Primary MODS is the direct result of a well-defined insult in which organ dysfunction occurs early and can be directly attributable to the insult itself. An example of primary MODS is organ dysfunction as the immediate result of trauma (e.g., pulmonary contusion, renal failure due to rhabdomyolysis, or the coagulopathy due to multiple transfusions). In primary MODS, the participation of an abnormal and excessive host inflammatory response in both the onset and progression of the syndrome is not as evident as it is in secondary MODS.

Secondary MODS develops, not in direct response to the insult itself, but as the consequence of a host response, and is identified within the context of SIRS. SIRS is also a continuous process, and describes an abnormal host response that is characterized by a generalized activation of the inflammatory reaction in organs remote from the initial insult. When the process is due to infection, the terms sepsis and SIRS are synonymous. Given that SIRS/sepsis is a continuous process, MODS may be understood to represent the more severe end of the spectrum of severity of illness that characterizes SIRS/sepsis. Thus, secondary MODS usually evolve after a latent period following the inciting injury or event, and is most commonly seen to complicate severe infection.



FIGURE 2. The different causes and results of primary and secondary multiple organ dysfunction syndrome (MODS).

TABLE 2. THE PIRO CONCEPT³⁹

	Clinical	Other Tests
P (predisposition)	Age, alcohol abuse, steroid or immunosuppressive therapy	Immunologic monitoring, genetic factors
I (infection)	Site-specific (e.g., pneumonia, peritonitis)	X-rays, CT scan, bacteriology
R (response)	Malaise, temperature, heart rate, respiratory rate	WBC, CRP, PCT, modified APTT
O (organ dysfn)	Arterial pressure, urine output, Glasgow coma score	Pa ₀₂ /FI ₀₂ , creatinine, bilirubin, platelets
Definition a	of abbreviations: APTT = activated pa	artial

Definition of abbreviations: APTT = activated partial thromboplastin time; <math>CRP = C-reactive protein; CT = computed tomography; PCT = procalcitonin; WBC = white blood cell count.

PATHOPHYSIOLOGY OF SEPSIS⁴⁰⁻⁵²

The pathogenesis of sepsis involves a complex process of cellular activation resulting in the release of proinflammatory mediators, such as cytokines, activation of neutrophils, monocytes, and microvascular endothelial cells, involvement of neuroendocrine reflexes, and activation of the complement, coagulation, and fibrinolytic systems.

Initiation of sepsis occurs as microbial components are recognized by soluble or cell-bound pattern recognition molecules or receptors, such as CD14 and Toll-like receptors (TLRs), activation of which induces the transcription of inflammatory and immune response genes, often via nuclear factor-*k*B–mediated mechanisms, resulting in the release of a number of endogenous mediators.

Cytokines, a family of cell signaling peptides with pro-and anti inflammatory properties, are some of the best known and most studied endogenous mediators associated with the development of organ system dysfunction in sepsis. Two of the first cytokines to be implicated in sepsis were tumor necrosis factor α (TNF- α) and interleukin 1 (IL-1).

TNF- α , first identified in 1975, is involved in leukocyte adhesion, local inflammation, neutrophil activation, generation of fever, suppression of erythropoiesis, decrease in fatty acid synthesis, and suppression of albumin synthesis, among others. The final critical steps demonstrating that these cytokines were involved in the development of severe sepsis came from studies showing that there was a correlation between the magnitude of circulating TNF- α levels and patient outcome, and then the observation that the injection of IL-1 or TNF- α into animals can reproduce all the hemodynamic and biochemical features of severe sepsis and organ failure. Furthermore, blocking the effects of TNF and IL-1 in models of severe infection prevented complications and improved outcomes. Other cytokines and proinflammatory mediators that are currently attracting considerable interest for their putative contribution to sepsis include highmobility group box1(HMGB1), protein, a late mediator of systemic inflammation, and macrophage migration inhibitory factor, MIF. An important advance in sepsis pathophysiology has been the growing realization of the links between the coagulation system and the immune response to sepsis, which led to the development of the only specific antisepsis treatment currently available, recombinant human activated protein C.

Role of Endotoxin and Other Bacterial Toxins

Endotoxin was first identified more than 100 years ago, but it was not until 1951 that Borden and Hall first suggested that it might have a role in the development of septic shock. Evidence for a pathogenic role of endotoxin came from the report by Taveira da Silva and colleagues of the effects of accidental self-administration of endotoxin by a lab technician. Endotoxin is frequently found in the blood of acutely ill patients with sepsis, even in the absence of demonstrable gram-negative infection, possibly as a result of bacterial translocation from the gut. Even patients with heart failure may have circulating endotoxin. Nevertheless, endotoxin levels are associated with a higher incidence of complications and have been shown to be an early predictor of bacteremia in febrile patients.

Other bacterial toxins, such as peptidoglycans or lipoteichoic acid, can be released by gram-positive microorganisms and induce the production of mediators associated with sepsis. Although early studies in patients attempted to relate the hemodynamic presentation with the type of microorganism (gram-positive vs. gram-negative), the results of these studies were inconsistent, and it became apparent that the hemodynamic response is not related to the type of organism. This does not mean, however, that the specific causative organism does not matter; although the innate immune response generated by the host may be similar for all microorganisms, there also appear to be adaptive pathogen-specific responses.

MANAGEMENT OF SEPSIS⁵³⁻⁶⁴

In 2003, critical care and infectious disease experts representing 11 international organizations developed management guidelines for severe sepsis and septic shock that would be of practical use for the bedside clinician, under the auspices of Surviving Sepsis Campaign, an international effort to increase awareness and improve outcome in severe sepsis. In 2003, critical care and infectious disease experts representing 11 international organizations developed management guidelines for severe sepsis and septic shock that would be of practical use for the bedside clinician, under the auspices of the Surviving Sepsis Campaign, an international effort to increase awareness and improve outcome in severe sepsis.

Key recommendations, listed by category and not by hierarchy, include early goal-directed resuscitation of the septic patient during the first 6 hrs after recognition; appropriate diagnostic studies to ascertain causative organisms before starting antibiotics; early administration of broad-spectrum antibiotic therapy; reassessment of antibiotic therapy with microbiology and clinical data to narrow coverage, when appropriate; a usual 7–10 days of antibiotic therapy guided by clinical response; source control with attention to the method that balances risks and benefits.

Every patient should be evaluated for the presence of a focus on infection amenable to source control measures, specifically the drainage of an abscess or local focus on infection, the debridement of infected necrotic tissue, the removal of a potentially infected device, or the definitive control of a source of ongoing microbial contamination If intravascular access devices are potentially the source of severe sepsis or septic shock, they should be promptly removed after establishing other vascular access.

Equivalence of crystalloid and colloid resuscitation⁵³; aggressive fluid challenge to restore mean circulating filling pressure; vasopressor preference for norepinephrine and dopamine; cautious use of vasopressin pending further studies; avoiding low-dose dopamine administration for renal protection; consideration of dobutamine inotropic therapy in some clinical situations.

Avoidance of supranormal oxygen delivery as a goal of therapy; stress-dose steroid therapy for septic shock; use of recombinant activated protein C in patients with severe sepsis and high risk for death; with resolution of tissue hypo- perfusion and in the absence of coronary artery disease or acute hemorrhage, targeting a hemoglobin of 7–9 g/dL; appropriate use of fresh frozen plasma and platelets.

A low tidal volume and limitation of inspiratory plateau pressure strategy for acute lung injury and acute respiratory distress syndrome; application of a minimal amount of positive end-expiratory pressure in acute lung injury/acute respiratory distress syndrome; a semirecumbent bed position unless contraindicated.

Protocols for weaning and sedation/analgesia, using either intermittent bolus sedation or continuous infusion sedation with daily interruptions/lightening; avoidance of neuromuscular blockers, if at all possible; maintenance of blood glucose <150 mg/dL after initial stabilization; equivalence of continuous veno-veno hemofiltration and intermittent hemodialysis; lack of utility of bicarbonate use for pH >7.15; use of deep vein thrombosis/stress ulcer prophylaxis; and consideration of limitation of support where appropriate.

Recombinant Human Activated Protein C⁵⁴

rhAPC is recommended in patients at high risk of death (Acute Physiology and Chronic Health Evaluation II >25, sepsis-induced multiple organ failure, Septic shock, or sepsis-induced acute respiratory distress syndrome [ARDS] and with no absolute contraindication related to bleeding risk or relative contraindication that outweighs the potential benefit of rhAPC.

TABLE 3. SOME OF THE MANY PROPOSED SEPSIS THERAPIES THAT HAVE UNDERGONE CLINICAL TRIALS

- 1. Antiendotoxin strategies (HA-1A, E5, LPS analogs).
- Anticytokine strategies (Anti-TNF antibodies, TNF receptors, IL-1ra).
- Strategies against other mediators (Nitric oxide—L-NMMA, L-NAME, methylene blue, PAF, acetylhydrolase, Arachidonic acid metabolites, Antioxidants).
- Strategies against coagulation system (Antithrombin III, Tissue factor pathway inhibitor).
- 5. Enhanced elimination (Hemofiltration, Immunestimulating strategies, GCSF, IFN- γ).
- 6. Immunonutrition

Definition of abbreviations:

GCSF = granulocyte colony-stimulating factor; IFN = interferon;IL = interleukin; LPS = lipopolysaccharide; PAF = platelet activating factor; TNF = tumor necrosis factor.

MATERIALS AND METHODS

Methods

A prospective, observational, study was carried out over a 5 month period.

Primary objectives

- The aims were to determine the incidence of sepsis spectra among 1000 consecutively admitted adult patients in medical wards of Madras Medical College and Government General Hospital, Chennai.
- To determine the mortality rates, as well as factors associated with the risk of death.
- To describe the epidemiology of sepsis syndrome in the tertiary care hospital -medical ward setting.
- To evaluate and define the patient's characteristics and etiology of SIRS/Sepsis spectra.
- To determine the co morbidities influencing the prognosis of septic patients.

- To determine the frequency of infecting organ systems involved.
- To determine the types of infection (acquisition and microbiology) involved.

Design and data collection

This was a prospective, observational, study. The study protocol was approved by the Ethics Committee of the coordinating centre. The study conducted after informed consent was obtained from participants. The study was carried out over a 5-month period, from May 16 to October 16, 2010, in medical wards.

All patients were screened for SIRS on admission and followed up till death or discharge. All data were collected on standardized forms in each ward.

Inclusion criteria:

- 1. Age greater than 12 years.
- 2. Patients admitted in medical units in Government General Hospital who fulfill the following criteria for SIRS/Sepsis were selected for the study.

Infection, documented or suspected, and any 2 of the following:

- Fever (core temp > 38.3 °C) or Hypothermia (core temp < 36 °C)</p>
- Heart rate > 90/min or > 2 SD above the normal value for age Tachypnea >20/min or > 2 SD above the normal value for age Leukocytosis (> 12,000/mm3) or Leukopenia (< 4,000/mm3) Hyperglycemia (Plasma glucose > 120 mg/dl or 7.7 mmol/l) without DM Altered mental status

Exclusion criteria

1. Patients Age less than 12 years

:

All data related to physiological and biological variables were checked against standardized ranges. Inconsistent or extreme values were thoroughly checked and corrected before analysis. Clinical and laboratory data to enable the Acute Physiology and Chronic Health Evaluation (APACHE) II Score [20] to be calculated were collected within the first 24 h after admission. Neurological status was determined using the Glasgow Coma Scale (GCS) prior to sedation.

Patients were considered to have an infection if this was microbiologically documented according to the standard definitions or at least clinically suspected requiring evidence such as the presence of white blood cells in a normally sterile body fluid, perforated viscus, chest X-ray consistent with pneumonia and associated with purulent tracheal secretion,
or a clinical syndrome associated with a high probability of infection. Infection was classified according to the organ of acquisition (CNS, RS, CVS, Skin & Soft tissue infection, GIT, GUrT, HB, Tropical infections), to the method of diagnosis (suspected, clinically documented through imaging, or microbiologically documented), to the microorganisms responsible when these were isolated, and to the organ(s) affected. It was recorded whether the initial antibiotic therapy was appropriate according to the antibiogram for the microorganisms responsible when these were isolated.

Statistical analysis

Quantitative data are described as mean \pm standard deviation (SD), medians and percentiles. Comparisons were performed using the Student t test or the Mann-Whitney U test, as appropriate; values of p < 0.05 were considered significant. Categorical data were analyzed by means of frequencies, percentages, and their confidence intervals. Patients were analyzed according to: (a) Spectral level of sepsis (at admission); (b) acquisition site of infection (CNS, RS, CVS, Skin & Soft tissue infection, GIT, GUrT, HB, Tropical infections); (c) micro-biological type of infection; (d) Risk factors associated with mortality were analyzed univariately and then multivariately by logistic regression; the degree of association with mortality was expressed as independent factors by means of odds ratios with their corresponding 95% confidence intervals. The statistical analysis was carried out by a professor at the Department of Biostatistics of Presidency College, who did not participate in collecting the data. All analyses were performed using SPSS statistical software.

Estimates of total cases expected annually were extrapolated from the number of cases, the period of observation, and the sampling fraction. Conflict of interest : Nil. External Financial support : Nil

OBSERVATIONS AND RESULTS



Out of the 1000 consecutively admitted patients 132 fulfilled the criteria for SIRS and were included in the study. 49.24% had sepsis, 32.58% had severe sepsis and 18.18% had septic shock. The death rate was 10.17% for sepsis, 18.60% for severe sepsis and 29.17% for septic shock.



Out of the 132 patients males were 77 and females 55. The incidence was the highest in the age group of 51-60 years and only in that age group female patients were more than male patients.



The previous chart demonstrates the distribution of male and female patients among the different categories of sepsis. Male patients were more in all the categories of sepsis.



The above chart reveals that the incidence of sepsis was roughly the same but the proportion of patients with severe illness was more with increased age.



The chart above shows the comorbidities in the various organ systems and the severity of illness associated with each. Comorbidities in CNS, GUrT, Hepatobiliary tract, CVS had higher percentage of incidence of severe disease, while if no comorbid condition is present proportion was less.



The above chart represents the distribution of the various source of infections in relation to the severity of sepsis. Tropical infections presented with milder disease predominantly while on comparison respiratory tract infections had higher proportion of more severe illness.



Respiratory tract remains the most common source of infection. The incidence of tropical infections was 24%. Next to respiratory system, genitor-urinary and hepatobiliary tract were the commonest source of infection.



Gram-positive and gram-negative organisms were isolated equally in the various specimens. CONS were the commonest isolate followed by Stap.aureus. Among the gram-negative organisms E.coli and klebsiella were the commonest. Candida was isolated from only one case. There was also isolate of MRSA from two samples of the same patient.



Positive blood cultures were obtained in only 32% of cases.Among the isolates CONS and Staph.aureus were again the most common. Acinetobacter, E.coli, Klebsiella and pseudomonas isolation was around 3% each. Only 2 of the 10 cases of widal positive enteric fever cases had positive blood culture.



Positive Urine cultures were obtained in only 30% of cases. Among the isolates E.coli, was the commonest. One each of candida, CONS and Proteus formed the rest of the isolates.



Sputum cuture had an yield of 35%. Klebsiella was the most common organism followed by throat commensals, CONS and staph. Strept.pneumoniae was present in one sample and so was Mycoplasma.



Cultures from other sites like ascitic fluid, pleural fluid, CSF, were mostly negative 79%. One each of E.coli, MRSA and pseudomonas was obtained. Evidence of tropical infections was positive in 28 cases.



The death rates were higher as the age advanced. The presence of other comorbid conditions places them under higher risk of mortality. The highest deaths were in the above 50 age group.



Females had a higher proportional mortality rate. 20% vs 12.18%.



Proportional mortality rate were higher for multi organ system comorbidities, followed by underlying malignancy, hematological diseases and cardiovascular co-morbidities. Though the lung was the commonest source of infection an underlying lung disease did not increase the mortality rate.



