

**A STUDY ON NOSOCOMIAL INFECTION IN
INTENSIVE MEDICAL CARE UNIT-INCIDENCE,
PATTERN AND ETIOLOGY**

**DISSERTATION
ON
M.D. DEGREE EXAMINATION
BRANCH I
(GENERAL MEDICINE)**



**THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY
CHENNAI – TAMILNADU.
TIRUNELVELI MEDICAL COLLEGE
TIRUNELVELI**

APRIL – 2014

CERTIFICATE

This is to certify that this dissertation titled **“A STUDY ON NOSOCOMIAL INFECTION IN INTENSIVE MEDICAL CARE UNIT-INCIDENCE, PATTERN AND ETIOLOGY”** is the bonafide Original work done by **Dr. R.RAMARAJ**, submitted as partial fulfillment for the requirements of M.D. Degree Examinations, General Medicine (Branch I) to be held in APRIL 2014. The period of study was from September 2012 to August 2013.

PROF. Dr. S.ALAGESAN, M.D., D.M (NEURO).

PROF. Dr. R. GEETHARANI, M.D.

Additional Professor of Medicine,

Professor and H.O.D

Unit Chief M V

Department of Medicine

Department of Medicine

Tirunelveli Medical College Hospital

Tirunelveli Medical College Hospital

Tirunelveli.

Tirunelveli.

THE DEAN

Tirunelveli Medical College hospital, Tirunelveli.



TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI,

STATE OF TAMILNADU, INDIA

PIN CODE:627011

Tel: 91-462-2572733, 2572734 Fax: 91-462-2572944



Estd:1965

Under the Directorate of Medical Education, Government of Tamilnadu.

Institutional Ethical Committee

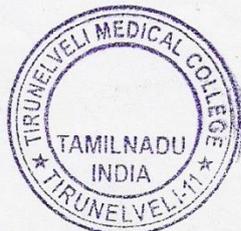
Certificate of Approval

This is to certify that the Institutional Ethical Committee of this College unanimously approves the Thesis /Dissertation/ Research Proposal submitted before this committee by Dr. R.RAMARAJ, A POST GRADUATE IN MD MEDICINE, Department of MEDICINE of Tirunelveli Medical College /Hospital, Tirunelveli titled **"A STUDY ON NOSOCOMIAL INFECTION IN INTENSIVE MEDICAL CARE UNIT – INCIDENCE, PATTERN AND ETIOLOGY"** registered by the IEC as 191/G.M./IEC/2012 dated. 11.07.2012. The Investigator is hereby advised to adhere to all the stipulated norms and conditions of this ethical committee.

Issued on this Date

11.07.2012

Under Seal



Secretary
Secretary,
Ethical Committee,
Tirunelveli Medical College,
Tirunelveli-11.

Originality

GradeMark

PeerMark

A study on nosocomial infection in intensive medical care unit-

BY RAMARAJ RAMASAMY



18%

SIMILAR

--

OUT OF 0

A STUDY ON NOSOCOMIAL INFECTION IN INTENSIVE MEDICAL CARE UNIT-INCIDENCE, PATTERN AND ETIOLOGY

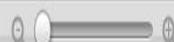
DISSERTATION
ON
M.D. DEGREE EXAMINATION
BRANCH I
(GENERAL MEDICINE)



THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY
CHENNAI - TAMILNADU.
TIRUNELVELI MEDICAL COLLEGE
TIRUNELVELI

Match Overview

Rank	Source	Similarity
1	misc.medscape.com Internet source	2%
2	Submitted to University... Student paper	1%
3	www.ayubmed.edu.pk Internet source	1%
4	www.indiachest.org Internet source	1%
5	healthcare.financialexp... Internet source	1%
6	Mehta, A. "Device-ass... Publication	1%
7	Leone, M. "Catheter-a... Publication	1%
8	Chawla, R. "Epidemiol... Publication	1%
9	www.isccm.org Internet source	1%



ACKNOWLEDGEMENT

I am extremely grateful to **The DEAN**, Tirunelveli Medical College, for granting me permission to do this dissertation work in Tirunelveli Medical college Hospital, Tirunelveli.

I express my sincere gratitude to the professor and the Head of the Department of Medicine **Prof. Dr.R.GEETHARANI, M.D.**, for her valuable support and guidance in preparing this dissertation.

I am greatly indebted to my Unit chief **Prof. Dr.S.ALAGESAN. M.D,D.M. (Neuro)** who inspired, encouraged and guided me in every step of this study.

I am thankful to Assistant Professors of my unit, **Dr.A.Prince Prabhakaran M.D, Dr. T.Grashia, M.D., Dr.S.Manikandan,M.D**, for their guidance and help throughout this work.

I thank the Microbiology,Biochemistry Departments for their help in investigation aspects.

I express my gratitude to all the patients who participated in this study.

I Thank GOD for his blessings.

DECLARATION

I Dr.R.Ramaraj, solemnly declare that dissertation titled “A STUDY ON NOSOCOMIAL INFECTION IN INTENSIVE MEDICAL CARE UNIT-INCIDENCE,PATTERN AND ETIOLOGY” is a bonafide work done by me at Government Tirunelveli medical college and Hospital from September 2012 to August 2013 under the guidance and supervision of my unit chief PROF.DR.S.ALAGESAN,M.D,D,M(NEURO), Professor of medicine.

This Dissertation is submitted to Tamilnadu DR.MGR.Medical University, towards partial fulfillment of requirement for the award of M.D. Degree (Branch-I) in GENERAL MEDICINE.

PLACE:Tirunelveli

DATE:

DR.R.RAMARAJ

CONTENTS

	Page No.
1. INTRODUCTION	1
2. AIM OF THE STUDY	4
3. REVIEW OF LITERATURE	5
4. MATERIALS AND METHODS	48
5. RESULTS AND OBSERVATIONS	51
6. DISCUSSION	76
7. CONCLUSION	81
BIBLIOGRAPHY	82
PROFORMA	92
MASTER CHART	95
ABBREVIATIONS	

ABSTRACT

A STUDY ON NOSOCOMIAL INFECTION IN INTENSIVE MEDICAL CARE UNIT-INCIDENCE,PATTERN AND ETIOLOGY.

R.RAMARAJ, FINAL YEAR POST GRADUATE,M.D.GENERAL MEDICINE,TIRUNELVELI MEDICAL COLLEGE HOPITAL,TIRUNELVELI.

BAKGROUND:

Nosocomial infection is defined as an infection which develops 48 hours after admission to hospital and which was not incubating at the time of admission.The nosocomial infection results in increase in hospital stay time,increased morbidity and mortality.The aim of this study was to find the incidence,Etiology of nosocomial infection in intensive medical care unit in Tirunelveli medical college hospital.It was conducted in 200 patients from September 2012 to august 2013.

METHODS:

All patients admitted in IMCU in Tirunelveli Medical college Hospital and stayed in the IMCU for more than 48 hours were included in the study.Data was included in a proforma and analysed using Epidemiological Information Package 2010 developed by Centre for disease control,Atlanta.

RESULTS:

During the study period out of 200 patients 16 patients developed Nosocomial infection.So the incidence of Nosocomial infection was 8%.The most common Nosocomial infection was urinary tract infection(5.5%) followed by respiratory infection in 2% and blood stream infection in 0.5%.The most common organism causing Nosocomial infection was Klebsiella(5%),E.Coli(2%) and pseudomonas(1%).

CONCLUSION:

Patients admitted in IMCU are at more risk for developing nosocomial infection than in general wards.In our study Urinary tract infection was the

commonest followed by respiratory and blood stream infection. Gram negative organisms were the most common cause in this study.

KEY WORDS:

Nosocomial infection, Hospital acquired infection, Intensive care unit infection.

INTRODUCTION

Nosocomial infection is defined as an infection which develops 48 hours after hospital admission or within 48 hours after being discharged^[1,36] and the infectious agent or toxin should not be incubating at the time of admission. The risk of nosocomial infection is 5 to 10 times higher in intensive medical care unit than in general wards^[5,38]. The nosocomial infection is more common in elderly, immunosuppression, diabetics, renal failure, family members with MDR organisms¹. After admission, a patient's flora acquires the characteristics of surrounding bacterial pool. Most infections which become clinically evident after 48 hours of hospitalization are hospital-acquired. Infections which occur after the discharge of the patient from the hospital is healthcare-associated if the organisms were acquired during the hospital stay.

Hospital-based programs of prevention, control and surveillance of nosocomial infections are in place since the 1950s.^[2] The Study on the Efficacy of Nosocomial Infection Control Project (SENIC) in 1970s showed that nosocomial rates could be reduced by 32% if infection surveillance were coupled with appropriate infection control programs.^[3] In 2005, the National Healthcare Safety Network (NHSN) was started in United states to integrate and succeed previous surveillance systems at the Centers for Disease Control and Prevention (CDC): National Nosocomial Infections Surveillance (NNIS), Dialysis Surveillance Network (DSN) and National

Surveillance System for Healthcare Workers (NaSH)^[4]. Both developed and resource-poor countries are faced with the burden of healthcare-associated infections. In a World Health Organization (WHO) cooperative study (55 hospitals in 14 countries from four WHO regions), about 8.7% of hospitalized patients had nosocomial infections.^[6]

A 6-year surveillance study from 2002-2007 involving intensive care units (ICUs) in Latin America, Asia, Africa, and Europe, using CDC's NNIS definitions, showed higher rates of central-line associated blood stream infections , ventilator associated pneumonias and catheter-associated urinary tract infections than those of comparable United States ICUs.^[7]

Patients are treated better in hospitals than in other places. But presence of a large number of patients under the same roof facilitate the spread of infection from one person to another. Infections in hospitals existed even in ancient times. Nosocomial infections in this era of powerful antibiotics still are important consequence of hospitalization. A minimum 4% of patients are discharged from the hospital after acquiring infections based on underlying disease of the patient, hospital size and numerous other factors. Nosocomial infection places a huge burden on the patient and country. It prolongs the hospital stay of the patient. So it affects the economy of the patient's family as the patient and his family members could not go to work. Indirectly it affects the productivity of the country.

The nosocomial infection can be prevented by maintaining asepsis in the concerned ward. Hand washing of the health personnel is the most important factor. In addition maintaining strict asepsis during urinary catheterization, during intubation, during insertion of vascular catheter is very important.