Mortality was higher when the source of infection was the lung, followed by genitourinary tract and hepatobiliary tract. Patients fared better when the source of infection was tropical infections, skin infections and nervous system infections. It is important to note that altered mental status at presentation carried a poorer prognosis.



The more the number of organ dysfunctions the more was the severity of disease in the grades of sepsis. More were the number of organs dysfunctioning as the severity of sepsis progressed and higher was the mortality when a higher number of organs dysfunctioned.



The more the number of organ dysfunctions the poorer was the prognosis and more was the mortality rate. From no deaths if only one organ is involved to 25% mortality with 2 organs to 45.45% mortality when 4 organs dysfunction.

DESCRIPTIVE STATISTICS

The following table shows the minimum and maximum values of the variables that were used in the analysis of relation to prognosis and outcome.

	Minimum	Maximum	Mean	Std. Deviation
GCS Score	3	15	12.66	3.475
Temperature	95.8	104.0	101.329	1.5808
Pulse rate	90	126	106.89	8.531
Respiratory rate	18.16	32.00	25.1897	3.14903
pH	6.91	7.54	7.2836	.12525
Sodium	126	146	136.55	5.764
Potassium	2.8	4.7	3.790	.5277
Hematocrit	22.536	43.820	34.19350	4.081372
Total Count	1800	33800	11767.73	4301.861
Platelet	.36	2.87	1.5123	.58341
Urea	31	162	57.14	23.237
Creatinine	.6	4.3	1.584	.9186
Systolic – BP	60	200	113.80	26.426
Diastolic – BP	40	120	73.42	13.864
Blood Sugar	66	287	116.62	33.136
Total Bilirubin	.4	5.4	1.584	1.0547
SGPT	31	214	62.53	26.624
SAP	34	157	66.10	21.559
Total Protein	5.0	8.3	6.326	.6591

	Minimum	Maximum	Mean	Std. Deviation
Albumin	2.0	4.9	3.322	.6207
pO ₂	70.1	97.0	89.356	5.3280
pCO ₂	26.0	63.1	42.686	7.7097
Hco3-	12.0	29.0	20.286	3.8433
PaO ₂ /FiO ₂	175.3	461.9	343.727	108.8780
Lactate	1.2	6.7	3.877	1.5501
APACHE-II score	23	53	33.39	6.482

The following table shows the variables with significant p values between the various categories of sepsis and in turn to the prognosis by using ANOVA.

		df	Mean Square	F	P value
Temperature	Between Groups	2	9.028	3.765	.026
	Within Groups	129	2.398		
pН	Between Groups	2	.134	9.714	.048
	Within Groups	129	.014		
Platelet	Between Groups	2	2.760	9.113	.032
	Within Groups	129	.303		
Urea	Between Groups	2	12342.012	34.571	.041
	Within Groups	129	357.004		
Creatinine	Between Groups	2	17.392	29.612	.027
	Within Groups	129	.587		

		df	Mean Square	F	P value
Systolic - BP	Between Groups	2	11451.402	21.541	.036
	Within Groups	129	531.597		
Diastolic - BP	Between Groups	2	6271.315	64.015	.029
	Within Groups	129	97.966		
Blood Sugar	Between Groups	2	1501.393	1.375	.256
	Within Groups	129	1091.754		
Total Bilirubin	Between Groups	2	25.932	35.642	.044
	Within Groups	129	.728		
SGPT	Between Groups	2	11417.504	21.034	.030
	Within Groups	129	542.804		
pO ₂ /FiO ₂	Between Groups	2	532803.25 5	141.040	.033
	Within Groups	129	3777.688		
Lactate	Between Groups	2	119.139	200.900	.045
	Within Groups	129	.593		
APACHE-II score	Between Groups	2	1025.520	38.320	.048
	Within Groups	129	26.762		

The following table shows the significance in prognostication of individual variables between the different groups of sepsis severity. The corresponding p values and the confidence limit are shown.

Dependent	(I) Category of	(J) Category of	Р	95% (Confidence
Variable	Severity	Severity	value	In	terval
				Lower	Upper
				Bound	Bound
Temperature	Severe Sepsis	Septic Shock	.021	.132	2.003
рН	SIRS/Sepsis	Septic Shock	.057	0036	.1296
Platelet	SIRS/Sepsis	Septic Shock	.016	.0559	.6792
Urea	Severe sepsis	Septic Shock	.277	-18.82	4.01
Creatinine	Severe sepsis	Septic Shock	.054	878	.048
Systolic –	SIRS/Sepsis	Severe sepsis	790	-13 71	7 78
BP			.170	15.71	7.70
Diastolic –	SIRS/Sepsis	Severe sepsis	973	-5.05	4 18
BP			.,,,,	5.05	1.10
Total	Severe sepsis	Septic Shock	004	-1 220	- 190
Bilirubin			.004	-1.220	170
SGPT	Severe sepsis	Septic Shock	.045	-26.27	1.88
PaO ₂ /FiO ₂	Septic Shock	Severe sepsis	.544	-53.651	20.614
Lactate	Septic Shock	Severe sepsis	.919	542	.388
APACHE-II	Severe sepsis	Septic Shock	005	-7 35	-1 09
score			.005	-1.55	-1.07

* The mean difference is significant at the .05 level.

		Out	tcom	e	Total
		Discharg	ge	Death	
Age Group in vears	Upto 20		18	1	19
5	21-30		22	2	24
	31-40		20	0	20
	41-50		19	3	22
	51-60		22	9	31
	Above		10	ſ	16
	60		10	U	10
		Value	df	Asyı	np. Sig. (2-sided)
Pearson Chi-Square		16.074 (a)	5	5	.007
Likelihood Ratio		18.024	5	5	.003
Linear-by-Linear Association		11.972	1	t 🔤	.001

Effect of Age Group on outcome

The greater the age the higher was the mortality rate as shown by the table above. Though the female sex had a higher proportional mortality rate the p values were not significant.

The presentation of hyperglycemia in the absence of diabetes history carried a poorer prognosis, underlying Diabetes had a bearing on the outcome though it was statistically significant. More patients with DM succumbed to their illness whereas non diabetic status had a better chance of survival.

Effect of DM on Outcome

			Outcome		Total
			Discharge	Death	
DM	Yes	Count	12	12	24
	No	Count	99	9	108
Total		Count	111	21	132

Effect of SHT on Outcome

			Outcome					Total
			Ľ	Discharge		Death		
SHT	Yes	Count		15			10	25
	No	Count		96			11	107
Total		Count		111			21	132
				Value		df	As	ymp. Sig. (2-sided)
Pearson	n Chi-Sq	uare		13.380(b))	1		.001
Continu	uity Corr	rection(a)		11.25)	1		.001
Likelih	ood Rati	0		11.147	7	1		.001
Fisher's	s Exact T	Test						
Linear- Associa	by-Linea tion	ar		13.278	3	1		.001

The presence of Hypertension had poorer survival rates that were statistically significant.

			Outcome		Total
			Discharge	Death	
Hypothemia	Yes	Count	3	2	5
		% within Hypothemia	60.0%	40.0%	100.0%
	No	Count	108	19	127
		% within Hypothemia	85.0%	15.0%	100.0%

Effect of Hypothemia on Outcome

The proportion of patients who died with hypothermia was more.

The ability to mount a response by producing fever conferred survival advantage. As was the case with leucopenia and leucocytosis, hyperglycemia and hypoglycemia. But a statistical significance was not got.

Effect of Leukopenia on Outcome

			Outcome		Total
			Discharge	Death	
Leukopenia	Yes	Count	12	4	16
		% within Leukopenia	75.0%	25.0%	100.0%
	No	Count	99	17	116
		% within Leukopenia	85.3%	14.7%	100.0%

			Outcome		Total
			Discharge	Death	
Hyperglycemia	Yes	Count	5	2	7
	% within		71 4%	28 60/2	100.0%
		Hyperglycemia	/ 1.4 /0	20.070	100.070
	No	Count	106	19	125
		% within	84.8%	15.2%	100.0%
		Hyperglycemia			100.070

Effect of Hyperglycemia on Outcome

Effect of No. of organ dysfunction on Outcome

		Outco	ome	Total
		Discharge	Death	
No.of organ dysfuntion	0	59	6	65
	1	4	0	4
	2	24	6	30
	3	18	4	22
	4	6	5	11
		Value	Asymp. Si	g. (2-sided)
Pearson Chi-Square		10.562(a)	.032	
Likelihood Ratio		9.610	.048	
Linear-by-Linear Association		7.353	.0	07

The number of organs that dysfunction has a strong association with the outcome. The p value was significant. The combinations of different dysfunctions were not important but the cumulative dysfunctioning of organ systems carried a higher mortality risk.

					Outcome		Total
					Discharge	Death	
Severity	SIRS/Sepsis	RS/Sepsis Count % within			59	6	65
					90.8%	9.2%	100.0%
	Severe sepsis	Count % within			35	8	43
					81.4%	18.6%	100.0%
	Septic Shock	Cou	unt		17	7	24
		% v	within		70.8%	29.2%	100.0%
			Value	df	Asym	Asymp. Sig. (2-sided)	
Pearson Chi-Square			5.554(a)	2	.032		
Likelihood Ratio			5.362	2	.038		
Linear-by-Linear Association			5.505	1	.019		

Effect of Category of Severity on Outcome

The severity of sepsis was also statistically significantly associated with mortality. The higher the grading of sepsis more was the mortality

DISCUSSION

The important findings of this study were: (a) the high incidence (13.2%) of sepsis in the medical wards and mortality (15.91%); (b) the factors associated with early death were higher organ dysfunctions at diagnosis, age, more severe category of sepsis, chronic co morbid illnesses, increased APACHE-II score, increased lactate levels.

Of the 1000 consecutive admissions during the period from May to October 2010, in the medical wards 132 patients satisfied the criteria of SIRS with documented evidence or strong clinical suspicion of infection. They were sub- classified further into SIRS/Sepsis, Severe sepsis and Septic shock based upon the definitions and criteria earlier described. Appropriate clinical presentation, imaging techniques and cultures from suspected compartments were used to confirm infection.

Standardized diagnostic criteria for sepsis, severe sepsis, septic shock, and organ dysfunction and failure associated with infection have enabled the epidemiological evaluation of septic syndromes, as well as of their progression in recent years and of the efficacy of new treatment measures. Using these diagnostic criteria, we found an incidence of Sepsis spectra of 13.2%, which is comparable to that of other series published in recent years.

Different authors have suggested that this progression might be related to the use of immunosuppressors, hospital malnutrition, alcoholism, cancer, diabetes mellitus, the growing invasiveness of both diagnostic and therapeutic measures, increased resistance of microorganisms, and the progressive aging of the population. The high incidence of other risk factors such as diabetes, chronic heart failure, kidney failure, liver failure, or respiratory failure compound this increase.

The projected incidence of sepsis spectra for the admissions covered by the medical wards in this hospital was 3854 per year, a figure that is lower than the incidence reported in recently published studies. This difference might be explained by seasonal bias, differences in the populations studied, and differences in access to hospitals. The mortality in this study (15.91%) differs from that published in the most recent series, which ranges from 28% to 48.4%. However, methodological differences with our study account for much of these differences. Selection bias could have been a reason. More number of patients had tropical infections and all of them recovered uneventfully

The incidence of sepsis spectra was 132/1000 (13.2%). Males formed 77 (58.33%) of the 132 and females 55 (41.66%). The incidence for the subsets were SIRS/Sepsis 49.24% (n=65), Severe sepsis 32.58% (n=43), Septic shock 18.18% (n=24). Across all age groups incidence were

more in the male gender. Incidence was highest in the age group of 51-60 years.

The Death rate was 15.91% (n=21), males 7.58% (n=10) and females 8.33% (n=11). The proportional death rate was higher among the females 20% (11/55) compared to males 12.99% (10/77). There was n statistical significance to this finding and is different from the other studies were mortality was more in the males. Among the subsets death was more frequent in the septic shock group than the severe sepsis or SIRS/Sepsis groups. The death rates were 9.2% 18.6% and 29.2% for SIRS/Sepsis, severe sepsis and septic shock respectively. The general trend of higher mortality with severity was as in the other studies but the percentage mortality rates were much lower than in those studies.

As in other recently published series, infection was most frequently located in the lungs. The incidence of tropical infections was much higher than in any of the quoted studies. Our region is endemic for malaria, enteric fever and leptospirosis. The incidence of dengue which has a seasonal preference was peaking during the time of this study. Hence around 21.21% of the cases were due to these infections. The better outcome following these infections could have been a factor for the difference in values by percentage on comparison to other studies.

The next common sources of infections were the genitourinary tract infections and hepato-biliary infections. Western studies show abdominal causes as the second most common infecting source. Our set up caters to a lot of patients who have decompensated liver disorder predominantly from alcoholic liver disorder and viral hepatitis. These patients commonly present with suspicion of spontaneous bacterial peritonitis. Though the cytology revealed evidence of infection and fluid was exudative, organisms could seldom be cultured. Though Diabetes mellitus is very prevalent skin and soft tissue infections seldom get admitted in the medical wards, this may be the reason skin and soft tissue infections were rarely the cause of sepsis in the ward.

Similar to the increased number of Gram-positive infections registered in recent years, the infections found in our patients were equally caused by Gram-positive and Gram-negative bacilli. The isolates and the percentage of the different microorganisms are as below .There was no correlation between the type of microorganisms and mortality, consistent with the earlier findings that all organisms whether gram-positive or negative were equipotent in producing sepsis.

Microbiological isolates were possible in only 54% of cases; one isolate from 39% and 2 from 15%.Candida was isolated from only one patient. The commonest isolate all samples included was CONS (22%), S.aureus (20%) and E.coli (14%) and Klebsiella (12%). One isolate of MRSA, S.pneumoniae and Enterococcus completed the gram positive spectrum. Pseudomonas, Acinetobacter, P.vulgaris, Citrobacter were the other gram negative isolates. Though Widal test was positive in 10 cases only one was culture positive. Mycoplasma was isolated from one sample.

Blood cultures were positive in 32% commonest isolates being CONS. Urine culture was positive in 30% cases the commonest isolate being E.coli. Sputum culture was positive in 35% and the commonest isolate was Klebsiella. E.coli was also the commonest isolate from all the other cultures - ascitic, pleural and cerebrospinal fluid. Though not intended in the study it was noted that almost all the isolates were susceptible to conventional antimicrobials like Ciprofloxacin, Amikacin, Sulphonamides and Erythromycin. The MRSA isolate was susceptible to vancomycin.

Among the cases 28 had tropical infections (Malaria, Enteric Fever, Dengue and Leptospirosis). Malaria and Enteric Fever with 10 cases each. 5 of them had leptospiriosis and 3 had dengue. All of them fully recovered.

The relation between increased mortality and both the number of organ failures at diagnosis and the progression toward multiple organ failure in septic patients is well established. To document the number of organ failure at diagnosis the criteria from SOFA score was used. Moreno et al. reported in 1999 that the there was a good correlation between organ failures and mortality. A Spanish multicentre study indicated that persistent organ failure is significantly associated with mortality.

The predictors of the outcome were underlying Diabetes or Hypertension. The prevalence of Diabetes mellitus and hypertension were 18.2% and 18.9% respectively similar to the last incidence in the study in NEJM analyzing sepsis between 1979 and 2000. Underlying comorbidities had a bearing in both determining the severity of illness and the mortality rate. When multi organ systems were affected the incidence of sepsis and the progression to higher stages were evident, the prognosis was also poor. No comobidities were present in 31 cases. CNS diseases were the commonest underlying cause followed by CVS, RS and HB system. Malignancy was present in only two patients. The percentage of severe manifestation of sepsis was higher with illness in CVS, GUrT, HB and CNS. Proportional mortality rates were higher for multi organ system comorbidities, malignancy, hematological diseases and cardiovascular comorbidities.

While analyzing the variables that were determining severity of sepsis and prognosis presentations with hypothermia (40%), leucopenia (25%), hyper-glycemia in the absence of Diabetes (28.6%), and acutely altered mental status (22.2%) had higher death rates, the same for the opposite entities were 15% with hyperthermia, 14.7% with leucocytosis, 15.2% without hyperglycemia, 14.3% without altered mental status. Though the commonest presentations in sepsis were fever, tachycardia, tachypnea, and leucocytosis, cases were admitted with hypothermia and leucopenia. Such presentations represent poorer response from the patient to an infection and hence place them under high risk of succumbing to the illness.

The category of severity at the time of presentation also had a bearing on the outcome. The less severe SIRS/Sepsis group had a death rate of 9.2%, while the rate was 18.6% for severe sepsis; it rose to 29.2% in the septic shock group.

Underlying co-morbid illnesses in multiple organ systems (60%), with underlying malignancy (50%), hematological diseases (40%), and CVS diseases (31%) had higher death rates. Those without any diseases, CNS diseases, HB diseases fared better. Though respiratory tract was the commonest site of infection an underlying RS disease did not have a poorer outcome. Poorer outcomes were noticed if the source of infection was the respiratory tract and the genitourinary tract. Outcomes were better with Tropical infections, CNS, Skin infections, GIT infections (all recovered).

Analyzing the physiological variables and the severity of illness it was noticed that the mean levels of systolic BP, Diastolic BP, Platelet counts, PaO2/FiO2 ratios, pH levels were lower in the septic shock group than the SIRS/Sepsis group, while the mean levels of APACHE-II scores, Lactate levels, Urea and creatinine levels, Total bilirubin and SGPT levels were higher in the septic shock group compared to SIRS/Sepsis group.

.

CONCLUSIONS

- The incidence of sepsis remains high in our population. Male patients were more in proportion. Higher age group had more severe disease and mortality rates. Females had higher mortality.
- \clubsuit The severity at presentation determines the prognosis.
- Underlying SHT, DM, and multi-system comorbidities at presentation have poorer outcomes.
- Hypothermia, Leucopenia, Hyperglycemia, altered mental status place the patients at poorer prognosis.
- Higher Creatinnine, Urea, Bilirubin, SGPT, Lactate, APACHE-II scores Lower pH (acidosis), platelet counts, Mean SBP and DBP, PaO₂/FiO₂ ratios were seen in more severe disease and portended poorer outcomes.
- * Respiratory tract remains the commonest source of infections.
- Both gram positive and gram negative organisms have equal incidence but do not have a bearing on the outcome.
Sepsis syndrome is common in academic hospitals, although the overall rates vary considerably with the patient population. A substantial fraction of cases occur outside ICUs. An understanding of the hospital wide epidemiology of sepsis syndrome is vital for rational planning and treatment of hospitalized patients with sepsis syndrome, especially as new and expensive therapeutic agents become available

LIMITATIONS OF THE STUDY

This study has some limitations.

First, although this is the first attempt of specifically designed epidemiological study of sepsis spectra in our medical wards the sample size was small and hence it is difficult to extrapolate the results to the general population.

Secondly, though there were differences between the different subgroups / outcome groups with regard to the different variables studied, a statistical significance could not be established in some of the variables. Nevertheless, this study can serve as a reference for future studies about these variables.

Third, the effect of seasonal variation on the incidence of sepsis is well known. [31]. The study period comprised of only summer-autumn, its effects are difficult to measure. The higher incidence of tropical infections may be because of this seasonal preference.

Thus, our results can also be useful as a reference for future studies about the impact of these measures when introduced on the outcome of patients with sepsis admitted to the medical wards in our setting.

LIST OF ABBREVIATIONS

1.	AcMenEn	:	Acute meningoencephalitis
2.	AGE	:	Acute gastroenteritis
3.	APACHE	:	Acute physiology and chronic health evaluation
4.	APTT	:	Activated partial thromboplastin time
5.	ARDS	:	Acute respiratory distress syndrome
6.	ASD	:	Atrial septal defect
7.	Asp Pn	:	Aspiration pneumonia
8.	BA	:	Bronchial asthma
9.	BrPn	:	Bronchopneumonia
10.	Ca Cx	:	Cancer cervix
11.	Ca Eso	:	Cancer esophagus
12.	CABpn	:	Community acquired Bronchopneumonia
13.	CAD	:	Coronary artery disease
14.	Cbl Mal	:	Cerebral malaria
15.	CD	:	Cluster of Differentiation
16.	CHF	:	Congestive heart failure
17.	ChrBritis	:	Chronic Bronchitis
18.	CIDP polyneuropa	: athy	Chronic inflammatory demyelinating
19.	CKD	:	Chronic kidney disease
20.	CML	:	Chronic myeloid leukemia

21.	CNS	:	Central Nervous system
22.	Comm	:	Commensals
23.	CONS	:	Coagulase negative staphylococcus
24.	COPD	:	Chronic obstructive pulmonary disease
25.	CRP	:	C-reactive protein
26.	CSOM	:	Chronic suppurative otitis media
27.	СТ	:	Computed tomography
28.	CVA	:	Cerebrovascular accident
29.	CVS	:	Cardiovascular system
30.	DCLD	:	Decompensated Liver Disease
31.	DCMP	:	Dilated cardiomyopathy
32.	Disch	:	Discharge
33.	DKA	:	Diabetic ketoacidosis
34.	DMT1/2	:	Diabetes mellitus type 1/2
35.	E.Coli	:	Escherechia coli
36.	EHPVO	:	Extrahepatic portal vein obstruction
37.	Ent Fev	:	Enteric fever
38.	GBS	:	Guillaine barre syndrome
39.	GCS	:	Glasgow coma scale
40.	GCSF	:	Granulocyte colony-stimulating factor
41.	GIT	:	Gastrointestinal system
42.	GUrT	:	Genitourinary system

43.	HB	:	Hepatobiliary system
44.	HE	:	Hepatic Encephalopathy
45.	HELLP	:	Hemolysis, Elevated liver enzymes, Low
	platelets		
46.	Hem	:	Hematological
47.	HIV	:	Human immunodeficiency virus
48.	HMGB1	:	High-mobility group box1
49.	HyKPP	:	Hypokalemic periodic paralysis
50.	ICU	:	Intensive Care Unit
51.	IFN	:	Interferon
52.	IL	:	Interleukin
53.	ILD	:	Interstitial lung disease
54.	ImmSup	:	Immunosuppression
55.	ITU	:	Intensive Trauma Unit
56.	Klebs	:	Klebsiella
57.	Lepto	:	Leptospirosis
58.	LPS	:	Lipopolysaccharide
59.	M.plasma	:	Mycoplasma
60.	Malig	:	Malignancy
61.	MIF	:	Macrophage migration inhibitory factor
62.	MODS	:	Multiple organ dysfunction syndrome
63.	MOS	:	Multiple organ systems

64.	MRSA	:	Methic illin resistant staph aureus
65.	Ν	:	No
66.	NG	:	No Growth
67.	P.vulg	:	Proteus vulgaris
68.	PAF	:	Platelet activating factor
69.	РСТ	:	Procalcitonin
70.	PHT	:	Portal hypertension
71.	PID	:	Pelvic inflammatory disease
72.	РрСМР	:	Postpartum cardiomyopathy
73.	PTb	:	Pulmonary tuberculosis
74.	PyNeph	:	Pyelonephritis
75.	RA	:	Rheumatoid arthritis
76.	rhAPC	:	Recombinant Human Activated Protein C
77.	RHD	:	Rheumatic heart disease
78.	RS	:	Respiratory system
79.	S.aureus	:	Staphylococcus aureus
80.	S.pneum	:	Streptococcus pneumonia
81.	SBP	:	Spontaneous bacterial peritonitis
82.	SD	:	Standard deviation
83.	Seiz Dis	:	Seizure disorder
84.	Sev Sep	:	Severe sepsis
85.	SHT	:	Systemic hypertension

86.	SIRS	:	Systemic inflammatory response syndrome
87.	SLE	:	Systemic lupus erythematosus
88.	SS	:	Septic shock syndrome
89.	TLR	:	Toll-like receptors
90.	$TNF-\alpha$:	Tumor necrosis factor α
91.	UGIB	:	Upper gastrointestinal bleed
92.	UTI	:	Urinary tract infection
93.	WBC	:	White blood cell count
94.	Y	:	Yes

Name	:	
Age	:	
Gender	:	
Complete Diagnosis	:	
Is the patient's history suggestive of	a new infection?	
 Pneumonia, empyema Urinary tract infection Acute abdominal infection Meningitis Skin/soft tissue infection 	 Bone/joint infection Wound infection Bloodstream catheter infection Endocarditis 	Implantable device infection Other
Are any two of following signs & syn laboratory values may have been ob	nptoms of infection both present and ner tained for inpatients but may not be avai	YesNo w to the patient? <u>Note</u> : lable for outpatients.
 Hyperthermia > 38.3 °C (101.0 °F) Hypothermia < 36 °C (96.8°F) Tachycardia > 90 bpm 	 Tachypnea > 20 bpm Acutely altered mental status Leukocytosis (WBC count >12,000 μL-1) 	Leukopenia (WBC count < 4000 μL-1) Hyperglycemia (plasma glucose >120 mg/dL) in the absence of diabetes
		YesNo
Are any of the following organ dysfund	tion criteria present at a site remote from	the site of the

PROFORMA

- 3. infection that are not considered to be chronic conditions? Note: the remote site stipulation is waived in the case of bilateral pulmonary infiltrates.

 - SBP < 90 mmHg or MAP < 65 mmHg
 SBP decrease > 40 mm Hg from baseline
 - SBP decrease > 40 mm Hg from baseline
 Bilateral pulmonary infiltrates with a new (or increased) oxygen requirement to maintain SpO2 > 90%
 Bilateral pulmonary infiltrates with PaO2/FIO2 ratio < 300

 - Creatinine > 2.0 mg/dl (176.8 mmol/L) or Urine Output < 0.5 ml/kg/hour for > 2 hours Bilirubin > 2 mg/dl (34.2 mmol/L)

 - Platelet count < 100,000

1.

2.

- Coagulopathy (INR >1.5 or aPTT >60 secs)
 Lactate > 2 mmol/L (18.0 mg/dl)

___Yes ___No

80

Table 2. The APACHE II Severity of Disease Classification System§

Physiologic Variable	High Abnormal Range Low Abnormal Range									
	+4	+3	+2	+1	0	+1	+2	+3	+4	Points
Temperature - rectal (C)	41	39 to 40.9		38.5 to 38.9	36 to 38.4	34 to 35.9	32 to 33.9	30 to 31.9	29.9	
Mean Arterál Pressure – mm Hg	160	1 30 to 159	110 to 129		70 to 109		50 to 69		49	
Heart Rate (ventricular response)	180	140 to 179	110 to 139		70 to 109		55 to 69	40 to 54	39	
Respiratory Rate	50	35 to 49		25 to 34	12 to 24	10 to 11	6 to 9		5	
(non-ventilated or ventilated)										
Oxygenation: A-a DO ₂ or PaO ₂ (mm Hg)	500	350 to 499	200 to 349		<200					
a. FIO ₂ 0.5 record A-aDO ₂		L		L						
b. FIO2 <0.5 record PaO2					PO≫70	PO2 61 to 70		PO2 55 to 60	PO2<55	
Arterial pH (preferred)	7.7	7.6 to 7.69		7.5 to 7.59	7.33 to 7.49		7.25 to 7.32	7.15 to 7.24	<7.15	
Serum HOOs (venous mEqII)	52	41 to 51.9	1	32 to 40.9	22 to 31.9		18 to 21.9	15 to 17.9	<15	1
(not preferred, but may use if no ABGs)										
Serum Sodium (mEq1)	180	160 to 179	155 to 159	150 to 154	130 to 149		120 to 129	111 to 119	110	
Serum Potassium (mEqI)	7	6 to 6.9		5.5 to 5.9	3.5 to 5.4	3 to 3.4	2.5 to 2.9		<2.5	
Serum Creatinine (mg/dl)	3.5	2 to 3.4	1.5 to 1.9		0.6 to 1.4		<0.6			
Double point score for acute renal failure	1									
Hematocrit (%)	60		50 to 59.9	46 to 49.9	30 to 45.9		20 to 29.9		<20	
White Blood Count (total/mm ³)	40		20 to 39.9	15 to 19.9	3 to 14.9		1 to 2.9		<1	
(in 1000s)										
Glasgow Coma Score (GCS)										
Score = 15 minus actual GCS										
A. Total Acute Physiology Score (sum of 12 a	bove points)									
B. Age points (years) 44 = 0;		45 to 54	=2; 5	35 to 64 =3;	65 to 74 = 5;	75=6				
C. Chronic Health Points (see below)										
Total ADACHE II Sears (add togo	Table ADACHE III Const. (add in a the spine from A (D) (C)									

Total APACHE II Score (add together the points from A+B+C) Chronic Health Points: If the patient has a history of severe organ system insufficiency or is immunocompromised as defined below, assign points as follows:

5 points for nonoperative or emergency postoperative patients

2 points for elective postoperative patients

Definitions: organ insufficiency or immunocompromised state must have been evident prior to this hospital admission and conform to the following criteria: Liver-biopsy proven cirrhosis and documented portal hypertension; episodes of past upper Gilibeieding attributed to portal hypertension; or prior episodes of hepatic failure/encephalopathylocoma. Cardiovascular – New York Heart Association Class IV. Respiratory – Chronic restrictive, obstructive, or vascular disease resulting in severe exercise restriction (i.e., unable to dimb stairs or perform household duties; or documented chonic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension (>40 mmHg), or respirator dependency. Renal – neceiving chonic dialysis. Immunocompromised – the patient has received therapy that suppresses resistance to infection (e.g., immunosuppression, chemotherapy, radiation, ong term or recent high dose steroids, or has a disease that is sufficiently advanced to suppress resistance to infection, e.g., leukemia, lymphoma, AIDS).

interpretation of Score:						
0 to 4 = ~4% death rate	10 to 14 = ~15% death rate	20 to 24 = ~40% death rate	30 to 34 = ~75% death rate			
5 to 9 = ~8% death rate	15 to 19 = ~25% death rate	25 to 29 = ~55% death rate	Over 34 = ~85% death rate			
Vitals ·						

vitals.					
Temp	Pulse	BP	Resp Rate	JVP	Hydn
-			-		Status

CBC	RFT	LFT	ABG	
TC	Urea	Bilirubin(T)	pН	
DC	Creatinine	Bilirubin(D)	pCO2	
ESR	Sodium	Bilirubin(I)	HCO3-	
Hb%	Potassium	SGOT	pO2	
PCV	Sugar	SGPT	BE	
Plat	BT	SAP	AG	
Bld Gp	PT/INR	TP	Lactate	
Rh	aPTT	Alb/Glob		
Туре				

MicroBiological Examination : As Appropriate for clinical setting

Blood	
Culture/Sensitivity	
Culture, Sensiti vity	
I Inin a	
Urine	
~	
Culture/Sensitivity	
-	
CSF	
Culture/Sensitivity	
Culture/Selisiti vity	
XX7 1	
Wound	
Culture/Sensitivity	
Sputum	
~F	
Culture/Sensitivity	
Culture, Sensiti vity	
0.1	
Others	
Culture/Sensitivity	

Smear MP/MF	Lepto MSAT	
Dengue IgM/IgG	Widal Test	

Peripheral Smear	
Chest X-ray	
USG – Abd	
ECG	
ЕСНО	

:

Treatment

Fluid Therapy (Total)

Crystalloids	
Colloids	
Blood Products	

Vasopressors/Inotropic Therapy (Total)

Isoproterenol	
Dopamine	
Epinephrine	
Norepinephrine	
Dobutamine	

Anti-Biotics Used/Dose / No.of Dys

Steroids	Yes /	No	Dose :
Mechanical Ventilation			
Glucose Control			
Renal Replacement			
Bicarbonate Therapy			
Deep Vein Thrombosis Prophylaxis			
Stress Ulcer Prophylaxis			
Sedation, Analgesia, and NM Blockade			
Surgical Interventions			