In this study we want to find the incidence of nosocomial infection and the organisms causing it so that appropriate precautionary measures could be taken. Also the empirical antibiotics could be given to cover these organisms.

AIM OF THE STUDY

1) To find incidence of Nosocomial infection in those patients admitted in intensive medical care unit in Tirunelveli Medical college hospital.

2) To find the etiological agents in such infections.

3) To determine the incidence of specific type of nosocomial infection.

REVIEW OF LITERATURE

DEFINITION

The term nosocomial infection is now known as hospital acquired infection (HAI) and expanded to health care associated infections (HCAI). It includes infections acquired in institutions other than the acute-care facilities (e.g. nursing homes) during hospital stay but not diagnosed till discharge and through outpatient care such as day surgery, dialysis, or those on home parenteral therapy. It is defined as a disease condition resulting from the presence of an infectious agent or its toxin which was not present or incubating at the time of admission to hospital. Usually the infection becomes evident 48 hours or more after admission¹.

The common sites of infection are:

- Respiratory tract
- Blood stream
- Urinary tract
- Surgical site.

HISTORICAL MILESTONES:

- Egyptian papyrus written in 3000 B.C gives details of hospital related infections. The absence of data regarding hospital related infections before this period does not mean absence of infection before this period.

- In Ayurveda (600 B.C) there is detailed description about hospital acquired infections and how to prevent or minimize them. There is also description about segregation of infective patients from normal persons.
- The great physician Charaka and pioneer of many surgeries Sushruta have written about the prevention of infection in clinical practice.
- The Herodotus records describe about the conditions of hospitals in Rome and Greek in 1000 to 600 B.C give evidence about the infections.
- Hippocrates in 400 B.C also mentioned about importance of hospital acquired infections and the means to prevent them.
- For several centuries the westerners believed that the cause for the disease is the contagion and disease may spread by wind and various air currents.

It was found that certain drugs had the ability to prevent or check the progress of infection.

In 1856 Louis Pasteur found that some bacteria was the reason for the fermentation of wine which can be prevented by heating during which the microorganisms were killed. In 1864 he proved that many such microorganisms existed in the atmosphere.. In a famous lecture to Acadimiede Medicine in 1873, Louis Pasteur said that “If I had the honour of being a surgeon, not only would I use absolutely clean instruments, but after

cleaning my hands with the greatest care would only use sponges previously raised to a heat of 1300-1500 Fahrenheit. I would still have to fear germs suspended in the air, and surrounding the bed of the patient.”

The presently famous work of Semmelweiss on causes for puerperal sepsis was not accepted during 1861. He found that puerperal sepsis was more common with doctors who examined patients after doing autopsy. Semmelweiss proposed that morbid matter were transferred to the hands of doctors from cadavers or other patients. This was responsible for the disease transmission. A drastic reduction in rate of infection was achieved by hand-washing with chlorinated lime.

Florence Nightingale noted in the book “**Notes on Hospitals**”-

“It may seem a strange principle to enunciate as the very requirement in a Hospital that it should do the sick no harm.”

The real rate of mortality and morbidity in large city hospitals is higher for same type of diseases than in patients getting treatment out of the hospital.

Florence Nightingale did not accept germ theory of disease. She gave guidelines regarding nursing care, design of the hospital and personal hygiene.

In **1869 Simpson** in his “The sequelae of amputation” found that in large city hospitals the incidence of sepsis was more than in rural practice.

As per **Lister's** theory of antiseptic wound packing of compound fracture by carbolic acid and sterilization of instruments, suture materials reduce the rate of infection. Decontamination of hands and is an important aseptic procedure.

Gustao Neubar introduced the use of protective mask and sterile gown during surgery in the year 1883. **Halsted** used rubber gloves during surgery for the first time in 1890. **Von Bergman** introduced steam sterilisation in **1896**. Use of the mask and gloves increased decreased infection rate during and after the surgery and improved the success rate of surgery.

Flugge established that tuberculosis spread by aerial and droplet spread in 1897. **Hutinel found** the isolation technique of diphtheria and many other bacteria in 1894.

In the 20th century the prevention of infection during and after surgery by aseptic techniques gained importance and this was given more importance than the antibiotic use.. Adequate Ventilation of the operation theatre was given paramount importance.

THE ERA OF ANTIBIOTICS

The discovery of penicillin reduced severe infection and sepsis caused by many bacteria including *Staphylococcus aureus*. Many streptococcal infections were prevented or treated effectively. So both severe infections and mild infections were mainly caused by staphylococcus.

Resistance to penicillin and other antibiotics emerged subsequently which resulted in severe infections and sepsis by S.Aureus. Air borne, dust borne mode of spread of infection were studied. Spread of infection through the infected hand of hospital workers and relatives were also studied. S.Aureus infections began to decrease due to the use of newer powerful and broad spectrum antibiotics. After the decrease in incidence of gram positive infections, infections due to gram-negative bacteria began to occur in more patients; Many outbreaks occurred due to the gram negative bacteria like Klebsiella and E.coli. Pseudomonas aeruginosa also caused a lot of infections particularly in hospitals. If a particular group of antibiotic is used regularly in a community then organisms which are resistant to that particular antibiotic began to emerge.

The Burden

The worldwide nosocomial infection rate ranges from 6% to 15%.^[37] In Asia it ranges from 4% to 48% of which 45% to 65% are lower respiratory tract infections. Highest prevalence occurs in intensive care units (ICUs), in acute surgical and orthopedic wards. In a surveillance conducted in 12 ICUs in India, the rate of HCAI was 4.9% and 9.6 per 1000 ICU days. Healthcare-associated infections result in excess length of stay, mortality and health care costs. In 2002 an estimated 1.7 million healthcare-associated infections occurred in the United States, resulting in 99,000 deaths.^[10] In

March 2009, the CDC released a report estimating overall annual direct medical costs of healthcare-associated infections that ranged from \$28-45 billion.^[11] Nosocomial infections occur in both adult and pediatric patients. Bloodstream infections, followed by pneumonia and urinary tract infections are the most common nosocomial infections in children; Urinary tract infections are the most common healthcare-associated infections in adults.^[12] Among pediatric patients, children younger than 1 year, babies with extremely low birth weight (≤ 1000 g) and children in the PICU or NICU have higher rates of healthcare-associated infections.^[13,14] Ninety-one percent of bloodstream infections were in patients with central intravenous lines (CVL), 95% of pneumonia cases were in patients undergoing mechanical ventilation, and 77% of urinary tract infections were in patients with urinary tract catheters.^[12]

The commonest organisms were:

- Pseudomonas
- Acinetobacter
- Staphylococcus aureus
- Methicillin resistant S.Aureus (MRSA)
- Enterobacteriaceae
- Candida species
- Enterococci

- *Stenotrophomonas*.

Common Sources of Infection

Causative organisms may be present on the skin, nose, mouth, gastrointestinal tract, or vagina of the patient. They may be acquired from external sources like health-care personnel, visitors, hospital equipments, medical devices, or the health-care environment. Most infections are of bacterial etiology, though fungal and viral infections may occur in immunosuppressed patients and those already on broad-spectrum antibiotics.

HOSPITAL ACQUIRED PNEUMONIA (HAP)

Definitions

Pneumonia occurring 48 hours or more after admission and which was not incubating at the time of admission is HAP. Intubation and mechanical ventilation (MV) is associated with 20-fold increase in risk of developing pneumonia.

Ventilator associated pneumonia (VAP) is pneumonia in a person who has a device to assist respiration through an endotracheal tube or tracheostomy tube for a period of at least 48 hours before the onset of infection. VAP represents 80% of episode of HAP. Mortality in VAP due to *Pseudomonas* and *Staphylococcus* is very high.

Health care associated pneumonia (HCAP) ^[31] is defined as pneumonia in any patient with at least one of the following risk factors:

1. Hospitalization in an acute care hospital for >2 days within the last 90 days.
2. Residence in a nursing home or long-term care facility within the last 90 days.
3. Receive outpatient intravenous antibiotics or chemotherapy or home wound care in last 30 days.
4. Attended a hospital clinic or haemodialysis clinic in the last 30 days.
5. Has a family member with known multi-drug resistant pathogens.

SYMPTOMS:

- 1) Cough
- 2) Breathlessness
- 3) Sputum production
- 4) Pleuritic chest pain
- 5) Elevated body temperature.

Symptoms can be absent or moderate in older patients.

Chest X-ray may give clue to etiology:

- 1) Interstitial pneumonia caused by intracellular pathogens.
- 2) Lobar pneumonia may be caused by *S.Pneumoniae*.

CXR allows for staging of severity according to localization and number of involved lobes.

CXR also helps to detect complications:

-Pleural effusion

-Cavitations

-Acute respiratory distress syndrome

CT scan:

-Cavitations in Tuberculosis

-Halo or crescent sign in aspergillosis of neutropenia patients.

Causative Organisms

HCAP may be early onset, that is within 4 days of hospitalization or late onset, beyond 4 days.

The organisms causing **early** infections are:

Moraxella catarrhalis,

Haemophilus influenza

S.Pneumoniae

Viruses

Late onset HCAP are caused by:

- Gram-negative bacteria
- Staphylococcus aureus
- Viruses
- Yeasts
- Fungi
- Legionella

- *Pneumocystis carinii*.

Late onset pathogens often are multi-drug resistant (MDR).^[33] Over 80% of nosocomial pneumonias are caused by Gram-negative bacteria. Now *Acinetobacter* is the organism which is of great concern.

In India, 38% of HAP are caused by *Acinetobacter*, *Pseudomonas* species(20%), *Klebsiella pneumonia* (23%) and MRSA (5%). Forty eight percent of VAP and 2.3% HAP are caused by MDR organisms (Table1), while 7.3% are polymicrobial.^[34,35] In most ICUs, MRSA although present is not as big a problem as in the western world.