Outcome

:

Чо		je	der	nia>38.3°C .0°F)	hemia 96.8°F)	a >90 bpm	a >20 bpm	sis (WBC 000 µL-1)	penia count עב-1)	mia (>120 n-diabetic	altered status		JU J	5				VITALS		
S.P	Name	Ag	Gen	Hyperthem (101.	Hypotl <36°C ((Tachycardi	Tachypnea	Leukocyto count >12,	Leuko (WBC <4,000	Hyperglyce mg/dL)noi	Acutely mental	Е	^	М	Т	Temp	Pulse	SBP	DBP	RR
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
1	Velu	34	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	114	110	78	23
2	Vijayaragavalu	44	Μ	Y	N	Y	Y	Y	Ν	Ν	Y	4	4	5	13	101.4	104	100	70	29
3	Kumaresan	17	М	Y	N	Y	Y	Y	Ν	Ν	Y	2	3	6	11	101.2	98	120	82	29
4	Saraswathy	54	F	Y	N	Y	Y	Y	Ν	Ν	Y	2	2	5	9	101.4	112	84	52	28
5	Mekala	26	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102.4	98	110	70	22
6	Kumar	38	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	4	5	12	101.4	116	110	80	26
7	Saraswathy	57	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	5	14	101	98	120	80	27
8	Vijayakumar	49	Μ	Y	N	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	98	100	74	23
9	Ramesh	30	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	4	6	13	101.4	100	106	80	21
10	Arpudhasagayaraj	30	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	4	5	12	101.4	118	110	76	30
11	Amaravathy	40	F	Y	N	Y	Y	Y	Ν	Y	Y	2	2	4	8	101.4	110	120	68	28
12	Soundary	43	F	Y	Ν	Y	Y	Y	Ν	Ν	Y	3	3	5	11	103	126	100	70	21
13	Shanthi	38	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	102	94	72	22
14	Thyagarajan	40	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102.6	120	80	60	27
15	Gurusamy	55	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	4	5	13	102.6	120	140	90	23
16	Mohan	58	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Y	2	3	5	10	95.8	100	102	72	24
17	Balan	30	Μ	Ν	Y	Y	Y	Y	N	Ν	Y	2	2	4	8	96.6	92	90	60	23

MASTER CHART

18	Gunalan	54	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102.6	102	110	70	24
19	Valliammal	44	F	Y	Ν	Y	Y	Y	Ν	Ν	Y	2	3	5	10	95.8	110	150	92	23
20	Rajendran	58	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	4	5	13	102	120	160	90	24
21	Ravi	44	М	Y	Ν	Y	Y	Y	Ν	Y	Ν	4	5	6	15	101.8	112	140	88	23
22	Selvaray	62	Μ	Ν	Y	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102	106	118	78	24
23	Jayalakshmi	70	F	Y	Ν	Y	Y	Ν	Y	Ν	Y	2	4	5	11	96.2	96	130	80	23
24	Chengammal	34	F	Y	Ν	Y	Y	Y	Ν	Y	Ν	3	5	6	14	102.2	102	100	50	27
25	Venkatasubbiah	45	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102.2	118	100	60	23
26	Jayanthi	53	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	3	5	11	101	108	120	40	29
27	Latha	30	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	2	2	4	8	102.4	108	70	56	28
28	Moorthy	27	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101	102	100	70	24
29	Nagaraj	32	Μ	Y	Ν	Y	Y	Ν	Y	Ν	Ν	4	5	6	15	103	116	110	74	26
30	Subbiah	23	М	Y	Ν	Y	Y	Y	Ν	Ν	Y	2	2	3	7	101.4	104	78	54	28
31	Vasanth	28	Μ	Y	Ν	Y	Y	Ν	Y	Ν	Ν	4	5	6	15	101.4	116	106	76	30
32	Vijaya	53	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	104	160	90	22
33	Chokkammal	51	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	4	5	12	104	108	150	94	28
34	Amul	21	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	122	120	80	28
35	Sathya	17	F	Ν	Y	Y	Y	Y	Ν	Ν	Ν	3	4	6	13	101.6	96	70	48	21
36	Kondammal	63	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	104	112	76	24
37	Раррі	43	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	100	118	76	30
38	Raja	17	М	Y	Ν	Y	Y	Y	Ν	Ν	Y	2	3	5	10	101.4	112	110	80	27
39	Ragini	16	F	Y	Ν	Y	Y	Ν	Y	Ν	Ν	4	5	6	15	101.4	108	108	78	21
40	Devaki	48	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102.6	118	150	90	26
41	Saravanan	24	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	106	120	74	27
42	Ashok	18	М	Y	Ν	Υ	Y	Υ	Ν	Ν	Ν	4	5	6	15	102.4	118	110	76	21
43	Naseer ahmed	54	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	108	170	100	25
44	Devi	21	F	Y	Ν	Y	Y	Y	N	Ν	N	3	4	6	13	101.4	106	100	70	30

45	Masthan	78	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	3	5	11	101.4	112	120	80	22
46	Rajarajan	40	Μ	Y	Ν	Y	Y	Ν	Y	Ν	Ν	3	5	6	14	101.6	110	110	70	22
47	Thangammal	52	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	110	120	76	27
48	Anjeeswaran	53	М	Y	Ν	Y	Y	Ν	Y	Ν	Y	2	2	4	8	102.2	104	80	50	26
49	Sundaresan	16	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	4	6	13	103	108	110	70	29
50	Lalitha	39	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102	94	102	70	25
51	Kondiah	70	М	Y	Ν	Y	Y	Y	Ν	Ν	Y	2	2	4	8	101.4	114	150	88	24
52	Ramachandran	70	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	4	5	12	102.2	118	200	120	24
53	Muralidaran	20	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102.4	120	110	70	24
54	Revathi	70	F	Y	Ν	Y	Y	Y	Ν	Ν	Y	2	2	4	8	96	100	80	40	28
55	Rajeswari	38	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	104	110	70	27
56	Anand	15	М	Y	Ν	Y	Y	Y	Ν	Ν	Y	3	3	4	10	101.4	98	110	80	23
57	Senthilkumar	25	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Y	3	3	5	11	101	100	100	70	24
58	Dhanammal	64	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	96	108	78	28
59	Beevi	52	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	114	110	80	27
60	Venakatramanamma	24	Μ	Y	Ν	Y	Y	Ν	Y	Ν	Ν	3	5	6	14	102.2	100	120	80	21
61	Jeganathan	18	Μ	Y	Ν	Y	Y	Ν	Y	Ν	Ν	4	5	6	15	101.6	96	110	78	27
62	Kuttiammal	55	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	102	160	90	30
63	Meenatchi	21	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	3	5	11	102	94	80	80	21
64	Thulasiammal	60	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	5	6	14	101.6	112	80	80	26
65	Muniyammal	60	F	Y	Ν	Y	Y	Y	Ν	Y	Ν	4	5	6	15	101.4	106	150	96	20
66	Shanthi	50	F	Y	Ν	Υ	Y	Υ	Ν	Ν	Ν	4	5	6	15	102	106	140	90	21
67	Viji	19	F	Y	Ν	Y	Y	Υ	Ν	Ν	Ν	4	5	6	15	101.4	122	180	90	29
68	Polammal	65	F	Ν	Y	Y	Y	Y	Ν	Y	Ν	2	2	4	8	101.4	116	70	50	27
69	Shanthi	35	F	Y	Ν	Y	Y	Ν	Y	Ν	Ν	4	5	6	15	102	104	100	68	27
70	Premavathy	57	F	Y	Ν	Y	Y	Y	Ν	Ν	Y	1	1	1	3	96	94	84	50	25
71	Bhavanakumar	51	М	Y	Ν	Y	Y	Ν	Y	Ν	Y	2	2	4	8	96.4	100	130	40	28

72	Nirmala	55	F	Y	Ν	Y	Y	Ν	Y	Ν	Ν	3	3	5	11	101.8	96	160	94	20
73	Thangadurai	56	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	5	6	14	101.4	104	170	96	23
74	Shanmughi	20	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.8	96	120	70	25
75	Parimala	25	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.6	96	116	76	20
76	Sakunthala	65	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102.8	108	170	90	25
77	Mohan	45	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	3	4	6	13	102	120	120	80	28
78	Selvi	40	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102	102	102	70	25
79	Subramani	36	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	4	5	13	101.4	100	100	66	22
80	Koottees	23	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	102.6	110	108	70	24
81	Loganathan	55	М	Y	Ν	Υ	Y	Υ	Ν	Ν	Ν	2	3	5	10	101.4	104	110	68	22
82	Kasi	40	М	Y	Ν	Υ	Y	Y	Ν	Ν	Ν	3	5	5	13	102.4	108	120	80	28
83	Sudharshan	21	М	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	1	10	102	102	110	80	29
84	Kishore	15	М	Y	Ν	Υ	Y	Υ	Ν	Ν	Y	2	2	4	8	96	90	120	80	24
85	Rajasekaran	67	М	Y	Ν	Υ	Y	Y	Ν	Ν	Ν	4	5	6	15	101.8	116	100	78	29
86	Indirani	60	F	Y	Ν	Y	Y	Y	Ν	Y	Ν	4	5	6	15	101.6	106	110	80	24
87	Arumugam	45	М	Y	Ν	Y	Y	Υ	Ν	Ν	Ν	3	5	6	14	101.4	118	116	76	21
88	Arumugam	41	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	1	5	10	102	98	70	50	23
89	Amaravalli	30	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.8	106	100	60	26
90	Loganathan	21	М	Y	Ν	Υ	Y	Υ	Ν	Ν	Ν	4	5	6	15	101.4	92	110	70	27
91	Hanifa	66	Μ	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	102	130	50	21
92	Dhanapriya	87	М	Ν	Y	Y	Y	Y	Ν	Ν	Y	2	2	5	9	96.2	116	70	50	20
93	Madan	18	М	Y	Ν	Y	Y	Υ	Ν	Ν	Ν	4	5	6	15	101.4	110	100	70	21
94	Govindaraj	80	М	Y	Ν	Υ	Y	Ν	Y	Ν	Y	2	2	4	8	101.6	108	110	80	29
95	Pushpammal	50	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.6	108	120	80	24
96	Lakshmi	55	F	Y	Ν	Υ	Y	Υ	Ν	Ν	Ν	4	5	6	15	102	120	110	80	31
97	Gunaselvi	58	F	Y	Ν	Y	Y	Y	Ν	Ν	Ν	4	5	6	15	101.4	118	106	76	26
98	Haribabu	16	Μ	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.4	100	110	78	28

99	Ayyasamy	64	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.8	110	100	70	24
100	Janakiraman	24	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.4	116	80	50	28
101	Kaliyuganga	51	F	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.8	98	120	80	27
102	Pandi	47	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.4	110	110	76	30
103	Mallika	43	F	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.6	94	120	80	29
104	Anjalai	15	F	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	102.4	108	110	70	29
105	Manjula	53	F	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.4	114	160	90	21
106	Eswari	44	F	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.4	96	110	70	29
107	Chandrasekaran	40	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.4	110	120	70	32
108	Muthalagan	53	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	102	122	110	78	27
109	Karunakaran	50	М	Y	N	Y	Y	Y	Ν	Ν	N	3	4	6	13	101.4	112	100	70	27
110	Kuppammal	60	F	Y	N	Y	Y	N	Y	Y	N	4	5	6	15	101.4	98	190	100	21
111	Sathya	34	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.4	96	100	80	21
112	Kasiammal	76	F	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.8	110	102	76	27
113	Gowri	38	F	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101	100	110	80	27
114	Elumalai	48	М	Y	N	Y	Y	N	Y	Ν	Y	2	2	4	8	101.4	112	88	58	20
115	Ponnusamy	53	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.4	114	180	102	26
116	Ruby	35	М	Y	N	Y	Y	Y	Ν	Ν	Y	2	2	5	9	101.4	108	96	66	27
117	Harishankar	15	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.8	110	112	87	26
118	Amudha	48	F	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	102.4	96	126	80	22
119	Pushpa	36	F	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101	98	110	80	29
120	Vignesh	18	М	Y	N	Y	Y	Y	Ν	Ν	N	3	3	5	11	101.4	116	120	80	23
121	Arun	23	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	102	126	110	70	27
122	Rubin	15	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	102	124	100	76	22
123	Sivanathan	29	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	102.8	98	88	56	18
124	Prakash	31	М	Y	N	Y	Y	Y	Ν	Ν	N	2	4	5	11	101.4	104	96	70	22
125	Abdul raheem	60	М	Y	N	Y	Y	Ν	Y	Ν	Y	2	2	4	8	102.6	122	80	50	26

126	Sekar	47	Μ	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	101.4	102	100	70	28
127	Senthil	27	М	Y	N	Y	Y	Ν	Y	Ν	N	3	4	6	13	101.4	112	80	64	22
128	Vinoth	19	М	Y	N	Y	Y	Y	Ν	Ν	Y	2	2	4	8	96.4	96	60	40	26
129	Preethishanthi	22	F	Y	N	Y	Y	Y	Ν	Ν	Y	3	1	5	9	101	98	106	72	20
130	Ramachandran	49	М	Y	Ν	Y	Y	Y	Ν	N	Ν	4	5	6	15	102.8	100	100	70	22
131	Ravanappa	47	М	Y	N	Y	Y	Y	Ν	Ν	Y	2	1	4	7	102.4	102	190	94	28
132	Thangappan	55	М	Y	N	Y	Y	Y	Ν	Ν	N	4	5	6	15	103	113	140	80	31

		CBC						RFT					LFT						ABG				gan oning	score
HCt	TC	DC	ESR	%dH	Plat	Urea	Creat	Sodium	Pottassium	sugar	Bil(T)	SGPT	SAP	ТР	Alb	Hd	FiO2	p02	pCO2	нсоз-	PaO2/FiO2	Lactate	No.of or dysfunctio	APACHE II
22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46
36.3	11600	66/28/6	31	11.6	2.68	31	1.0	137	4.4	98	1.0	40	43	6.1	3.4	7.34	21.0	94.0	46.0	24.0	447.6	2.0	0	30
31.3	12000	66/28/6	52	10.0	0.94	78	1.6	129	4.4	80	4.9	86	67	5.6	2.3	7.29	40.0	85.1	37.0	17.5	212.8	4.2	4	35
35.7	12600	70/26/4	25	11.4	2.66	40	0.8	143	4.5	96	0.9	49	45	6.7	4.0	7.50	21.0	92.4	32.0	26.0	440.0	3.0	0	30
31.3	12800	64/28/8	30	10.0	1.10	69	1.8	130	4.4	129	3.3	98	40	6.2	3.0	7.24	40.0	81.1	28.0	12.0	202.8	4.6	3	38
35.2	12080	82/15/3	38	11.3	2.09	34	0.7	131	3.7	112	1.2	46	93	6.7	3.2	7.41	21.0	94.3	38.0	23.2	449.0	2.4	0	24
41.3	12160	70/23/7	58	13.2	1.92	37	1.0	129	3.0	117	1.0	51	92	6.4	3.6	7.49	21.0	94.0	28.0	22.0	447.6	2.3	0	29
32.6	13400	66/28/6	78	10.4	1.37	44	1.1	135	3.1	124	1.2	56	77	6.0	3.3	7.35	21.0	92.0	48.0	25.8	438.1	2.0	0	28
30.7	12184	61/35/4	45	9.8	2.44	36	1.2	139	3.5	76	0.9	38	38	6.1	3.0	7.39	21.0	91.0	39.0	23.0	433.3	1.8	0	23
35.2	11880	61/35/4	60	11.2	1.57	39	0.9	128	2.8	88	0.8	41	80	6.0	3.3	7.29	21.0	92.0	60.0	28.0	438.1	2.9	0	29
35.1	11840	65/28/7	76	11.2	1.60	78	2.3	132	4.0	110	1.1	38	54	6.3	2.3	7.03	40.0	87.0	45.0	14.0	217.5	4.3	3	38
35.2	11960	61/32/7	45	11.2	1.20	114	3.6	133	3.0	126	1.2	50	72	5.8	2.8	7.15	40.0	94.0	54.0	20.0	235.0	4.4	2	34
28.2	33800	72/22/6	21	9.0	0.90	43	0.8	130	3.0	94	0.9	41	82	6.1	2.6	7.05	40.0	91.2	39.7	12.7	227.9	6.1	3	44
32.6	12120	60/34/6	54	10.4	1.04	41	0.9	129	3.7	102	1.1	64	70	6.3	3.3	7.33	21.0	95.0	44.6	23.0	452.4	1.8	0	25
35.2	11520	64/28/8	45	11.2	1.54	73	1.6	139	4.3	66	1.9	64	49	6.2	2.4	7.06	40.0	86.0	43.0	13.9	215.0	4.5	2	40
30.7	12200	61/35/4	25	9.8	1.02	92	2.9	134	3.7	107	1.7	87	48	6.3	2.3	7.35	40.0	84.0	26.0	14.0	210.0	5.7	3	36
37.6	13800	74/22/4	49	12.0	1.91	46	0.8	146	3.8	120	1.0	36	46	6.3	3.2	7.49	21.0	90.0	34.0	26.0	428.6	3.4	0	37
35.1	11800	64/28/8	70	11.2	1.74	54	1.6	133	4.2	97	1.2	44	54	7.7	4.7	7.29	40.0	87.3	34.0	16.0	218.2	4.7	2	34

40.7	13000	69/26/5	39	13.0	2.30	44	1.2	132	4.1	103	1.1	55	69	6.8	3.4	7.33	21.0	94.0	45.0	23.0	447.6	2.0	0	28
40.7	11960	68/26/6	77	13.0	1.19	39	1.1	136	3.6	111	0.9	70	100	5.6	2.6	7.11	21.0	91.5	52.0	18.0	435.6	2.3	0	40
36.3	14000	66/23/11	78	11.6	0.98	98	3.1	129	4.1	102	2.2	66	48	6.9	3.8	7.07	40.0	84.0	49.0	16.0	210.0	6.0	4	45
40.7	12256	74/22/4	38	13.0	1.04	36	0.9	145	3.3	132	0.9	32	56	6.4	3.1	7.31	21.0	89.0	45.3	22.0	423.8	1.9	0	29
31.3	12184	65/28/7	39	10.0	2.27	47	1.0	135	3.2	112	0.7	42	43	6.2	3.5	7.39	21.0	97.0	41.0	24.0	461.9	1.8	0	28
30.7	2900	66/23/11	26	9.8	1.02	47	0.8	128	3.4	136	0.8	55	84	5.3	3.2	7.36	21.0	88.9	36.0	19.8	423.3	2.0	0	36
34.4	11560	74/22/4	58	11.0	0.95	43	1.1	131	4.4	142	3.3	134	81	6.0	3.0	7.13	40.0	90.0	51.0	18.3	225.0	6.1	4	30
35.0	12112	76/22/2	63	11.2	1.85	33	1.2	134	3.6	92	2.7	78	34	6.6	2.6	7.33	21.0	91.5	43.2	22.1	435.7	2.9	0	33
32.2	11400	70/23/7	78	10.3	0.80	90	4.3	135	3.6	157	4.4	97	61	6.1	2.9	7.14	40.0	97.0	43.7	15.8	242.5	6.1	4	43
22.5	11760	62/32/6	49	7.2	0.60	74	2.9	137	4.3	111	5.4	214	143	5.1	2.9	7.43	40.0	89.0	37.0	23.9	222.5	5.3	4	37
43.8	12040	61/32/7	79	14.0	0.70	62	2.0	133	3.2	86	1.0	36	83	6.1	3.8	7.31	40.0	93.0	45.8	22.4	232.5	4.3	4	29
35.1	2160	80/13/7	57	11.2	1.87	43	0.9	136	3.5	112	0.6	41	99	5.3	3.0	7.40	21.0	91.0	36.9	22.3	433.3	3.1	0	29
 33.2	12400	66/28/6	69	10.6	1.09	77	2.3	133	4.4	107	2.5	146	51	6.8	3.3	7.25	40.0	79.6	37.0	16.0	199.0	4.9	4	37
 35.2	3100	69/26/5	65	11.2	1.02	34	0.6	136	4.5	94	0.8	37	43	6.4	2.9	7.27	21.0	93.0	50.0	22.6	442.9	2.9	0	28
38.8	12200	60/34/6	77	12.4	2.87	59	1.6	144	3.9	146	0.7	40	46	5.5	2.7	7.36	21.0	95.0	45.0	24.8	452.4	1.9	0	32
33.9	16000	61/35/4	60	10.8	1.48	64	1.7	140	3.9	130	1.6	68	85	5.9	3.0	7.18	40.0	86.3	49.0	19.0	215.8	6.2	2	37
 31.3	12400	61/35/4	68	10.0	2.02	47	0.9	130	2.9	106	1.1	53	100	5.5	3.2	7.35	21.0	92.3	41.7	22.4	439.5	3.7	0	28
33.2	12800	64/32/4	46	10.6	0.77	79	1.9	135	3.0	162	0.8	66	96	6.8	4.2	7.05	40.0	95.3	51.0	16.2	238.1	6.1	2	33
33.2	12192	64/28/8	58	10.6	1.53	83	2.9	132	3.5	104	0.9	61	79	8.0	4.8	7.32	40.0	86.0	32.0	16.0	215.0	5.7	3	31
31.3	12080	70/27/3	29	10.0	1.37	54	0.8	142	3.6	95	1.0	37	85	5.8	3.2	7.36	21.0	93.5	42.4	23.0	445.2	2.3	0	24
35.1	13000	70/23/7	26	11.2	0.77	70	1.6	136	3.8	118	1.9	120	62	6.4	3.4	7.08	40.0	94.1	45.0	14.9	235.3	6.5	2	37
27.5	3200	70/27/3	74	8.8	1.32	43	0.8	146	4.2	134	0.9	56	50	6.7	3.6	7.37	21.0	93.0	38.0	21.0	442.9	2.0	0	25
 34.9	12160	64/28/8	61	11.1	1.12	134	4.2	131	4.0	114	1.3	88	38	5.1	2.5	7.28	40.0	79.0	28.0	13.0	197.5	4.2	3	37
 32.2	12280	66/23/11	45	10.3	1.94	43	1.1	146	3.7	98	0.8	48	74	6.4	3.5	7.33	21.0	93.0	45.0	23.0	442.9	2.0	0	24
40.1	13200	60/34/6	30	12.8	1.31	47	0.7	132	3.5	112	1.0	70	77	6.4	4.0	7.37	21.0	91.0	38.0	21.0	433.3	2.1	0	26
40.7	13200	74/22/4	17	13.0	1.45	54	1.0	137	4.3	120	0.9	67	62	5.6	2.9	7.33	21.0	94.6	41.1	21.1	450.5	2.0	0	26
35.7	12600	65/28/7	66	11.4	0.91	53	1.4	137	3.0	103	2.5	135	75	5.5	3.0	7.26	40.0	91.0	46.0	20.3	227.5	4.1	3	29

40.1	14000	74/22/4	42	12.8	1.35	60	1.3	144	4.7	88	1.0	69	96	6.0	2.9	7.21	40.0	95.3	57.3	23.3	238.3	4.9	2	38
27.9	3680	49/46/5	22	8.9	1.17	40	1.1	140	4.6	106	0.7	42	74	6.7	4.1	7.40	21.0	93.1	40.1	24.0	443.3	3.0	0	29
35.1	11480	62/32/6	30	11.2	2.28	47	0.9	137	4.0	98	0.9	71	50	7.8	3.5	7.23	21.0	89.9	49.7	21.0	428.1	2.1	0	31
30.7	3300	62/32/6	36	9.8	2.85	80	2.2	128	3.8	200	2.8	133	47	6.1	4.0	7.14	40.0	94.9	39.5	14.4	237.3	5.9	4	43
38.5	12200	81/13/6	60	12.3	1.75	72	1.2	129	4.3	100	0.8	49	54	6.3	3.0	7.27	40.0	95.4	31.0	14.0	238.6	5.6	2	33
35.2	12000	82/15/3	16	11.3	1.20	45	0.9	134	3.9	108	1.2	45	37	6.9	3.4	7.32	21.0	93.6	41.8	21.0	445.7	1.9	0	27
33.9	13200	76/22/2	22	10.8	1.13	56	0.9	134	3.8	167	0.7	41	95	6.8	3.5	7.42	21.0	93.0	37.5	23.5	442.9	1.8	0	37
31.3	13400	72/23/5	46	10.0	1.18	77	3.9	144	4.3	97	0.8	53	60	5.8	3.2	7.38	21.0	91.3	37.9	21.8	434.8	3.0	0	38
36.3	13200	69/26/5	44	11.6	1.58	40	0.9	143	3.8	94	0.9	61	50	5.4	3.1	7.28	21.0	93.8	38.0	17.4	446.7	1.2	0	28
33.2	13600	80/13/7	76	10.6	2.26	65	3.9	134	3.3	122	0.4	42	91	6.1	4.3	7.40	40.0	91.9	41.3	24.8	229.7	3.4	2	50
35.3	12080	62/32/6	79	11.3	0.99	73	3.8	138	4.2	91	2.2	57	46	6.4	4.6	7.05	40.0	84.4	62.5	19.9	211.0	5.7	3	47
37.6	12200	72/23/5	71	12.0	1.53	51	3.9	129	4.2	104	4.9	47	53	7.4	4.3	7.17	40.0	71.9	61.1	23.4	179.8	3.8	3	47
32.6	12160	62/32/6	23	10.4	1.21	46	1.1	133	4.3	98	1.6	50	39	6.2	3.4	7.41	40.0	91.9	42.0	26.0	229.8	5.7	2	27
35.1	12200	62/32/6	26	11.2	1.64	33	0.8	138	3.1	122	1.1	65	59	6.5	3.0	7.20	21.0	94.0	42.0	16.7	447.6	1.5	0	31
32.9	11440	64/32/4	54	10.5	1.53	45	1.2	142	4.0	117	0.7	58	93	6.7	3.7	7.19	21.0	92.4	49.0	19.3	440.0	2.0	0	31
38.8	2240	76/22/2	73	12.4	0.92	62	1.3	138	4.5	99	2.5	63	71	5.0	2.7	7.25	40.0	88.6	47.0	20.5	221.5	4.3	2	27
32.2	3400	62/32/6	43	10.3	1.78	40	1.1	129	3.8	95	1.4	65	66	5.7	3.2	7.36	21.0	92.0	41.0	22.6	438.1	2.4	0	27
35.0	12400	65/28/7	35	11.2	0.77	38	1.1	133	3.4	94	1.3	48	59	6.9	4.1	7.27	21.0	93.2	49.3	22.3	443.8	4.4	2	30
27.5	33,400	64/28/8	59	8.8	0.64	118	3.6	132	4.0	121	2.3	59	77	7.0	4.0	7.29	40.0	92.8	35.0	16.5	231.9	5.6	3	41
31.0	13200	72/23/5	28	9.9	1.33	34	0.9	138	3.3	105	2.6	73	56	6.3	3.3	7.26	40.0	86.9	42.6	19.0	217.3	5.8	2	34
35.7	12200	72/23/5	17	11.4	1.03	58	2.5	135	3.3	196	4.2	107	58	6.4	3.0	7.23	21.0	85.5	49.3	20.7	407.1	5.2	3	38
35.7	12200	64/28/8	16	11.4	2.11	45	0.7	132	4.4	97	0.7	33	67	6.0	3.3	7.33	21.0	92.4	42.0	21.3	440.0	1.9	0	26
32.6	14000	61/32/7	37	10.4	1.74	68	1.7	136	3.4	104	3.0	62	65	5.5	2.4	7.06	21.0	94.1	53.0	17.1	448.0	5.5	1	33
32.2	14300	69/23/8	76	10.3	1.49	123	3.6	142	3.5	142	3.1	63	78	6.6	4.9	7.30	40.0	84.0	58.1	13.1	210.0	5.5	3	44
30.7	1800	81/13/6	52	9.8	1.79	54	0.8	141	4.2	112	0.9	78	48	6.6	3.1	7.39	21.0	95.2	40.8	24.2	453.3	1.8	0	25
34.9	13600	68/26/6	62	11.1	0.94	59	1.9	133	4.0	106	2.4	41	81	5.7	2.4	7.39	40.0	86.9	41.9	24.7	217.2	5.6	2	42
31.3	2700	65/28/7	52	10.0	0.78	87	2.5	130	4.7	119	3.2	41	44	6.1	3.1	7.42	21.0	93.4	40.5	25.8	444.8	4.9	3	37

l	26.9	3600	64/28/8	37	8.6	2.64	97	3.0	133	3.1	90	0.6	66	38	8.0	4.6	7.05	21.0	91.2	44.0	14.1	434.5	4.7	2	46
	34.9	13200	69/23/8	47	11.2	0.97	61	1.1	132	3.3	172	1.9	77	76	6.8	3.5	7.16	40.0	79.1	40.5	15.3	197.8	6.0	4	36
-	30.7	12200	74/22/4	73	9.8	1.84	33	0.8	141	4.4	88	1.0	42	99	6.5	3.5	7.34	21.0	92.2	40.7	21.2	439.0	2.0	0	25
	27.5	12120	74/22/4	42	8.8	0.88	48	1.2	128	4.7	102	1.8	71	99	5.8	2.9	7.19	21.0	93.5	53.8	21.2	445.1	6.4	2	29
-	38.8	16000	66/28/6	39	12.4	1.67	41	0.9	145	4.5	92	0.9	46	56	5.6	3.0	7.24	21.0	87.6	53.2	22.9	417.3	2.8	0	35
	26.9	12120	72/23/5	23	8.6	1.24	41	0.7	146	4.2	120	1.1	66	56	6.8	3.7	7.28	21.0	86.8	45.0	20.9	413.1	1.3	0	35
	35.2	11440	74/22/4	20	11.2	0.90	38	1.1	143	3.0	101	0.7	45	90	6.2	3.8	7.37	21.0	92.5	38.0	21.4	440.4	2.0	0	26
	26.9	11400	70/26/4	41	8.6	0.89	52	1.3	146	3.8	86	2.3	67	48	6.0	2.6	7.12	40.0	84.8	43.0	15.1	212.1	5.9	2	36
	35.1	12360	68/26/6	25	11.2	1.65	37	1.0	130	4.5	93	1.1	67	93	5.9	2.9	7.31	21.0	90.6	38.9	19.1	431.5	3.9	0	30
	35.0	12800	62/32/6	38	11.2	1.98	49	0.9	139	3.5	70	0.9	61	40	5.5	2.0	7.20	21.0	91.3	63.1	16.2	435.0	3.6	0	39
	35.1	11400	61/35/4	21	11.2	1.86	70	1.5	138	2.8	91	1.5	64	62	8.3	4.9	7.12	40.0	93.6	46.3	16.3	234.0	6.6	2	35
	38.5	12520	62/32/6	19	12.3	1.46	74	1.8	129	3.4	117	0.6	71	42	6.9	4.5	7.42	40.0	94.6	40.6	25.9	236.6	4.9	2	35
	36.3	12400	79/15/6	13	11.6	1.60	36	0.9	146	4.5	119	1.0	63	62	6.9	3.4	7.30	21.0	70.4	51.9	24.7	335.5	4.1	0	33
	32.6	16600	74/22/4	19	10.4	1.40	33	0.8	132	3.0	104	0.9	33	73	7.0	4.4	7.39	21.0	86.2	34.0	19.9	410.3	1.7	0	33
	27.5	12160	69/26/5	41	8.8	1.11	62	2.3	142	4.2	134	0.8	42	37	7.6	3.9	7.33	21.0	89.7	47.5	24.4	427.1	3.1	0	37
	35.0	12128	62/32/6	34	11.2	1.91	59	1.0	145	3.4	212	1.5	83	98	7.6	4.7	7.20	21.0	82.2	58.1	23.4	391.4	5.2	2	32
	22.5	12160	64/28/8	73	7.2	1.17	87	2.7	137	4.4	113	5.2	83	58	5.1	2.4	7.43	40.0	73.3	36.7	23.6	183.1	4.7	3	34
	37.6	11720	72/23/5	38	12.0	0.78	59	0.8	136	3.1	108	1.1	32	47	6.7	3.2	7.35	21.0	92.0	38.0	20.4	438.1	3.2	0	26
	27.9	12480	70/27/3	53	8.9	2.53	39	0.9	145	4.0	90	0.9	56	88	7.1	3.4	7.39	21.0	95.0	40.0	23.6	452.4	3.6	0	26
	41.3	15600	68/26/6	41	13.2	1.34	40	1.6	130	3.7	208	1.1	49	40	6.2	3.1	7.22	21.0	88.6	48.9	20.2	422.1	4.4	2	38
	38.8	13200	59/31/10	52	12.4	1.02	162	4.0	145	3.8	147	1.6	79	67	5.8	2.6	7.11	40.0	95.4	44.1	15.4	238.5	5.6	3	51
	38.8	13600	74/22/4	40	12.4	2.73	54	0.9	137	3.6	111	0.9	61	77	5.9	3.4	7.40	21.0	90.5	41.4	24.6	431.0	1.8	0	25
	32.2	3300	70/27/3	46	10.3	2.19	89	2.4	133	3.1	287	3.0	76	77	6.0	2.7	7.19	40.0	79.1	50.7	19.9	197.7	6.2	2	45
	27.5	14400	62/32/6	66	8.8	2.73	42	2.1	146	3.1	190	1.1	44	73	6.0	3.8	7.38	21.0	91.3	44.0	25.4	435.0	2.9	0	32
	34.9	13000	70/27/3	38	11.2	2.20	72	1.4	141	3.0	100	0.9	71	48	6.9	3.1	7.54	21.0	90.3	32.0	29.0	430.1	5.6	1	37
	30.7	14200	76/22/2	37	9.8	2.21	46	2.9	138	3.1	74	2.0	66	80	5.2	2.3	7.36	40.0	77.7	37.8	20.7	194.2	4.4	2	53
ſ	33.9	12400	66/28/7	30	10.8	1.64	35	1.0	129	4.2	102	1.2	44	69	6.3	3.9	7.34	21.0	94.1	48.9	25.3	448.0	3.0	0	26