Table 1: Risk Factors for Multidrug Resistant(MDR) Infections^[18]

Regular dialysis

Immunosuppression

Heart disease

Renal failure

Hepatic failure

High incidence of antibiotic resistance in the community

Presence of a family member with MDR organism

Table 2: Risk Factors for HAP and VAP^[15,16]

Male

Elderly age

Pre-existing diseases-pulmonary, diabetes, dialysis

Immuno suppression

Presence of intubation

Enteral feeding

Mechanical ventilation

Supine position

APACHE II score > 15

Previous use of antibiotic for > 2weeks

Multi-organ failure

Reintubation due to failed weaning

Use of paralytics, sedative

Length of ICU stay

- Diagnosis of HAP or VAP is made in the presence of progressive radiographic infiltrates or pleural effusion and at least 2 of the 4 clinical signs of infection –
- Fever > 38⁰C,
- Purulent secretions,
- Leucocytosis or Leucopaenia,
- Decreasing oxygenation.

Blood cultures are rarely positive. Positive pleural effusion culture is considered as specific. However spread of infection to pleural space is rare.

Analysis of lower respiratory secretion is the most commonly used technique

to find organisms causing pneumonia. Microscopy and culture of sputum or endotracheal aspirates are associated with a high percentage of false positive results because of colonization of upper respiratory tract or trachea - bronchial tree. If culture of endotracheal secretions is sterile in a patient with no change in antimicrobial therapy within the last 72 hours Ventilator associated pneumonia can be ruled out with high probability.

-Negative predictive value >90%.

-Extra pulmonary infectious process must be evaluated.

Management

1. Identification of pulmonary infection is the first step.
2. Appropriate culture is required.
3. Semi-quantitative or quantitative cultures of lower respiratory tract should be performed if HAP or VAP is suspected. Endotracheal aspirates, bronchoalveolar lavage (BAL), protected specimen brush (PSB) are required to isolate organisms. A quantitative endotracheal culture or non-bronchoscopic BAL is more relevant in the Indian set-up. Recent start or change of antibiotics in the preceding 24 to 72 hours may give rise to false negative reports.
4. A broad-spectrum antibiotic should be started at the earliest in all clinically unstable patients regardless of culture reports as delay is associated with increased mortality. Choice of empirical antibiotics is guided by the local data on risk factors, local prevalence of organisms

and resistance patterns. Broad spectrum antibiotics covering Gram-negative and Gram-positive organisms are usually started. A re-evaluation is done at 48 to 72 hours. Once culture sensitivity reports are available de-escalation may be done.

A clinical pulmonary infection score (CPIS), based on temperature, total leukocyte count, chest radiographic findings, respiratory secretions, endotracheal aspirate cultures and oxygenation status has been developed to predict presence of VAP. If CPIS is less than 6 both at baseline and at 72 hours, most clinicians would safely allow stopping antibiotics.

Guidelines for initial empiric antibiotic treatment:

- If no risk factors for MDR pathogens and early onset VAP (duration of hospitalization less than 5 days) we may give monotherapy or limited spectrum antibiotic.
- In patients with late onset (>5 days) or with risk factors for MDR pathogens a broad spectrum antibiotic or a combination of antibiotics should be given.
- Initial choice should take in to account:
 - Patient characteristics
 - Underlying diseases
 - Contraindications to certain antibiotics.

De-escalation strategy:

Once the culture results are available change the broad spectrum antibiotic to a narrow spectrum to which the organism is susceptible. This prevents the development of resistance.

Duration of therapy:

If aminoglycosides are used treatment may be stopped after 7 days. No clear consensus has been reached as to the duration of antimicrobial therapy for ventilator-associated pneumonia (VAP). Many experts treat for 14-21 days. However, shorter course of antibiotic therapy (about 1 wk) may be adequate therapy for some cases.^[17]

Response to therapy:

Improvement is usually apparent after 48 to 72 hours of antibiotic therapy. Fever and hypoxemia are the best indicators for monitoring treatment.

- Temperature becomes less than 38° C or
- Pao₂/Fio₂ becomes more than 250 within 72 hours of adequate treatment.

BLOOD STREAM INFECTION (BSI)

Epidemiology

Primary blood stream infections are identified by growth of pathogenic bacteria or fungi (that are not related to another site of infection)

from one or more blood cultures. Skin contaminants like coagulase Staphylococcus or Diphtheroids are considered causative of BSI, if more than one blood culture is positive along with presence of systemic signs and symptoms of infection like fever, chills, and hypotension. An alternative focus of infection should be absent.

CATHETER ASSOCIATED BLOOD STREAM INFECTIONS:

Catheter associated blood stream infections (CABSI) is said to be present if fever occurs during and up to 48 hours after removal of central venous catheter or arterial catheter but diagnosis does not require growth of same organism from the blood and the catheter.

Catheter related blood stream infection (CRBSI)

Diagnosis of CRBSI requires growth of same organism quantitative or semi-quantitative from the blood as well as the catheter.

CRBSI is seen in 5% patients with indwelling vascular uncoated catheter and almost 2 to 5 infections per 1000 catheter days. All lines arterial or central venous are risky. The incidence of CRBSI increase with the duration of catheterization,^[26,27,28] number of ports, and number of manipulations. Mortality may be almost 8% in Staphylococcus aureus bacteraemia. Fever, hypotension, purulence at exit site, blocked lumen, all may herald CRBSI. BSI due to short peripheral intravenous catheters is very low but phlebitis is very common. Line removal should be considered if the line is no longer needed; if the infection is caused by *S.*

aureus, *Candida* species, or mycobacteria; if the patient is critically ill; if the bacteremia does not clear in 48-72 hours; if symptoms of bloodstream infection persist beyond 48-72 hours; and if noninfectious valvular heart disease, endocarditis, metastatic infection, or septic thrombophlebitis is present.^[17]

In a report from north India, incidence of CRBSI was 19.4%. Organisms causing nosocomial BSIs were *Pseudomonas* (33% episodes), and *Acinetobacter*, *Escherichia coli*, *Candida* species, coagulase-negative *Staphylococci* and *S.Aureus*.

PATHOGENESIS:

First step is the colonization of the catheter. For non-cuffed catheters skin insertion site is the source of colonization. For cuffed catheters lumen of the hub is the primary source of entry. Micro organisms are introduced via the hand of the medical personnel while manipulating the hub. Colonization is universal after insertion of a central venous catheter but is independent of catheter related infection.

Second step in pathogenesis is the formation of biofilm of extracellular polysaccharide rich slimy material by organisms. It promotes adhesiveness of bacteria to the surface of the catheter. Also resists antibiotics.

Femoral catheterization is associated with a higher rate of infection and thrombotic complications when compared to subclavian catheterization.

Transparent occlusive dressings produce a warm environment. So they are associated with a high rate of infection than gauze dressing of the catheter.

Clinical manifestations:

- 1) Local manifestations
- 2) Systemic manifestations

Local manifestations:

- Erythema
- Edema
- Tenderness
- Purulent discharge

SYSTEMIC MANIFESTATIONS:

- Fever and chills
- Hypotension
- Hyperventilation
- Altered mental status
- Nausea and vomiting
- Abdominal pain
- Diarrhea

Exit site infection:

Purulent drainage from the catheter exit site or erythema, tenderness and swelling within 2 cm of the catheter exit site and colonization of the catheter if removed.

Port-pocket infection:

Erythema or necrosis of the skin or subcutaneous tissue either over or around the reservoir of the implanted catheter and colonization of the catheter if removed.

Tunnel infection:

Erythema, tenderness and induration of the tissues above the catheter and more than 2 cm from the exit site and colonization of the catheter if removed.

Diagnosis

BSI is identified by the growth of pathogenic bacteria or fungi (that are not related to another site of infection) from one or more blood cultures drawn from peripheral veins. At least two sets of blood cultures must be drawn in each instance. Three sets may be needed to establish continuous bacteraemia.

Different methods of diagnosing CRBSI have been described. Some require removal of the catheter (qualitative, semi-quantitative and quantitative cultures) while some can be done while retaining the catheter in place(qualitative or quantitative blood cultures from catheter). The best method is to obtain paired blood cultures, one from the central catheter and

another from the peripheral venous blood and the different time to culture positivity is noted. If central line sample shows positivity 2 hours earlier than the peripheral culture, it is a CRBSI.

CATHETER SPARING DIAGNOTIC METHODS:

- Paired blood cultures simultaneously from the central vein and peripheral vein.
- Both blood samples drawn less than 10 minutes apart with the same volume of blood.
- CVL/PERIPHERALRATIO of CFU of 5:1 represents true infection.
- Acridine orange cytospin technique:
Positive test indicates presence of bacteria. It is a rapid diagnostic test. It takes only 30 minutes for this test.
- Catheter-drawn quantitative blood culture is the method in which a single quantitative blood culture is drawn from central venous catheter. Cutoff of 100 CFU/ml establishes the diagnosis. Major drawback is that it cannot distinguish between CRBSI and high grade bacteremia.

DIAGNOSTIC METHODS REQUIRING CATHETER REMOVAL:

- Semi quantitative roll-plate catheter culture:

It is the international reference diagnostic method. Consists of rolling a 3 to 5 cm section of the distal tip of the central venous catheter over a agar plate. Cutoff of >15 CFU defines catheter colonization.

- **QUANTITATIVE CATHETER CULTURES:**

Involves flushing a catheter segment in a broth with a target of retrieving organisms from both surfaces of catheter. Threshold of >1000 CFU correlated best with colonization.

- **STAIN AND MICROSCOPY RAPID DIAGNOSTIC TECHNIQUES:**

It includes staining the removed catheter segments and subsequent fields indicate colonization.

Acridine orange staining is used for rapid diagnosis in which fluorescence is indicative of positivity.

PREVENTIVE STRATEGY:

- Central venous catheters should be used only if medically necessary and should be removed as early as possible.
- Hand washing
- Maximal sterile barriers during insertion.
- Cutaneous antiseptics with chlorhexidine.
- Avoidance of femoral site.

ANTIMICROBIAL CATHETER LOCK SOLUTIONS:

It involves flushing catheter lumen and then filling with 2 to 3 ml of a combination of anti-coagulant and a anti-microbial agent. Dwell time varies between 20 to 24 hours. Not possible if catheter has to be used. It is used in catheters which have to be kept for more than 30 days. Combination of vancomycin and heparin with or without ciprofloxacin is used. Minocycline and EDTA can also be used.

ANTIMICROBIAL IMPREGNATION OF CATHETERS:

Consists of impregnation of outer or inner surface of catheters with antibiotics. Slow release of antimicrobials will prevent initial colonization and biofilm formation. Concern has been expressed regarding development of resistant organisms in these patients.