40.1	14400	70/27/3	16	12.8	1.14	39	0.6	146	2.9	114	0.8	61	41	5.9	3.1	7.36	21.0	89.6	42.0	23.0	426.7	3.7	0	31
41.3	12200	72/23/5	21	13.2	2.34	36	1.0	144	3.7	76	3.1	143	157	5.6	2.8	7.27	40.0	87.0	44.4	20.2	217.4	6.6	3	28
33.2	11520	64/28/8	27	10.6	1.94	56	1.6	130	4.6	240	1.0	53	43	6.2	2.8	7.10	40.0	82.1	56.4	19.2	205.4	4.2	2	33
32.6	12152	61/32/7	21	10.4	1.74	40	1.0	130	3.6	104	1.1	40	74	6.8	3.6	7.33	21.0	91.4	45.6	23.4	435.2	2.4	0	28
35.1	12040	69/23/8	15	11.2	1.08	35	0.9	136	4.5	148	0.6	31	41	5.7	3.0	6.91	21.0	95.9	57.4	15.5	456.5	3.1	0	29
30.7	12600	80/13/7	26	9.8	1.59	46	0.7	141	4.3	106	0.5	58	43	6.5	3.5	7.02	21.0	84.6	43.0	13.2	402.8	2.0	0	29
32.6	12120	74/22/4	56	10.4	0.95	47	1.4	136	4.6	101	2.0	85	40	7.2	3.3	7.36	21.0	94.0	32.4	17.7	447.6	5.0	1	27
30.7	12104	66/23/11	39	9.8	1.88	58	2.9	143	3.6	101	0.8	35	76	7.5	4.2	7.21	21.0	90.8	48.9	20.0	432.4	3.1	0	30
35.1	12200	65/28/7	29	11.2	2.41	76	3.4	132	3.7	96	0.7	39	93	7.3	3.8	7.24	21.0	85.7	43.2	18.4	408.0	2.6	0	32
31.3	12400	82/15/3	49	10.0	0.98	39	0.9	129	3.0	159	1.2	45	73	6.2	3.5	7.16	21.0	87.0	44.6	16.7	414.1	4.0	0	35
31.3	14300	74/22/4	65	10.0	0.88	95	2.3	146	3.0	84	1.5	71	71	6.2	3.7	7.21	40.0	70.1	51.0	20.8	175.3	6.7	3	42
38.5	2700	74/22/4	37	12.3	2.48	56	0.9	138	4.6	153	1.3	61	80	7.3	3.3	7.30	21.0	91.0	42.7	20.7	433.2	2.9	0	33
40.7	11520	70/27/3	51	13.0	1.22	31	0.8	137	3.6	102	1.0	36	75	6.2	3.9	7.30	21.0	92.3	39.7	19.0	439.5	3.2	0	25
35.1	13800	66/28/6	56	11.2	1.41	43	1.1	146	3.7	171	0.9	45	97	7.1	3.0	7.35	21.0	91.0	43.0	22.9	433.3	3.1	0	33
35.2	12040	74/22/4	41	11.3	1.29	35	0.9	143	3.9	117	1.6	79	96	7.0	4.3	7.40	40.0	88.0	28.0	17.0	219.9	4.9	2	24
26.0	2700	69/26/5	28	8.3	0.96	104	2.3	135	3.5	143	3.4	96	43	5.0	2.2	7.36	40.0	84.1	41.7	22.8	210.2	5.0	4	44
35.1	12600	61/32/7	20	11.2	1.36	55	1.9	140	4.0	145	2.2	71	76	7.2	3.9	6.97	40.0	85.0	47.6	13.8	212.5	6.1	2	39
35.0	11440	66/28/7	28	11.2	1.45	91	2.3	139	4.6	94	0.7	56	54	6.2	2.5	7.23	21.0	90.0	36.2	15.3	428.6	5.8	2	36
40.7	12800	66/28/6	14	13.0	1.43	41	1.0	144	4.1	112	1.3	50	64	6.1	3.4	7.32	21.0	94.0	39.0	19.6	447.6	3.1	0	29
36.3	12176	74/22/4	44	11.6	2.48	31	0.9	138	3.3	150	0.7	52	94	6.9	4.3	7.42	21.0	86.4	39.3	24.5	411.4	3.4	0	31
35.0	12200	64/32/4	72	11.2	2.35	40	1.2	136	4.0	117	0.9	42	73	6.4	3.1	7.47	21.0	83.3	35.2	25.5	396.6	2.5	0	24
41.3	13800	82/15/3	16	13.2	1.43	51	1.7	145	4.1	102	2.6	88	39	6.2	2.8	7.22	40.0	83.1	41.0	16.9	207.8	4.2	1	34
40.1	12320	74/22/4	14	12.8	1.73	31	0.7	145	4.2	92	1.3	41	44	6.7	3.2	7.36	21.0	91.4	42.9	23.2	435.1	2.5	0	27
35.7	13000	74/22/4	34	11.4	1.27	43	0.9	134	2.9	117	2.9	53	63	6.1	3.6	7.41	21.0	91.3	40.5	25.2	434.8	1.9	0	28
35.2	11920	74/22/4	43	11.2	0.87	71	1.7	128	4.7	119	2.3	78	84	7.0	3.5	7.18	40.0	85.3	54.1	21.1	213.3	5.3	3	33
31.3	11680	79/15/6	39	10.0	1.91	77	1.5	137	4.2	88	3.7	84	44	5.6	2.3	7.33	40.0	85.4	32.0	16.3	213.6	5.6	3	34
33.9	3100	61/35/4	27	10.8	1.47	41	1.1	146	3.1	175	3.2	73	68	6.1	3.2	7.04	40.0	86.8	41.0	13.0	216.9	4.6	4	51

	34.9	12136	74/22/4	33	11.2	0.88	93	2.4	128	3.6	124	1.5	82	69	6.9	3.7	7.50	40.0	82.2	27.9	22.5	205.4	4.1	2	37
ſ	35.2	2120	64/28/8	30	11.2	0.92	82	2.2	137	3.3	90	1.7	47	78	6.1	3.3	7.36	21.0	91.9	32.0	17.3	437.6	6.0	3	31
ſ	26.6	14200	64/28/8	61	8.5	1.29	54	1.3	129	4.0	109	3.0	70	38	6.9	3.0	7.39	40.0	87.7	37.5	22.0	219.1	4.4	2	36
	31.3	12440	69/23/8	20	10.0	2.67	52	2.0	144	3.4	120	0.8	60	53	6.0	3.7	7.26	40.0	85.9	47.4	21.0	214.7	4.5	2	35
	40.7	12192	65/28/7	14	13.0	0.90	54	2.0	143	4.3	91	2.3	72	35	5.4	2.6	7.49	40.0	89.4	30.1	23.1	223.5	5.1	3	36
ſ	34.9	12144	82/15/3	41	11.2	2.86	39	1.1	130	3.8	121	1.1	32	59	6.3	3.2	7.43	21.0	95.1	33.2	21.6	452.7	1.7	0	35
ſ	40.7	14,300	81/17/2	13	13.0	0.36	42	1.2	126	3.4	109	1.5	55	64	5.8	2.9	7.29	40.0	88.0	30.0	14.0	220.0	4.9	3	37

		Culture/	Sensitivity		-	/MF	AT	Dengue	Test	ected	bid OS		agnosis	ome
Blood	Urine	CSF	Mound	Sputum	Others	MP	SM	IgM/IgG	Wida	OS inf	Comor	Category	Complete Di	Outo
47	48	49	50	51	52	53	54	55	56	57	58	59	60	61
NG	E.Coli	×	×	×	×	ve	ve	ve	ve	GUT	CNS	SIRS/Sepsis	CIDP/	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	GIT	HB	SevSepsis	DCLD/PHT/SBP/HE	Dis
CONS	×	×	×	NG	×	ve	ve	ve	ve	RS	CNS	SIRS/Sepsis	Seiz dis	Dis
P.Vulg	P.Vulg	×	×	×	×	ve	ve	ve	ve	GUT	MOS	SS	DMT2/SHT/CVA/UTI	Dis
NG	×	×	×	×	×	ve	ve	ve	+ ve	TropInf	Nil	SIRS/Sepsis	Ent Fev/	Dis
NG	×	×	×	Comm	×	ve	ve	ve	ve	RS	Nil	SIRS/Sepsis	BrPn	Dis
CONS	×	×	×	×	×	ve	ve	ve	+ ve	TropInf	Nil	SIRS/Sepsis	Ent Fev/	Dis
NG	×	×	×	×	×	+ ve	ve	ve	ve	TropInf	Nil	SIRS/Sepsis	Malaria	Dis
NG	×	×	×	NG	×	ve	ve	ve	ve	RS	CNS	SIRS/Sepsis	HyKPP/AspPn	Dis
NG	×	×	×	NG	×	ve	ve	ve	ve	RS	CNS	SevSepsis	Psychosis/BA	Dis
NG	×	NG	×	×	×	+ve	ve	ve	ve	TropInf	MOS	SevSepsis	DMT2/Cbl Mal	Dis
NG	×	×	×	NG	×	ve	ve	ve	ve	RS	Hem	SevSepsis	CML/BrPn	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	GIT	Nil	SIRS/Sepsis	AGE/	Dis
NG	×	×	×	×	NG	ve	ve	ve	ve	GIT	HB	SS	DCLD/PHT/SBP	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	RS	MOS	SevSepsis	BrPn/SHT	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	RS	CNS	SIRS/Sepsis	CVA/SeizDis	Dis
NG	×	×	×	NG	×	ve	ve	ve	ve	RS	CNS	SevSepsis	Young CVA/AspPn	Dis
NG	×	×	×	×	×	+ve	ve	ve	ve	TropInf	Nil	SIRS/Sepsis	Malaria	Dis

S.Aures	×	×	×	NG	×	ve	ve	ve	ve	RS	MOS	SIRS/Sepsis	SHT/RA/ILD	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	RS	MOS	SevSepsis	COPD/SHT/CHF	Death
CONS	×	×	×	Comm	×	ve	ve	ve	ve	RS	RS	SIRS/Sepsis	BA/SHT	Dis
NG	×	×	×	×	×	ve	₊ve	ve	ve	TropInf	Nil	SIRS/Sepsis	lepto	Dis
NG	NG	×	×	×	×	ve	ve	ve	ve	GUT	MOS	SIRS/Sepsis	DMT2/PyNeph	Dis
NG	×	×	×	×	×	ve	₊ve	ve	₊ve	TropInf	Nil	SevSepsis	Lepto/EntFev/	Dis
NG	×	×	×	×	NG	ve	ve	ve	ve	GIT	HB	SIRS/Sepsis	DCLD/PHT/SBP	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SS	DMT2/SHT/CKD	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	Hem	SS	HELLP Synd	Death
NG	×	×	×	×	×	ve	ve	₊ve	ve	TropInf	Nil	SevSepsis	dengue	Dis
NG	×	×	×	NG	×	ve	ve	ve	ve	RS	RS	SIRS/Sepsis	ChrBritis/	Dis
NG	×	×	×	CONS	×	ve	ve	ve	ve	RS	CNS	SS	Seiz;AspPn;	Dis
NG	×	×	×	M.Plasma	×	ve	ve	ve	ve	RS	MOS	SIRS/Sepsis	Psychosis/PTb Seq	Dis
CitroB	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SIRS/Sepsis	DMT2/SHT/CKD	Dis
NG	NG	×	×	×	×	ve	ve	ve	ve	GUT	MOS	SevSepsis	DMT2/SHT/UTI	Dis
CONS	CONS	×	×	×	×	ve	ve	ve	ve	GUT	CVS	SIRS/Sepsis	PpCMP/CHF	Dis
NG	NG	×	×	×	×	ve	ve	ve	ve	GUT	MOS	SS	DMT1;UTI	Death
S.Aureus	×	×	×	S.Aureus	×	ve	ve	ve	ve	RS	RS	SevSepsis	Bronchiectasis	Dis
S.Typhi	×	×	×	×	×	ve	ve	ve	₊ve	TropInf	Nil	SIRS/Sepsis	Ent Fev/	Dis
MRSA	×	NG	MRSA	×	×	ve	ve	ve	ve	CNS	CNS	SevSepsis	CSOM;AcMenEn	Dis
NG	E.Coli	×	×	×	×	ve	ve	ve	ve	GUT	MOS	SIRS/Sepsis	DMT1;UTI	Dis
Klebs	×	×	×	Klebs	×	ve	ve	ve	ve	RS	MOS	SevSepsis	SHT/CAD/BrPn	Death
NG	×	×	×	×	×	ve	ve	ve	+ve	TropInf	Nil	SIRS/Sepsis	Ent Fev/	Dis
NG	×	×	×	×	×	ve	ve	ve	+ve	TropInf	Nil	SIRS/Sepsis	Ent Fev/	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SIRS/Sepsis	SHT;CVA;	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	CVS	SevSepsis	RHD/CHF	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	RS	CNS	SevSepsis	CVA;AspPn	Dis

NG	×	×	×	NG	×	ve	ve	ve	ve	RS	RS	SIRS/Sepsis	Tb Seq/Cavity	Dis
NG	×	×	×	×	×	₊ve	ve	ve	ve	TropInf	Nil	SIRS/Sepsis	Malaria	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SS	DMT2;DKA	Dis
NG	×	×	×	S.Pneum	×	ve	ve	ve	ve	RS	Nil	SevSepsis	CABPn	Dis
NG	NG	×	×	×	×	ve	ve	ve	ve	GUT	Nil	SIRS/Sepsis	UTI	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SIRS/Sepsis	DMT2/SHT/CVA	Death
NG	NG	×	×	NG	×	ve	ve	ve	ve	RS	MOS	SIRS/Sepsis	SHT/CAD/CKD	Death
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	CVS	SIRS/Sepsis	CHD/ASD	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SS	SHT;CVA;	Death
NG	×	×	×	NG	×	ve	ve	ve	ve	RS	Nil	SevSepsis	BrPn	Dis
NG	×	NG	×	×	×	ve	ve	ve	ve	CNS	Nil	SevSepsis	AcMenEnc	Dis
NG	×	×	×	×	×	₊ve	ve	ve	ve	TropInf	Nil	SevSepsis	Cbl Malaria	Dis
E.coli	NG	×	×	×	×	ve	ve	ve	ve	GUT	Nil	SIRS/Sepsis	PID/UTI	Dis
NG	×	×	×	×	×	ve	ve	ve	₊ve	TropInf	Nil	SIRS/Sepsis	Ent Fev/	Dis
NG	×	×	×	×	×	ve	ve	₊ve	ve	TropInf	Nil	SevSepsis	dengue	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	GIT	SIRS/Sepsis	Gastritis	Dis
NG	NG	×	×	NG	×	ve	₊ve	ve	₊ve	TropInf	MOS	SevSepsis	SHT/Lepto/Ent Fev	Dis
EntCoccos	×	×	×	×	×	ve	ve	ve	ve	Occult	Hem	SS	CML;	Death
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	CVS	SS	RHD/CHF	Dis
Klebs	NG	×	×	NG	×	ve	ve	ve	ve	RS	MOS	SevSepsis	DMT2;SHT;CAD;CHF	Death
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	CVS	SIRS/Sepsis	SHT/CAD	Dis
S.Aureus	×	×	×	×	NG	ve	ve	ve	ve	GIT	GUT	SevSepsis	Nephrotic synd	Dis
NG	E.Coli	×	×	×	×	ve	ve	ve	ve	GUT	CNS	SS	CVA/UTI	Dis
NG	NG	×	×	×	×	ve	ve	ve	ve	GUT	MOS	SIRS/Sepsis	SLE/Lupus nephritis	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SS	SHT;CVA;	Dis
CONS	×	×	NG	×	×	ve	ve	ve	ve	Skin	MOS	SS	SHT;CVA;BedSore	Dis
NG	NG	×	×	×	NG	ve	ve	ve	ve	Occult	MOS	SevSepsis	SLE/CKD/Vol OvLoad	Death

NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SevSepsis	DMT2;SHT;CAD;CHF	Death
Klebs	×	×	×	×	×	ve	ve	ve	ve	Occult	Hem	SIRS/Sepsis	F XIII def/Hematoma	Dis
NG	×	×	×	PTb/Comm	×	ve	ve	ve	ve	RS	RS	SevSepsis	Active PTb/FibCav	Dis
Klebs	NG	NG	×	NG	×	ve	ve	ve	ve	Occult	MOS	SIRS/Sepsis	SHT/SeizDis/	Dis
Acineto	×	NG	×	×	×	ve	ve	ve	ve	CNS	RS	SIRS/Sepsis	COPD/	Dis
CONS	×	×	×	×	×	ve	ve	ve	ve	TropInf	Nil	SIRS/Sepsis	dengue	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	HB	SevSepsis	DCLD/PHT/SBP	Dis
S.Aureus	×	×	×	×	NG	ve	ve	ve	ve	GIT	HB	SIRS/Sepsis	EHPVO/PHT/SBP	Dis
E.Coli	×	×	×	×	×	ve	ve	ve	ve	GIT	HB	SIRS/Sepsis	DCLD/PHT/SBP	Death
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	CVS	SevSepsis	RHD/CHF	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Skin	CNS	SevSepsis	GBS/BedSore	Dis
CONS	×	NG	×	×	×	ve	ve	ve	ve	CNS	Nil	SIRS/Sepsis	AcMenEnc	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	RS	SIRS/Sepsis	COPD	Dis
NG	NG	×	×	×	×	ve	ve	ve	ve	GUT	Malig	SIRS/Sepsis	CaCX/UTI	death
S.Aureus	×	×	×	×	×	ve	ve	ve	ve	Skin	MOS	SevSepsis	DMT2;Cellulitis	Dis
E.Coli	E.Coli	×	×	×	×	ve	ve	ve	ve	GUT	CNS	SS	CVA;UTI	Dis
NG	×	×	×	×	×	ve	ve	₊ve	ve	TropInf	Nil	SIRS/Sepsis	dengue	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	Hem	SIRS/Sepsis	Thallesemia	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SS	DMT2;SHT;CAD;CHF	Dis
S.Aureus	NG	×	×	NG	×	ve	ve	ve	ve	Occult	MOS	SS	DMT2;SHT;CKD	Death
NG	×	×	×	×	×	ve	₊ve	ve	ve	TropInf	Nil	SIRS/Sepsis	Lepto	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SevSepsis	DMT2;DKA;AspPn;	Death
Acineto	NG	×	×	NG	×	ve	ve	ve	ve	Occult	MOS	SIRS/Sepsis	DMT2;CAD	Dis
CONS	×	×	×	Klebs	×	ve	ve	ve	ve	RS	Malig	SevSepsis	CaEso/Collapse lung	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	GIT	HB	SS	DCLD/PHT/SBP	Dis
NG	×	×	×	Klebs	×	ve	ve	ve	ve	RS	Nil	SIRS/Sepsis	CABPn	Dis
S.Typhi	×	×	×	×	×	ve	ve	ve	₊ve	TropInf	Nil	SIRS/Sepsis	Ent Fev/	Dis

E.coli	×	×	E.Coli	×	×	ve	ve	ve	ve	HB	Nil	SS	Liver Abscess	Dis
Pseudo	×	×	×	Pseudo	×	ve	ve	ve	ve	RS	MOS	SevSepsis	DMT2;BrPn	Death
NG	×	×	×	NG	×	ve	ve	ve	ve	RS	RS	SIRS/Sepsis	Tb Seq/Cavity	Dis
S.Aureus	NG	×	×	NG	×	ve	ve	ve	ve	Occult	MOS	SIRS/Sepsis	DMT2/DKA/	Death
S.Aureus	×	×	×	S.Aureus	×	ve	ve	ve	ve	RS	Nil	SIRS/Sepsis	CABPn	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Skin	MOS	SevSepsis	SHT/Cellulitis leg	Dis
NG	×	×	×	×	×	₊ve	ve	ve	ve	TropInf	Nil	SIRS/Sepsis	Malaria	Dis
Acineto	×	×	×	×	×	ve	ve	ve	₊ve	TropInf	Nil	SIRS/Sepsis	Ent Fev/	Dis
NG	NG	×	×	NG	×	ve	ve	ve	ve	RS	MOS	SIRS/Sepsis	COPD/DMT2	Dis
NG	×	×	×	×	NG	ve	ve	ve	ve	GIT	HB	SevSepsis	DCLD/PHT/SBP	Dis
NG	×	×	×	×	NG	ve	ve	ve	ve	GIT	HB	SIRS/Sepsis	CLD/SHT/SBP	Dis
NG	×	×	×	×	×	₊ve	ve	ve	ve	TropInf	Nil	SIRS/Sepsis	Malaria	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SIRS/Sepsis	DMT2/DCMP/CHF	Death
NG	×	×	×	NG	×	ve	ve	ve	ve	RS	Nil	SevSepsis	BrPn	Dis
NG	×	×	×	×	NG	ve	ve	ve	ve	GIT	MOS	SS	DMT2;DCLD/PHT/UGIB;	Death
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SevSepsis	DMT2;SHT;CAD;CHF	Death
Pseudo	Candida	×	×	×	×	ve	ve	ve	ve	GUT	MOS	SevSepsis	Tb Seq/CAD/UTI	Dis
CONS	×	×	×	×	×	₊ve	ve	ve	ve	TropInf	Nil	SIRS/Sepsis	Malaria	Dis
Acineto	×	×	×	NG	×	ve	ve	ve	ve	RS	MOS	SIRS/Sepsis	BA/DMT2	Dis
S.Aureus	×	×	×	CONS	×	ve	ve	ve	ve	RS	Nil	SIRS/Sepsis	CABrPn	Dis
CONS	×	×	×	Comm	×	ve	ve	ve	ve	RS	Nil	SevSepsis	BrPn	Dis
NG	×	×	×	×	×	₊ve	ve	ve	ve	TropInf	Nil	SIRS/Sepsis	Malaria	Dis
NG	×	×	×	×	×	ve	₊ve	ve	ve	TropInf	Nil	SIRS/Sepsis	Lepto	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	CVS	SS	RHD/CHF	Dis
NG	×	×	×	×	NG	ve	ve	ve	ve	GIT	HB	SevSepsis	DCLD/PHT/UGIB	Dis
NG	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SS	DMT2/SHT/CAD/CVA	Death
NG	×	×	Pseudo	×	×	ve	ve	ve	ve	RS	Nil	SevSepsis	Empyema/	Dis

NG	×	×	×	×	×	ve	ve	ve	ve	GIT	Nil	SS	AGE/	Dis
NG	×	NG	×	×	×	₊ve	ve	ve	ve	TropInf	Nil	SS	Cbl Malaria	Dis
Pseudo	×	×	×	NG	×	ve	ve	ve	ve	RS	MOS	SS	RHD/Embolic CVA	Dis
Pseudo/ S.Aureus	×	×	×	NG	×	ve	ve	ve	ve	RS	ImmSup	SevSepsis	HIV/BrEctasis	Dis
CONS	×	×	×	×	×	ve	ve	ve	ve	Occult	MOS	SIRS/Sepsis	SHT;CVA;	Dis
NG	×	×	×	Klebs	×	ve	ve	ve	ve	RS	MOS	SevSepsis	SHT;CAD;BrPn	Dis

BIBLIOGRAPHY

- 1. Schottmueller und Behandlung der Sepsis. Inn Med 1914;31:257-280.
- 2. http:// emedicine. medscape.com / article/169640.
- Angus DC, Linde-Zwirble WT, Lidicker J, Clermont G, Carcillo J, Pinsky MR. Epidemiology of severe sepsis in the United States: analysis of incidence, outcome, and associated costs of care. *Crit Care Med* 2001;29:1303–1310.
- 4. Vincent JL, Sakr Y, Sprung CL, Ranieri VM, Reinhart K, Gerlach H, Moreno R, Carlet J, Le Gall JR, Payen D. Sepsis in European Intensive care Units: Results of the SOAP study. *Crit Care Med.*
- 5. S.Todi, S.Chatterjee, S.Sahu, and M.Bhattacharyya. Epidemiology of severe sepsis in India: an update: *Crit Care*. 2010; 14(Suppl 1): P382.
- Verbrugh HA, Mintjes-De Groot AJ, Broers DA: Bacteremia in 2 general hospitals: the tip of the iceberg of hospital infections. Ned Tijdschr Geneeskd 1986, 130:441-445.
- Kieft H, Hoepelman AIM, Zhou W, Rozenberg-Arska M, Struyvenberg A, Verhoef J: The sepsis syndrome in a Dutch University Hospital. *Arch Intern Med* 1993, 153:2241-2247.
- RIVM: http://www.rivm.nl website Nationaal Kompas Volksgezondheid, versie 1.4; Sepsis (National Compass of Public Health, version 1.4; Sepsis.
- 9. Aukje van Gestel Prevalence and incidence of severe sepsis in Dutch intensive care units. *Critical Care* 2004, 8:R153-R162.
- 10. Failure Ronco C, Bellomo R, Kellum JA Classification, Incidence, and Outcomes of Sepsis and Multiple Organ: *Acute Kidney Injury*. Contrib Nephrol. Basel, Karger, 2007, vol 156, pp 64–74.

- 11. Brun-Buisson C, Doyon F, Carlet J, Dellamonica P, Gouin F, Lepoutre A, Mercier JC, Offenstadt G, Régnier B. Incidence, risk factors, and outcome of severe sepsis and septic shock in adults. A multicenter prospective study in intensive care units. French ICU Group for Severe Sepsis ; JAMA. 1995 Sep 27;274(12):968-74.
- 12. Finfer S, Bellomo R, Lipman J, French C, Dobb G, Myburgh JAdultpopulation incidence of severe sepsis in Australian and New Zealand intensive care units.. *Intensive Care Med*. 2004 Apr;30(4): 589-96.
- 13.Ponce de León SP, et al.Prevalence of infections in intensive care units in Mexico: a multicenter study. *Crit Care Med*. 2000 May; 28(5): 1316-21.
- 14. Derek C. Angus, MD, MPH, FCCM; Walter T. Linde-Zwirble; Jeffrey Lidicker, MA; Gilles Clermont, MD; Joseph Carcillo, MD; Michael R. Pinsky, MD, FCCM. Epidemiology of severe sepsis in the United States: Analysis of incidence, outcome, and associated costs of care.
- 15.Le Gall JR, Alberti C, Brun Buisson C;Epidemiology of infection and sepsis in intensive care unit patients; *Bull Acad Natl Med.* 2004;188(7):1115-25; discussion 1125-6.
- 16.Ali H Al Khafaji, Multisystem Organ Failure of Sepsis; http:// emedicine.medscape.com/article/169640.
- 17.Brun-Buisson C. The epidemiology of the systemic inflammatory response. *Intensive Care Med.* 2000;26 Suppl 1:S64-74.
- 18. Alberti C, Brun-Buisson C, Burchardi H, Martin C, Goodman S, Artigas A, Sicignano A, Palazzo M, Moreno R, Boulmé R, Lepage E, Le Gall R. Epidemiology of sepsis and infection in ICU patients from an international multicentre cohort study. *Intensive Care Med.* 2002 Feb;28(2):108-21.

- 19. Silva E, Pedro Mde A, et al Brazilian Sepsis Epidemiological Study (BASES study). ; *Crit Care*. 2004 Aug;8(4):R251-60.
- 20. Schoenberg MH, Weiss M, Radermacher P. Outcome of patients with sepsis and septic shock after ICU treatment. *Langenbecks Arch Surg.* 1998 Mar;383(1):44-821.
- 21.Padkin A, Goldfrad C, Brady AR, Young D, Black N, Rowan K Epidemiology of severe sepsis occurring in the first 24 hrs in intensive care units in England, Wales, and Northern Ireland. *Crit Care Med.* 2003 Sep;31(9):2332-8.
- 22. Martin GS, Mannino DM, Eaton S, Moss M The epidemiology of sepsis in the United States from 1979 through 2000.. *N Engl J Med*. 2003 Apr 17;348(16):1546-54.
- 23. Danai, Pajman A. MD; Sinha, Sumita MD; Moss, Marc MD; Haber, Michael J. PhD; Martin, Greg S. MSc, MD Seasonal variation in the epidemiology of sepsis *JAMA* Vol. 278 No. 3, July 16, 1997.
- 24. Martin GS, Mannino DM, Eaton S, Moss M. The epidemiology of sepsis in the United States from 1979 through 2000. N Engl J Med 2003;348:1546–1554.
- 25. Finfer S, Bellomo R, Lipman J, French C, Dobb G, Myburgh J. Adultpopulation incidence of severe sepsis in Australian and New Zealand intensive care units. *Intensive Care Med.* 2004 Apr;30(4): 589-96.
- 26. Alberti C, Brun-Buisson C, Burchardi H, Martin C, Goodman S, Artigas A, Sicignano A, Palazzo M, Moreno R, Boulmé R, Lepage E, Le Gall R Epidemiology of sepsis and infection in ICU patients from an international multicentre cohort study. *Intensive Care Med*. 2002 Feb;28(2):108-21.