Management

Management includes:

- Confirming the source of infection
- Determining the choice of antimicrobials
- Determining the duration of therapy
- Deciding whether to remove the catheters

Catheter should be removed if :

- CRBSI is suspected
- Purulence at the insertion site

- Haemodynamically unstable
- Organ dysfunction
- Fungal sepsis
- MDR organisms
- Once the diagnosis is confirmed.

Routine replacement over a guide wire is not recommended. Empirical antibiotics should be started in seriously ill patients according to the local microbiological flora and this may require a change according to the culture sensitivity reports. Duration of antibiotics is tailored according to the causative organism and by the presence or absence of any complication. Fungal sepsis should be considered in patients at risk like in those with prior antibiotic exposure, parenteral nutrition,^[29,30] abdominal surgeries, and immune compromised host.

SURGICAL SITE INFECTION (SSI)

Epidemiology

In India incidence of postoperative infections in hospitals varies from 10% to 25%. Wound infections affect nearly 20% of post-operative cases .These occur due to close contact of medical and paramedical staff with the patient at various stages of treatment. In a north Indian hospital, incidence of wound infection in post-operative elective surgeries ranged from 11% to 70% due to S.Aureus and 30% due to E.Coli.^[19,20]

Surgical patients are at risk of infection for many reasons. Surgery is inherently invasive. It creates portal of entry in natural epithelial barriers for pathogens to invade the host. Surgical illness is immune suppressive (trauma, burns, malignant tumors). There may be therapeutic immune suppression following solid organ transplantation.

During surgery patients may be given general anesthesia. These patients will have:

- Period of reduced consciousness during emergence
- Risk of pulmonary aspiration of gastric contents
- Nosocomial pneumonia occurs more frequently among surgical patients than comparably ill medical patients.

CONTROL OF BLOOD SUGAR:

Hyperglycemia is deleterious to host immune function. Poor peri-operative control of blood sugar increases the risk of infection and worsens outcome from sepsis. Blood sugar value >200 mg/dl any time on first postoperative day increases the risk of surgical site infection 4 times. Blood glucose level should be maintained below 140 mg/dl. Some studies show that it decreases the mortality by 20 to 40%. There is less incidence of nosocomial infection and less organ dysfunction.

BLOOD TRANSFUSION:

Blood transfusion increases the risk of infection. Transfusion exerts immunosuppressive effects through:

- Presentation of leucocyte antigens.
- Induction of shift to T-Helper 2 phenotype
- Leucocytes depleted red blood cell transfusion does not decrease the risk of infection.

Stored blood leads to loss of 2,3-diphospho glycerate and adenosine triphosphate. This leads to loss of membrane deformability. This causes disruption of nutrient blood flow and impaired oxygen offloading. Thus blood transfusion does not increase oxygen delivery to critically ill patients with sepsis. It may increase the risk of organ dysfunction.

Table3: Factors Determining Nosocomial Wound Infection

Factors related to surgical procedures:

- Pre-operative shaving-1 day before operation,
- Type of surgery
- Anesthesia
- Wound drains
- Tissue damage
- Blood loss

Host factors:

- Age
- Immunity
- Diabetes

- Nutrition
- Obesity
- Antibiotic.

Diagnosis and Management

Signs of wound infection are:

- Local redness
- Swelling
- Wound discharge
- Fever
- In severe cases shock and organ dysfunction.

Appropriate culture from wound, drain and blood should be sent and empirical antibiotics started. Prevention of SSI includes treating infections harboured by the host before surgery, good antiseptic precautions, and antibiotic prophylaxis within 1 hour of surgery, hair clipping rather removal and optimum post-operative care including good glycemic control.

URINARY TRACT INFECTIONS (UTI)

UTI's in hospital are mostly due to urological manipulation or the presence of indwelling catheters.

Risk for UTI is high in:

- Female

- Diabetics
- Elderly
- Peripartum period
- Prolonged Duration of catheterization.

Catheter-Associated Urinary Tract infections (CAUTI)

A diagnosis of CAUTI is confirmed when a patient meets one of the two criteria. The first is when a patient with a urinary catheter has one or more of the following symptoms with no other recognized cause:

- Fever (temperature $>38^{\circ}\text{C}$),
- Urgency or suprapubic tenderness with
- Culture-positive urine showing more than 10^5 colony-forming units per ml, with no more than two microorganisms isolated.

The second criterion is when a patient with a urinary catheter has at least two of the following criteria with no other recognized cause:

- Positive dipstick analysis for leucocyte esterase or nitrate,
- Pyuria (>10 leucocytes per ml of urine),
- Organism seen on gram-stain or physician diagnosis of urinary tract infection.
- In a report from India, 24% of nosocomial infections were due to UTI and all had indwelling catheters. In another study age and urinary catheterization were independent risk factors for

UTI.^[46] Commonest isolated pathogen is E.coli, others include Enterobacter, S.epidermidis, S.aureus, and Serratia.

Pathophysiology:

Except for distal urethra the urinary tract is normally sterile.

Resistance to UTI is due to:

Exposure to uropathogenic bacteria.

Age

Hormonal status

Urine flow

Insertion of a urinary catheter allows organisms to access the bladder.Catheter induces an inflammation in the urethra.Allows bacteria to ascend in space between urethral mucosa and catheter.

Catheter allows formation of biofilm.It consists of adherent organisms,extracellular products,host components deposited on catheter surfaces.Biofilm protects organism from antimicrobials and host immune response.

Ascending route of infection is common in women due to their short urethra.

Internal route of infection through lumen of catheter is due to reflux of pathogens from drainage system in to bladder.It also occurs when the drainage system fails to close or with contamination of urine in the collecting bag.

MICROBIOLOGY:

Common organisms which cause UTI are:

- Escherichia coli
- Pseudomonas aeruginosa
- Enterococci
- Poly microbial infections in few cases (5 to 12%).

In IMCU gram negative organisms cause more than 70% of cases.

IMPACT OF UTI IN IMCU:

Nosocomial UTI is responsible for significant morbidity to the patients. But the urinary tract is the source of sepsis in only 10 to 14% of cases far less than the lung.

Urosepsis is inflammation of the upper urinary tract which causes seeding of the blood with bacteria which causes local and distant destruction of tissues.

PREVENTION OF UTI:

- Reducing the duration of catheterization is the most important step in prevention of UTI.
- Indwelling catheters are to be used only when necessary
- Sterile techniques are to be used during catheterization
- Closed system of drainage is to be used
- Samples must be taken aseptically

- Irrigation is to be avoided.

URINARY DRAINAGE SYSTEM:

- Maintenance of a closed drainage system is good method for prevention.
- Hand washing should be performed immediately before and after any manipulation of the catheter site or apparatus.
- If small volume of fresh urine is needed for investigation the distal end of the catheter or the sampling port should be cleaned with a disinfectant and urine should be aspirated with a clean syringe.
- Large volumes of urine should be should be obtained aseptically from the drainage bag.
- Unobstructed flow should be maintained.
- Catheter and collection tube should be prevented from kinking.
- Collecting bag should be emptied regularly using a separate collecting container for each patient.
- Poorly functioning or obstructed catheter should be irrigated or replaced.
- Collection bags should be kept below the level of the bladder.
- Indwelling catheter should not be arbitrarily changed at fixed intervals.

Types of urethral catheters:

- Silver alloy catheters reduce the incidence of symptomatic UTI.
- Catheters coated with minocycline and rifampin or nitrofurantoin reduces bacteriuria.

Meatal care:

Twice daily meatal care does not reduce rate of infection. Vesical irrigation with antibiotics is not recommended as it does not reduce infection rate. The organisms also become more resistant.

Alternatives to urinary catheter:

- Condom catheters
- Suprapubic catheterization
- Intermittent urethral catheterization.

Suprapubic catheterization is advantageous as compared to indwelling catheters with respect to bacteriuria, recatheterisation and discomfort.

Management:

- Asymptomatic bacteriuria in catheterized patients is not to be treated.
- Symptomatic UTI should be treated.
- Antibiotics selected should have good tissue penetration, minimal side effects, should attain high urinary levels.

- High urinary levels should be present for an adequate period to eliminate the organisms. Renal concentration of cephalosporins remained higher than minimal inhibitory concentration for the most common bacteria. B-lactam antibiotics have a low pka, poor lipid solubility and penetrate poorly into prostate. Good penetration into prostate tissue has been demonstrated with aminoglycosides, fluoroquinolones, sulfonamides, nitrofurantoin. Renal toxic drugs should be avoided.

TREATMENT OF COMPLICATED UTI:

- Antibiotics should be started within the first hour after taking culture samples.
- Empirical therapy should include one or more antibiotics presumed to have activity against the presumed organism.
- For septic shock a combination of b-lactam with anti-pseudomonal activity and a fluoroquinolone should be used.

TRACHEOBRONCHITIS

It is a very common problem, characterized by at least 2 of the 4, namely fever, cough, new or increased sputum production, rhonchi, or wheezing and at least one of the following: positive culture obtained by deep tracheal aspirate or bronchoscopy or positive antigen on respiratory secretions but without radiographic evidence of pneumonia.

SINUSITIS

This entity is often overlooked in febrile patients especially when nasogastric or nasotracheal tubes are present. Apart from imaging, aspiration of affected sinus is necessary to diagnose the causative organism.

GASTROINTESTINAL INFECTIONS IN THE IMCU:

- 10 TO 30% of nosocomial diarrhea is due to clostridium difficile. Other causes are:
- Antibiotics
- Chemotherapeutic agents
- Proton pump inhibitors
- Tube feeding
- Laxatives
- Idiopathic

Empirical treatment is advised in severe cases while lab tests are pending.

Other pathogens are:

- Rota virus
- Noro virus
- Salmonella species

Diarrhea in immune-compromised host:

- They are at risk of developing opportunistic infections. Use of various chemotherapeutic agents and immune modulators also predispose to diarrhea. Organisms which cause diarrhea in this group are:

- Clostridium difficile
- Salmonella enterica
- Noroo virus
- Cryptosporidium
- Isospora
- Cyclospora
- Cytomegalovirus
- Mycobacterium avium intracellulare

Rapid diagnostic tests:

- Direct stool examination for ova, cysts, parasites
- Stool test for clostridium difficile toxin
- PCR test for cytomegalovirus, herpes virus
- Stool and blood cultures

If these tests do not provide specific diagnosis endoscopy and mucosal biopsy are done to find the etiology.

CLOSTRIDIUM DIFFICILE ASSOCIATED DIARRHOEA (CDAD)

In hospitalized patients C.difficile is one of the most important causes of diarrhea. Illness ranges from mild watery stool to life-threatening colitis and toxic megacolon.

The identifiable risk factors for this include previous antibiotic treatment chemotherapeutics, immune suppressives, surgery, exposure to gastric acid suppressants, low immunity and advanced age. Metronidazole and oral vancomycin are the drugs of choice.

MANAGEMENT:

- Correct dehydration.
- Anti microbials

Dehydration:

Mild:

3 to 5% loss in body weight. Patients have increased thirst and slightly dry mucous membrane. Treated with ORS 50 ml/kg over first 2 to 4 hours.

Moderate:

6 to 9 % loss in body weight. Patients have loss of skin turgor, dry mucous membranes, tenting of skin. Treatment is with ORS 100 ml/kg over first 2 to 4 hours.

Severe dehydration:

>10% of loss in body weight. Patients have lethargy, altered consciousness, prolonged skin retraction time, cool extremities and

decreased capillary refill time. Treatment is with immediate IV fluid replacement with 20 ml/kg of ringers lactate solution to restore perfusion and mental status. Continue with 100 ml/kg ORS.

Empirical antimicrobial treatment.

PREVENTION AND CONTROL OF NOSOCOMIAL INFECTIONS

A hospital infection control committee comprising of a senior microbiologist, intensivist, physician and surgeon is essential to prevent and control HAI. A central sterile supply department (CSSD) should be involved in dealing with sterile equipment and stores. Periodic surveillance of infections is important. Microorganisms, sensitivity patterns, antibiotic use, outcomes, all must be audited. Antibiotic policy should be formulated and revised regularly for effective therapy.

STRATEGIES TO BE ADOPTED TO COMBAT HCAI

1. Environmental factors:

- Adequate bed-space ratio
- Identifying infected zones
- Proper disposal of biomedical wastes in protocol containers
- Ensure food hygiene
- Routine checking of potable, dialysis water
- Ventilation strategies for operating theatres, isolation areas for infected or immune compromised cases

2. Specific standard precautions for all patients in health care settings as recommended by Centers for Disease Control and Prevention:^[32]

- Hand hygiene^[40] with alcohol based rubs is to be performed after examining each patient, before and after every procedure or handling patient's body fluids. In suspected C.difficile infection hands are to be washed with soap and water.
- Respiratory and cough etiquettes are to be followed.
- Mask, eye protection or face shield is to be worn for procedures which might involve splashes.
- N95 or higher masks for diseases transmitted by respiratory aerosols like tuberculosis, some viruses.
- Gloves are to be used where recommended. Masks and gowns are to be worn while handling patients infected with Acinetobacter, MRSA or MDR pathogens.
- Appropriate handling of soiled linen and equipment and disinfection of environmental surfaces.
- Used needles are not to be bent, broken by hand or recapped.
- For patient resuscitation, a mouthpiece, resuscitation bag are needed to prevent contact with oral secretions.

- For injected medications, single dose vials are preferable to multiple dose vials.

With the better availability of technology, India also faces the problems of HAI with its attendant emergence of MDR pathogens. As a consequence of these the outcome in the form of patient survival and cost of therapy is worrying. Strict infection control policies and judicious use of antibiotic will be the cornerstone of combating this problem.

PREVIOUS STUDIES:

1) Nosocomial infections in intensive care unit- Martin langer, Ida salvo, Massimo mussico.

2) A Study on incidence of nosocomial infections in a university hospital.- L.Ortona, G.Federico, M.Fantoni. Study was carried in a 1800 bed hospital over 9 months period. Nosocomial infections were 6.5% per 100 discharges. UTI was the most common. E.coli, proteus, klebsiella were the causative organisms. Catheterisation was the most important risk factor.

3) Risk factors and outcome of nosocomial infections.- results of a matched case control study of ICU patients. April 1998. Emmanualegirou, Francois Stephen, Ananovoara. Studied about the relation between underlying disease, severity of illness, therapeutic drugs and incidence of nosocomial infections. Mortality attributable to nosocomial infections was 44%.

4) Nosocomial blood stream infections in US hospitals—A prospective nationwide surveillance. 24,000 blood stream infections were recorded in 49 hospitals over a 7 year period. Gram positive organisms were responsible for 65% cases, gram negative organisms were responsible for 25% cases, fungi 9.5%.

5) Prospective incidence study of nosocomial infections in a paediatric intensive care unit—in 2003. Pons M, Serra M. 15% patients had nosocomial infections. 51% patients had bacteremia, 19% patients had UTI, 17% had respiratory infections.

6) Study on the efficacy of nosocomial infection control (SENIC project) in 1998 by Hughes M. Evaluated the nosocomial infection control programmes in US. 32% of infections were preventable.

6) Alexis M Elward, et al 55 - Washington – prospective study 2000 - rates, risk factors, and outcomes of ventilator-associated pneumonia in pediatric intensive care unit . The incidence of ventilator-associated pneumonia was 3.3% and 5.1% in mechanically ventilated patients. The most common organisms were *Pseudomonas aeruginosa* (29.4%), *Klebsiella pneumoniae* (14.7%), *Staphylococcus aureus* (11.8%), yeast (8.8%), *Haemophilus influenzae* (8.8%), *Streptococcus pneumoniae* (5.9%). Multiple factors were analysed for risk factors.

Ventilator-associated pneumonia was associated with the following procedures: reintubation, tracheostomy, transfusion, transport out of the

PICU, the presence of a central line, multiple central venous catheters, Bronchoscopy.

Patients with VAP had higher mortality rate (20% vs 7%) which approached statistical significance.

7)Emad H. Ibrahim, et al 58 from Washington did a prospective cohort study identify the occurrence of ventilator-associated pneumonia in a community hospital, and to determine the risk factors for VAP and the influence of VAP on patient outcomes in a nonteaching institution.

Eight hundred eighty patients received mechanical ventilation and comprised the study cohort. One hundred thirty-two patients (15.0%) who received mechanical ventilation acquired VAP during their ICU stay. Patients with VAP were also statistically more likely to require reintubation, tracheostomy, multiple central venous lines, and to receive treatment with histamine type-2 receptor antagonists or sucralfate. Newman CD. Catheter related blood stream infections in the paediatric intensive care unit. 2006

8)GastmeierP,GeffersC,BrandtC. Effectiveness of a nation-wide nosocomial infection surveillance system for reducing nosocomial infections.

9)IYAD I.AL RUN-Community acquired urinary tract infection causing microorganisms among paraplegic patients in Gaza. E. Coli was the most common organism causing community acquired UTI. Urogenic bladder and bladder catheterisation is the most common risk factor.

10) **YUAN, YUAN**-Incidence and factors associated with nosocomial infection in a intensive care unit of an urban hospital in china. The infection rate was 6.5%.

11) **Rahim baghei**.2007. An epidemiological study of nosocomial infections in the patients admitted in the intensive care unit in the urmia imam reza hospital. The incidence of nosocomial infection was 8.5%. Most common infections were pneumonia, UTI. Most common organism causing pneumonia was pseudomonas aeruginosa. Urinary tract infection was caused by E. Coli.

12) **Ritesh agarwal** -2005. Epidemiology, risk factors, outcome of nosocomial infections in a respiratory intensive care unit in North India. 33% patients had infection. 23% patients had pneumonia while 7.5% had bacteremia, 1.5% had UTI. The most common organisms were Acinetobacter (34%), Pseudomonas aeruginosa (23%), Escherichia coli (15%).

13) **Akash deep**-2004- Clinical and microbiological profile of nosocomial infections in a paediatric intensive care unit. The rate of nosocomial infections was 27%. The incidence of urinary, respiratory, blood stream infections were 56%, 34%, 10%. Klebsiella was the most common organism.

14) **Mehta. A.**-2007- Device associated Nosocomial infection rates in intensive care units in seven cities. Health care associated infection occurred in 9%. Blood stream associated infection occurred in 7.92 per 1000 ICU days. VAP occurred in 9 per 1000 ICU days. UTI occurred in 1.4 per 1000 ICU days.

15) **Mohamed saleem**-2012-Prevalence of nosocomial infections in surgical wards in a tertiary care hospital in lucknow. 20% had nosocomial infection. Older patients had increased infection than younger age. *Escherichia coli* was the most common organism followed by *staphylococcus* and *acinetobacter*, *pseudomonas aeruginosa*, *klebsiella*.

16) **Umesh.S.Kamat**-2009-Epidemiology of hospital acquired Urinary tract infection in a medical college hospital in Goa. Overall infection rate was 8%. 33% of catheterised patients had UTI. *E.Coli*, *pseudomonas*, *klebsiella*, *candida* were the organisms responsible.

STUDY JUSTIFICATION

Almost all patients admitted in Intensive Medical Care Unit (IMCU) of our hospital is in critical condition. Many patients develop nosocomial infection. The causative agents and risk factors vary in each IMCU. Nosocomial infection increases morbidity and mortality in critically ill Patients. This increases the duration of stay, need for prolonged antibiotic administration and increased utilisation of hospital resources. Many studies on nosocomial infection had been done in western countries. There has been limited data from developing countries especially India. There are no prior studies from our institute on nosocomial infection. Hence it was decided to study the incidence and etiological agents of nosocomial infection in our IMCU. The results of the study will be helpful in finding the etiological agents and help in formulating antibiotic policy for nosocomial infection. This can reduce irrational use of antibiotics and subsequently prevent colonisation of multidrug resistant organisms.

MATERIALS AND METHODS

Materials and methods:

Selection criterion:

All patients admitted in **IMCU** in Tirunelveli medical college hospital for more than 48 hours.

Total number of patients under study- 200

Period of Study:

All the patients admitted as inpatients in Intensive medical care unit in Tirunelveli Medical College Hospital, during the period of September 2012 – November 2013 were included in this study.

STUDY DESIGN:

Prospective study.