- 27. Schoenberg MH, Weiss M, Radermacher P. Outcome of patients with sepsis and septic shock after ICU treatment. *Langenbecks Arch Surg.* 1998 Mar;383(1):44-8.
- 28. Ponce de León SP, et al. Prevalence of infections in intensive care units in Mexico: a multicenter study. *Crit Care Med.* 2000 May; 28(5): 1316-21.
- 29.Greg S. Martin, M.D., David M. Mannino, M.D., Stephanie Eaton, M.D,and Marc Moss, M.D. The Epidemiology of Sepsis in the United States from 1979 through 2000; *N Engl J Med* 2003;348:1546-54.
- 30. Brun-Buisson C, Doyon F, Carlet J, Dellamonica P, Gouin F, Lepoutre A, Mercier JC, Offenstadt G, Régnier B. Incidence, risk factors, and outcome of severe sepsis and septic shock in adults. A multicenter prospective study in intensive care units. French ICU Group for Severe Sepsis ; JAMA. 1995 Sep 27;274(12):968-74.
- 31. Author: R Phillip Dellinger, MD, Septic Shock http://emedicine.medscape.com/article/168402-overview.
- 32. Angus DC, Linde-Zwirble WT, Lidicker J, Clermont G, Carcillo J, Pinsky MR: Epidemiology of severe sepsis in the United States: Analysis of incidence. Outcome and associated costs of care. *Crit Care Med* 2001, 29:1303-1310.
- 33. Alberti C, Brun-Buisson C, Burchardi H, Martin C, Goodman S, Artigas A, Sicignano A, Palazzo M, Moreno R, Boulme R, Lepage E, Le Gall R: Epidemiology of sepsis and infection in ICU patients from an international multicentre cohort study. *Intensive Care Med* 2002, 28: 108-121.
- 34. Brun-Buisson C, Doyon F, Carlet J, Dellamonica P, Gouin F, Lepoutre A, Mercier JC, Offenstadt G, Régnier B. Incidence, risk factors, and

outcome of severe sepsis and septic shock in adults. A multicenter prospective study in intensive care units. French ICU Group for Severe Sepsis. *JAMA*. 1995 Sep 27;274(12):968-74.

- 35. Boussekey N, Cantrel J, et al Epidemiology, prognosis, and evolution of management of septic shock in a French intensive care unit: a five years survey. *Crit Care Res pract*. 2010;2010: 436427
- 36.Brun-Buisson C. The epidemiology of the systemic inflammatory response. *Intensive Care Med.* 2000;26 Suppl 1:S64-74.
- 37.Ponce de León-Rosales SP, Molinar-Ramos F, Domínguez-Cherit G, Rangel-Frausto MS, Vázquez-Ramos VG. Prevalence of infections in intensive care units in Mexico: a multicenter study. *Crit Care Med.* 2000 May;28(5):1316-21.
- 38. ACCP/SCCM Consensus Conference (1992) Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. *Crit Care Med* 20:864–875.
- 39. Opal SM. Concept of PIRO as a new conceptual framework to understand sepsis. *Pediatr Crit Care Med* 2005;6:S55–S60.
- 40. Martin C, Boisson C, Haccoun M, Thomachot L, Mege JL. Patterns of cytokine evolution (tumor necrosis factor-alpha and interleukin-6) after septic shock, hemorrhagic shock, and severe trauma. *Crit Care Med* 1997;25:1771–1773.
- 41.Sama AE, D'Amore J, Chen G, Wang H. Bench to bedside: HMGB1-a novel proinflammatory cytokine and potential therapeutic target for septic patients in the emergency department. *Acad Emerg Med* 2004;11:867–873.

- 42. Bozza FA, Gomes RN, Japiassu AM, Soares M, Castro-Faria-Neto HC, Bozza PT, Bozza MT. Macrophage migration inhibitory factor levels correlate with fatal outcome in sepsis. *Shock* 2004;22:309–313.
- 43. Amaral A, Opal SM, Vincent JL. Coagulation in sepsis. *Intensive Care Med* 2004;30:1032–1040.
- 44. Taveira da Silva AM, Kaulbach HC, Chuidian FS, Lambert DR, Suffredini AF, Danner RL. Brief report: shock and multiple-organ dysfunction after self-administration of Salmonella endotoxin. *N Engl J Med* 1993;328:1457–1460.
- 45.Levin J, et al. Detection of endotoxin in the blood of patients with sepsis due to gran-negative bacteria. *N Engl J Med* 1970;283:1313–1316.
- 46.Bates DW, Parsonnet J, et al. Limulus amebocyte lysate assay for detection of endotoxin in patients with sepsis syndrome. AMCC Sepsis Project Working Group. *Clin Infect Dis* 1998;27:582–591.
- 47. Marshall JC, Walker PM, et al . Measurement of endotoxin activity in critically ill patients using whole blood neutrophil dependent chemiluminescence. *Crit Care* 2002;6:342–348.
- 48.Marshall JC, Foster D, Vincent JL, Cook DJ, Cohen J, Dellinger RP, Opal S, et al. Diagnostic and prognostic implications of endotoxemia in critical illness: results of the MEDIC study. *J Infect Dis* 2004; 190: 527–534.
- 49. Parsons PE, Worthen GS, Moore EE, Tate RM, Henson PM. The association of circulating endotoxin with the development of the adult respiratory distress syndrome. *Am Rev Respir Dis* 1989;140: 294–301.

- 50. Van Deventer SJ, Buller HR, ten Cate JW, Sturk A, Pauw W. Endotoxaemia: an early predictor of septicaemia in febrile patients. *Lancet* 1988;1:605–608.
- 51. Wang JE, Dahle MK, McDonald M, Foster SJ, Aasen AO, Thiemermann C. Peptidoglycan and lipoteichoic acid in gram-positive bacterial sepsis: receptors, signal transduction, biological effects, and synergism. *Shock* 2003;20:402–414.
- 52.Blain CM, Anderson TO, Pietras RJ, Gunnar RM. Immediate hemodynamic effects of gram-negative vs gram-positive bacteremia in man. Arch Intern Med 1970;126:260–265.
- 53. Wilson RF, Sarver EJ, Rizzo J. Hemodynamic changes, treatment, and prognosis in clinical shock. *Arch Surg* 1971;102:21–24.
- 54. Bernard GR, Vincent JL, Laterre PF, LaRosa SP, Dhainaut JF, Lopez-Rodriguez A, Steingrub JS, Garber GE, Helterbrand JD, Ely EW, et al. Efficacy and safety of recombinant human activated protein C for severe sepsis. *N Engl J Med* 2001;344:699–709.
- 55. Volk HD, Reinke P, Krausch D, Zuckermann H, Asadullah K, Müller JM, Döcke WD, Kox WJ. Monocyte deactivation: rationale for a new therapeutic strategy in sepsis. *Intensive Care Med* 1996;22:S474–S481.
- 56. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. *Crit Care Med* 1985;13: 818–829.
- 57.Udhoji VN, Wel MH. Hemodynamic and metabolic studies on shock associated with bacteremia. *Ann Intern Med* 1965;62:966–978.
- 58. Abraham E, Shoemaker WC, Cheng PH. Cardio respiratory responses to fluid administration in peritonitis. *Crit Care Med* 1984;12:664–668.
- 59. Groeneveld AB, Bronsveld W, Thijs LG. Hemodynamic determinants of mortality in human septic shock. *Surgery* 1986;99:140–152.
- 60. MacCannell KL, McNay JL, Meyer MB, Goldberg LI. Dopamine in the treatment of hypotension and shock. *N Engl J Med* 1966;275:1389– 1398.
- 61.Landry DW, Levin HR, Gallant EM, Ashton RC Jr, Seo S, D'Alessandro D, Oz MC, Oliver JA. Vasopressin deficiency contributes to the vasodilation of septic shock. *Circulation* 1997;95:1122–1125.
- 62. Hayes MA, et al. Elevation of systemic oxygen delivery in the treatment of critically ill patients. *N Engl J Med* 1994;330:1717–1722.
- 63. Vincent JL. Hemodynamic support in septic shock: guidelines for the management of severe sepsis and septic shock. International Sepsis Forum. *Intensive Care Med* 2001;27:S80–S92.
- 64.Bone RC, et al. Sepsis syndrome: a valid clinical entity. Methylprednisolone Severe Sepsis Study Group. *Crit Care Med* 17: 389–393.
- 65. Wiles JB, Cerra FB, Siegel JH, Border JR. The systemic septic response: does the organism matter? *Crit Care Med* 1980;8:55–60.
- 66. Moine P, Abraham E. Immunomodulation and sepsis: impact of the pathogen. *Shock* 2004;22:297–308.
- 67. Yu SL, Chen HW, Yang et al . Differential gene expression in gramnegative and gram-positive sepsis. *Am J Respir Crit Care Med* 2004;169:1135–1143.
- 68.Bone RC, Fisher CJ, Clemmer TP, Slotman GJ, Metz CA, Balk RA. Sepsis syndrome: a valid clinical entity. *Crit Care Med* 1989;17: 389–393.

- 69.Poeze M, Ramsay G, et al. An international sepsis survey: a study of doctors' knowledge and perception about sepsis. *Crit Care* 2004;8:R409–R413.
- 70.Levy MM, et al. 2001 SCCM/ESICM/ACCP/ATS/SIS International Sepsis Definitions Conference. *Crit Care Med* 2003;31:1250–1256.
- 71. Friedman G, Silva E, Vincent JL. Has the mortality of septic shock changed with time? *Crit Care Med* 1998;26:2078–2086.
- 72. Alberti C, Brun-Buisson C, et al. Influence of systemic inflammatory response syndrome and sepsis on outcome of critically ill infected patients. *Am J Respir Crit Care Med* 2003;168:77–84.
- 73.Sands KE, Bates DW, et al . Epidemiology of sepsis syndrome in 8 academic medical centers. Academic Medical Center Consortium Sepsis Project Working Group. JAMA 1997;278:234–240.
- 74. Vincent JL, Bihari D, Suter PM, Bruining HA, White JL, Nicolas-Chanoine MH, Wolff M, Spencer RJ, Hemmer M; Members of the EPIC International Advisory Group. The prevalence of nosocomial infection in intensive care units in Europe: the results of the EPIC study. JAMA 1995;274:639–644.
- 75.Brun-Buisson C, Meshaka P, Pinton P, Vallet B. EPISEPSIS: a reappraisal of the epidemiology and outcome of severe sepsis in French intensive care units. *Intensive Care Med* 2004;30:580–588.
- 76. Finfer S, Bellomo R, Lipman J, French C, Dobb G, Myburgh J. Adultpopulation incidence of severe sepsis in Australian and New Zealand intensive care units. *Intensive Care Med* 2004;30:589–596.
- 77.Presterl E, Staudinger T, et al. Cytokine profile and correlation to the APACHE III and MPM II scores in patients with sepsis. Am J Respir Crit Care Med 1997;156:825–832. Guidet B, Aegerter P, Gauzit R,

Meshaka P, Dreyfuss D. Incidence and impact of organ dysfunctions associated with sepsis. *Chest* 2005;127:942–951.

- 78. Vincent JL, et al. Use of the SOFA score to assess the incidence of organ dysfunction/failure in intensive care units: results of a multicentric, prospective study. *Crit Care Med* 1998;26:1793–1800.
- 79.Luzzani A, Polati E, Dorizzi R, Rungatscher A, Pavan R, Merlini A. Comparison of procalcitonin and C-reactive protein as markers of sepsis. *Crit Care Med* 2003;31:1737–1741.
- 80. Villar J, Maca-Meyer N, Perez-Mendez L, Flores C. Bench-to-bedside review: understanding genetic predisposition to sepsis. *Crit Care* 2004;8:180–189.
- 81. Appoloni O, Dupont E, Andrien M, Duchateau J, Vincent JL. Association of TNF2, a TNF^a promoter polymorphism, with plasma TNF^a levels and mortality in septic shock. *Am J Med* 2001;110:486– 488.
- 82. Cohen J, Cristofaro P, Carlet J, Opal S. New method of classifying infections in critically ill patients. *Crit Care Med* 2004;32:1510–1526.
- 83.Roman-Marchant O, Orellana-Jimenez CE, De Backer D, Melot C, Vincent JL. Septic shock of early or late onset: does it matter? *Chest* 2004;126:173–178.
- 84. Vincent JL, Gris P, Coffernils M, Leon M, Pinsky MR, Reuse C, Kahn RJ. Myocardial depression characterizes the fatal course of septic shock. *Surgery* 1992;111:660–667.
- 85.Duff JH, Groves AC, McLean APH, MacLean LD. Defective oxygen consumption in septic shock. Surg Gynecol Obstet 1969;128:1051– 1060.

- 86. Fink MP. Bench-to-bedside review: cytopathic hypoxia. *Crit Care* 2002;6:491–499.
- 87. Kluger MJ, Ringler DH, et al. Fever and survival. *Science* 1975;188:166–168.
- 88. Kregel KC. Heat shock proteins: modifying factors in physiological stress responses and acquired thermotolerance. J Appl Physiol 2002;92:2177–2186.
- 89.Patel BM, Chittock DR, Russell JA, Walley KR. Beneficial effects of short-term vasopressin infusion during severe septic shock. *Anesthesiology* 2002;96:576–582.
- 90.Bone RC (1991) Sepsis, the sepsis syndrome, multiorgan failure: a plea for comparable definitions. *Ann Intern Med* 114: 332–333.
- 91.Bone RC (1992) Toward an epidemiological and natural history of SIRS. JAMA 268:3452–3455.
- 92. Knaus WA, Sun X, Nystom PO, Wagner DP (1992) Evaluation of definitions for sepsis. *Chest* 101:1656–1662.
- 93.McLauchlan GJ, et al. Outcome of patients with abdominal sepsis treated in an intensive care unit. *Brit J Surg* 82:524–529.
- 94.Pittet D, Thiévent B, Wenzel RP, Li N, Gurman G, Suter PM (1993) Importance of pre-existing co-morbidities for prognosis of septicemia in critically ill patients. Intensive Care Med 19: 265–272.
- 95.Pittet D, Rangel-Frausto S, Li N, Tarara D, Costigan M, Rempe L, Jebson P, Wenzel RP (1995) Systemic inflammatory response syndrome, sepsis, severe sepsis and septic shock: incidence, morbidities and outcomes in surgical ICU patients. *Intensive Care Med* 21:302– 309.
- 96. Harrisons Text Book of Internal Medicine 17th edition, Ch265.

1	INSTITUTIONAL ETHICAL COMMU	TEE	
MADE	RAS MEDICAL COLLEGE, CHENN	AI-600 003	*
L.Dis.No.14597/ME5	5/Ethics Dean/MMC/2010	Telephone 2 Fax 044 Dated : 12	25363970 4 2535115 2.05.2010
Title of the work	"The incidence, Etible Prognosis energy po SEPSIS Spectra admit Ward in a tertiary of	gy Kisk V litients wi lied to the care Hospi	th SIRS/ medical Hal.
Principal Investigator Designation Department	Pa in MD General P	edicine.	

Machon Medical College 2 GGH, Ch-3. The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 12th May 2010 at 2.p.m in Pharmacology Seminar Hall, Madras Medical College, Chennai -3

The members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

- 1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
- 2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
- 3. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
- 4. You should not deviate form the area of the work for which you applied for ethical clearance.
- 5. You should inform the IEC immediately, in case of any adverse events or serious adverse
- reactions. You should abide to the rules and regulation of the institution(s). 6.
- 7. You should complete the work within the specified period and if any extension of time is
- required, you should apply for permission again and do the work. You should submit the summary of the work to the ethical committee on completion of the 8. work.
- 9. You should not claim funds from the Institution while doing the work or on completion.
- 10. You should understand that the members of IEC have the right to monitor the work with prior intimation.

SECRETARY IEC, MMC, CHENNAL

JNA1

DEAN MADRAS MEDICAL COLLEGE, CHENNAI -3