Geographic distribution:

Geographic distribution of the patients were predominantly from rural areas of Tirunelveli, Tenkasi, Tuticorin Districts.

Exclusion criterion:

- All patients admitted in IMCU for less than 48 hours.
- Patients with evidence of sepsis at admission.
- Patients with proven pre-existing infection.

Limitation of the Study:

1. Repeated cultures could not be performed in IMCU as patients were shifted to medicine ward.

Selection and study of this patients were done as mentioned in the proforma.

METHODOLOGY:

All the patients were asked a thorough and detailed history and general and systemic examination were done. Incidence, rate of infection, also known as cumulative incidence rate method, is to measure the frequency of new cases of nosocomial infections occurred in a given time. Since the measurement data and the method required are easy to be collected and calculated, it was widely used by many articles.^[21,22,23] However, the weakness of this method is that it does not consider the time of hospitalization as well as some other risk factors that would influence the incidence rate. As indicated by researches, the Nosocomial infection incidence rate was nearly zero in the first day of admission, significantly increased after 1 week's stay, peaked during 4 to 7 weeks' stay, then dropped as the time went on.^[24,25]

After careful clinical examination of the patients all were submitted to the following investigations.

I. BASIC LAB INVESTIGATIONS:

- a) Complete blood count.
- b) Blood-Sugar, Urea, Creatinine.
- c) Liver function tests
- d) Urine analysis.

II. CULTURE

- URINE
- SPUTUM
- BLOOD
- STOOL

All the culture samples were delivered to laboratory in a sterile manner immediately.

III. CHEST X RAY.

Collection of samples:

URINE:

10 to 20 ml of mid-stream urine was collected in a sterile, dry, clean, wide necked bottle by explaining to the patient to avoid contamination i.e a clean catch sample. The bottles were labelled with name, date and time of collection of sample. The sample was sent to the lab immediately and processed.

From catheterized patients urine sample was collected by disinfecting the wall of catheter at its juncture with the drainage tube. Urine was aspirated from a sterile disposable syringe.

BLOOD:

For blood culture the veni puncture site was washed with soap,rinsed with sterile water,cleaned with a swab of 70% alcohol and then dried.Blood was drawn with sterile 10 ml syringe and transferred to blood culture bottle containing thioglycate broth,tryptic soya broth and the bottles were gently rotated to ensure mixing of blood with the broth.The whole procedure is done in an aseptic manner to avoid contamination.Blood was drawn from two separate sites and two samples were sent.Catheter tip was also sent in a culture bottle.

SPUTUM:

Sputum was collected after the patient rinses his mouth with sterile distilled water to remove excessive saliva and food debris.The patients were asked to cough deeply and the expectorated sputum was collected in a sterilized screw capped open mouth containers.The suction material from the endotracheal tube was also collected in a sterile container and sent to the lab immediately. Endotracheal tube tip was also sent for culture.

RESULTS AND OBSERVATIONS

Statistical analysis:

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Yate's corrected chi square test was used to test the significance of difference between qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS AND OBSERVATION

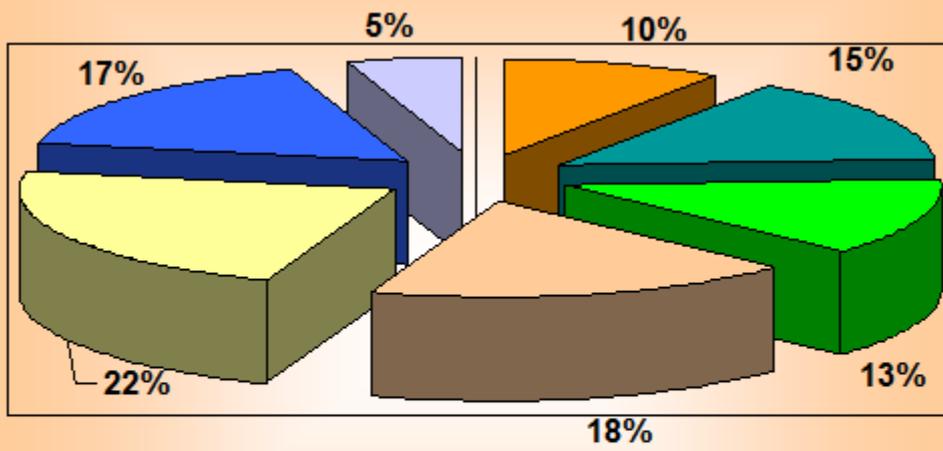
I. Relation between age and infection:

The total number of patients included in the study was 200 during the period of 2012 – 2013 in Tirunelveli Medical College and Hospital.

The Age and infection distributions were compiled in tabular columns as follows:

Age group	Cases	
	No	%
Up to 20 years	19	9.5
21-30 years	29	14.5
31-40 years	26	13.0
41-50 years	37	18.5
51-60 years	45	22.5
61-70 years	34	17.0
>70 yrs	10	5.0
Total	200	100
Range	13 - 80 years	
Mean	46.0 years	
SD	17.3 years	

The above table describes the number of patients admitted in relation with age of patient. The mean age of admission of patients in IMCU was 46 years. 37% of patients were below 40 years of age and 63% of patients were above 40 years. 19 patients were below 20 years of age, 29 patients were between 21 to 30 years of age, 26 patients were between 31 to 40 years of age, 37 patients were between 41 to 50 years of age, 45 patients were between 51 to 60 years age, 34 patients were between 61 to 70 years and 10 patients were more than 70 years of age. The most number of patients admitted were in the age group of 51 to 60 years of age -22.5%. As in all medical wards old age people are admitted more than younger people in IMCU. This is because old age people have decreased immunity, associated diseases like diabetes, hypertension, Coronary heart disease, Carcinoma. Also they may be smokers and alcoholics. The rate of recovery is good in young patients compared to old age patients. Our study reveals that more patients were admitted to our hospital IMCU above 40 years of age than younger patients.



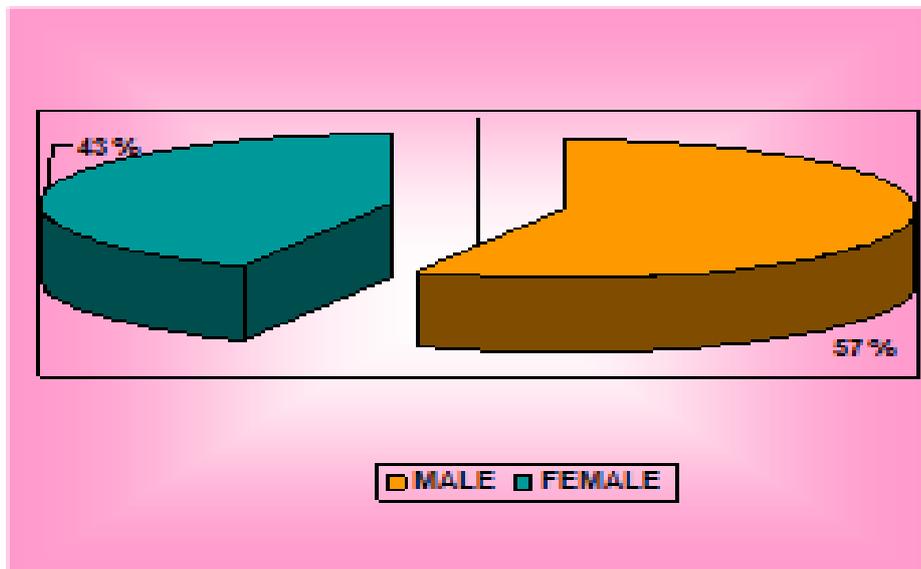
II Sex distribution

The total number of patients included in the study was 200 during the period of 2012 – 2013 in Tirunelveli Medical College and Hospital. 114 were male and 86 were female patients. The Age, Sex distributions were compiled in tabular column as follows:

Sex	Cases	
	No	%
Male	114	57
Female	86	43
Total	200	100

Total male patients were 114 and female patients were 86 in this study. 57% of the patients were male and 43% were female. More male patients are admitted in IMCU than female patients.

SEX DISTRIBUTION

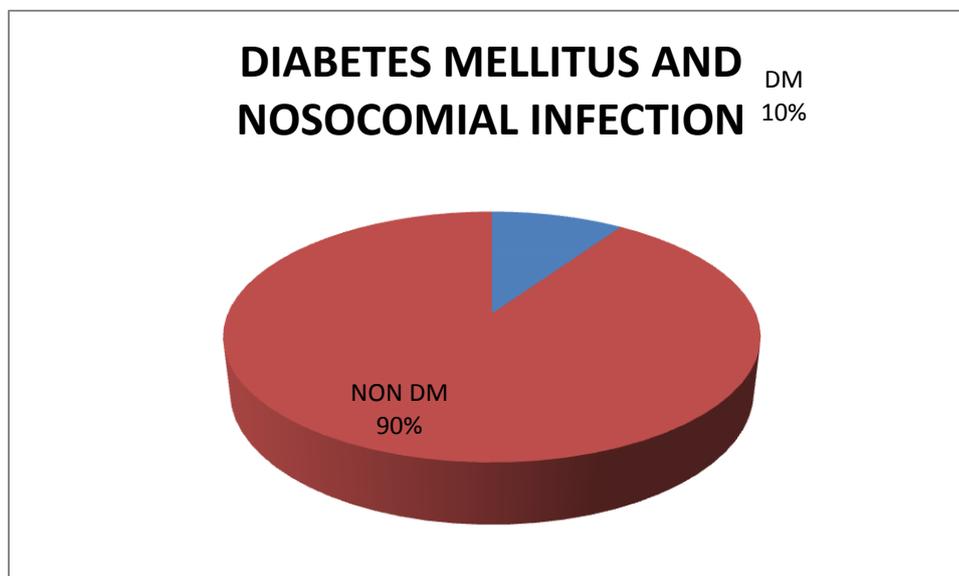


SIGNIFICANCE OF RISK FACTORS

Diabetes mellitus and Nosocomial infection

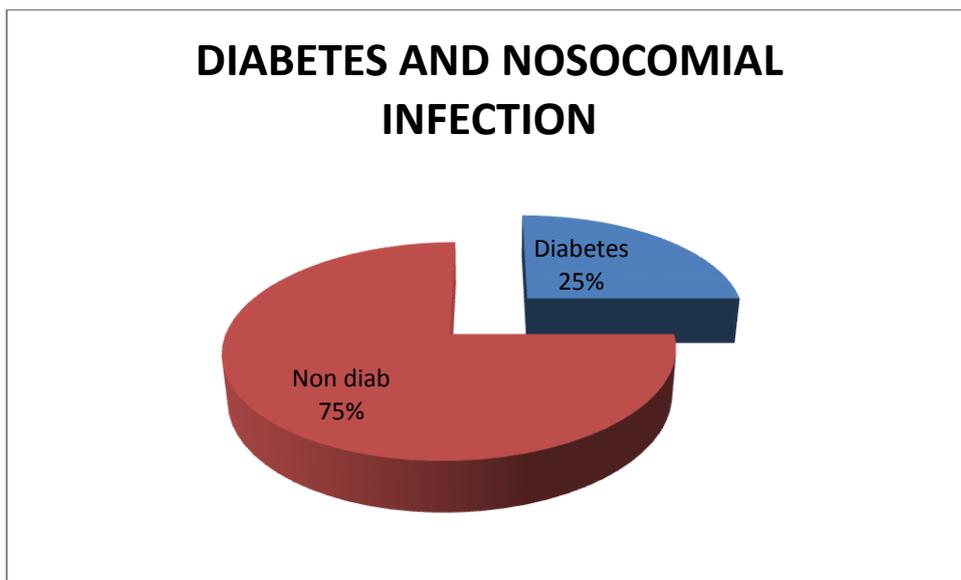
Out of the 200 patients in our study 30 patients had previous history of hypertension and 42 patients had previous history of diabetes out of which 12 patients had both.

Column1	Column2
DIABETES	42
HYPERTENSION	30
BOTH DIABETES,HT	12
TOTAL PATIENTS	200



RISK FACTOR	NUMBER OF PATIENTS
DM	4
NON DM	38
TOTAL	42

Out of 42 diabetic patients 4 had nosocomial infection which is around 10%.



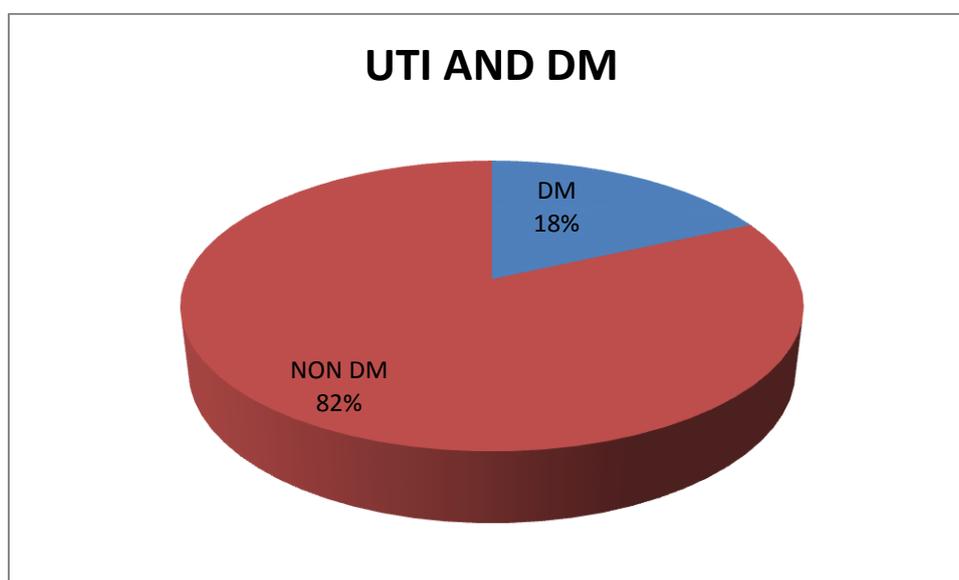
This picture shows that out of 16 patients with nosocomial infection 4 were diabetic i.e 25% were diabetic.

RISK FACTOR	NUMBER OF PATIENTS
DM	4
NON DM	12
TOTAL	16

Relation between UTI and Diabetes Mellitus

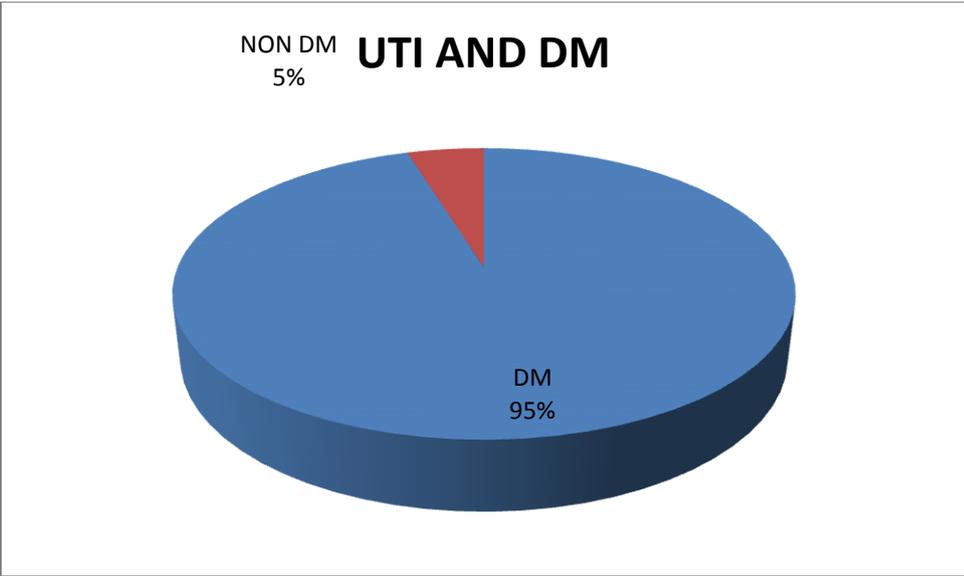
RISK FACTOR	NUMBER OF PATIENTS
DM	2
NON DM	9
TOTAL	11

In our study there were 11 cases of Urinary tract nosocomial infection. Out of these only 2 patients were diabetic. This amounts to 18% of the patients with UTI.



RISK FACTOR	NUMBER OF PATIENTS
DM	2
NON DM	40
TOTAL	42

Out of 42 Diabetics only 2 patients had UTI. This is 5% of the patients with UTI.

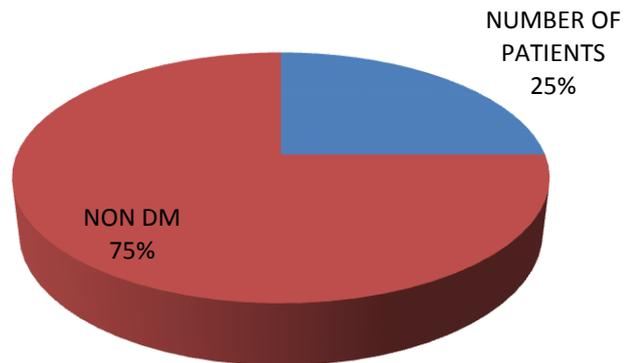


Respiratory Nosocomial infection and Diabetes Mellitus.

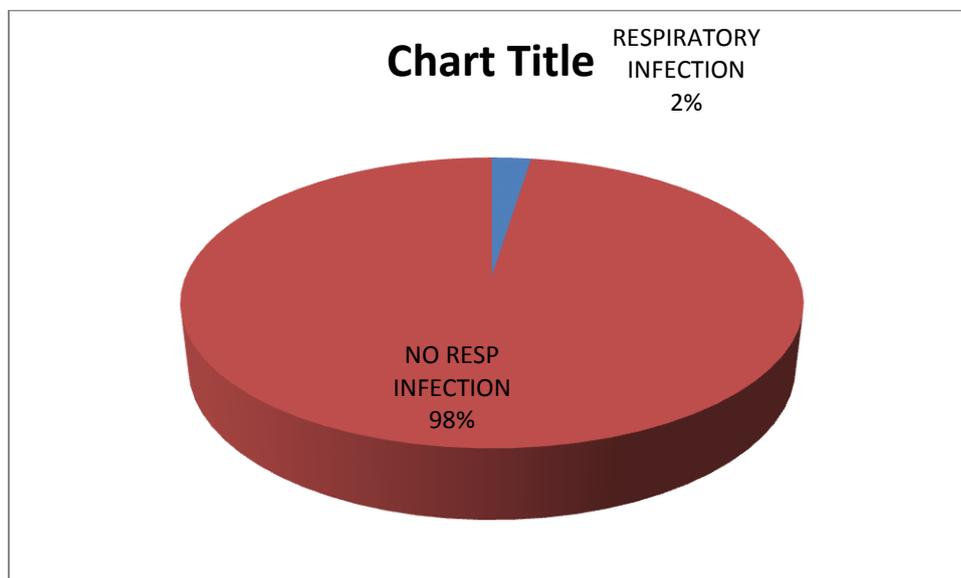
RISK FACTOR	NUMBER OF PATIENTS
DM	1
NON DM	3
TOTAL	4

There were 4 patients out of 200 who had respiratory tract infection. Out of these 4 patients only one patient had Diabetes mellitus. So 25% of the patients with respiratory infection had Diabetes Mellitus.

RESPIRATORY INFECTION AND DIABETES MELLITUS



Respiratory infection and Diabetes mellitus.

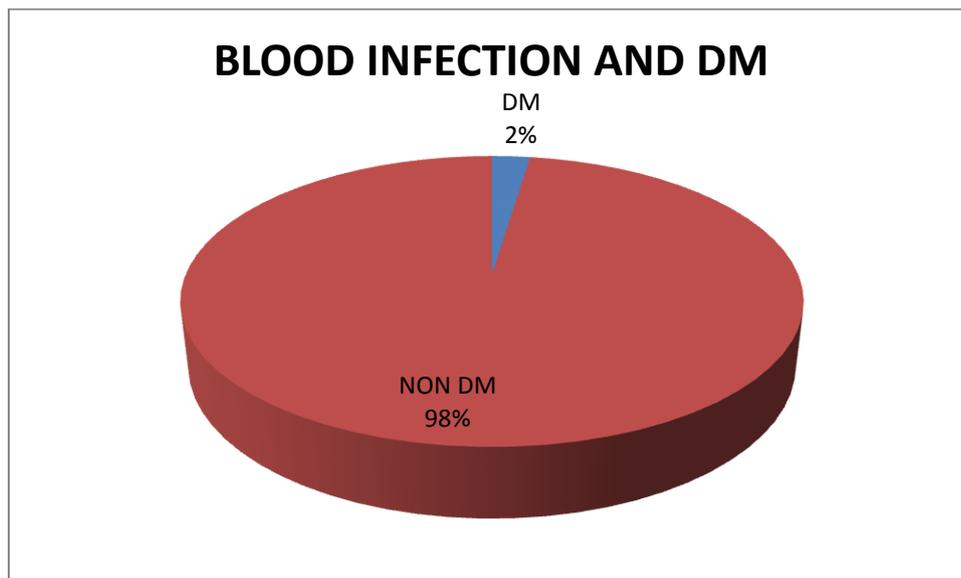


In this study 42 diabetic patients were admitted in IMCU. But only one had respiratory infection. This amounts to 2%.

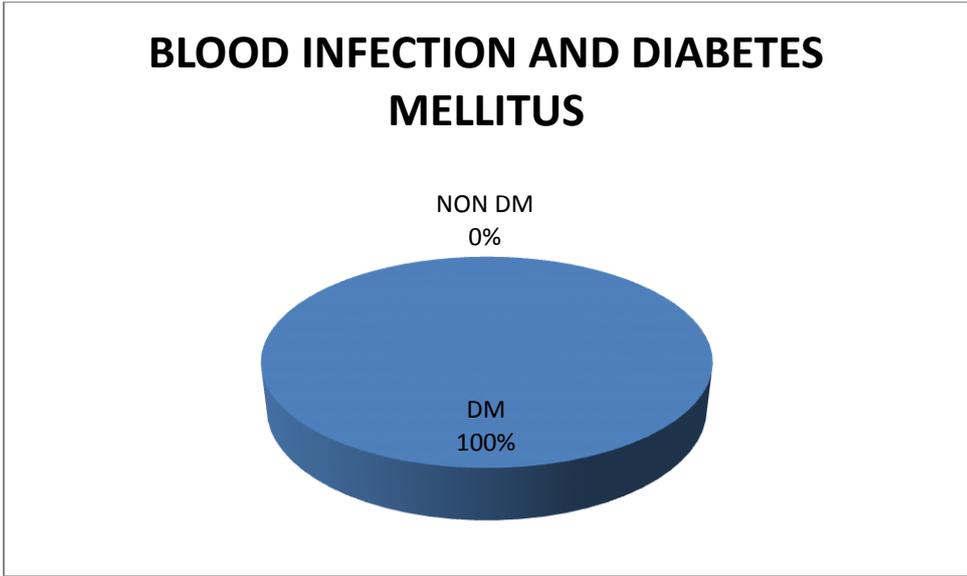
INFECTION	NUMBER OF PATIENTS
RESPIRATORY INFECTION	1
NO RESP INFECTION	41
TOTAL	42

BLOOD STREAM INFECTION AND DIABETES MELLITUS

INFECTION	NUMBER OF PATIENTS
BLOOD INFECTION	1
NO BLOOD INFECTION	41
TOTAL	42



Out of 42 diabetic patients only one had blood stream infection. This amounts to 2% of the infection.



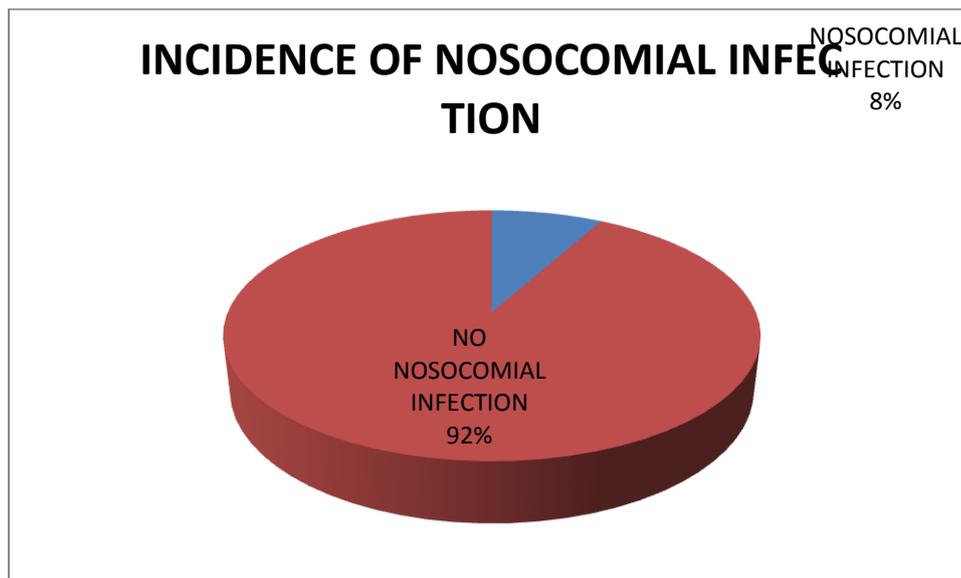
RISK FACTOR	NUMBER OF PATIENTS
DM	1
NON DM	0
TOTAL	1

In this study only one patient had Blood stream infection who was a diabetic.

INCIDENCE OF NOSOCOMIAL INFECTIONS

Table 3 : Incidence of Nosocomial infections

Parameter	Value
Total ICU admissions during study period	200
Number of nosocomial infections	16
Incidence of nosocomial infection	8%



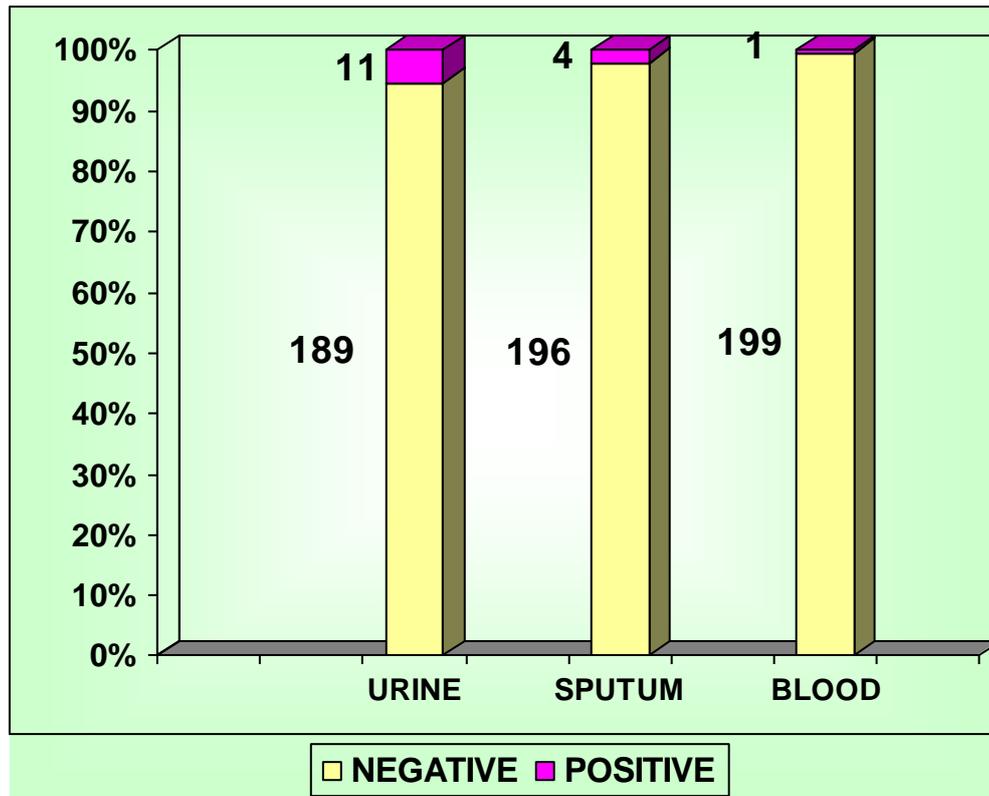
Out of 20 patients 16 patients had nosocomial infection which amounts to incidence of 8%.

Table 5 : Nosocomial infections as per various cultures

11 patients had urinary tract infection.4 patients had hospital acquired pneumonia and only 1 patient had blood stream infection.5.5% patients had urinary infection,2% patients had sputum infection and 0.5% patients had blood infection.

Culture	Positive cases		Negative cases	
	No	%	No	%
Urine	11	5.5	189	94.5
Sputum	4	2.0	196	98.0
Blood	1	0.5	199	99.5
Total	16	8.0	184	92.0
'p' value between Urine & sputum culture Urine & blood Sputum & blood culture	0.1143 Not significant 0.0083 Significant 0.1859 Not significant			

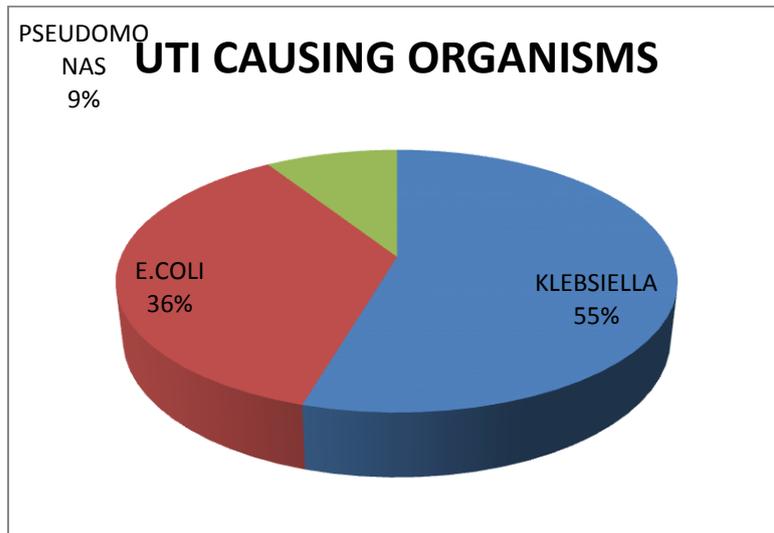
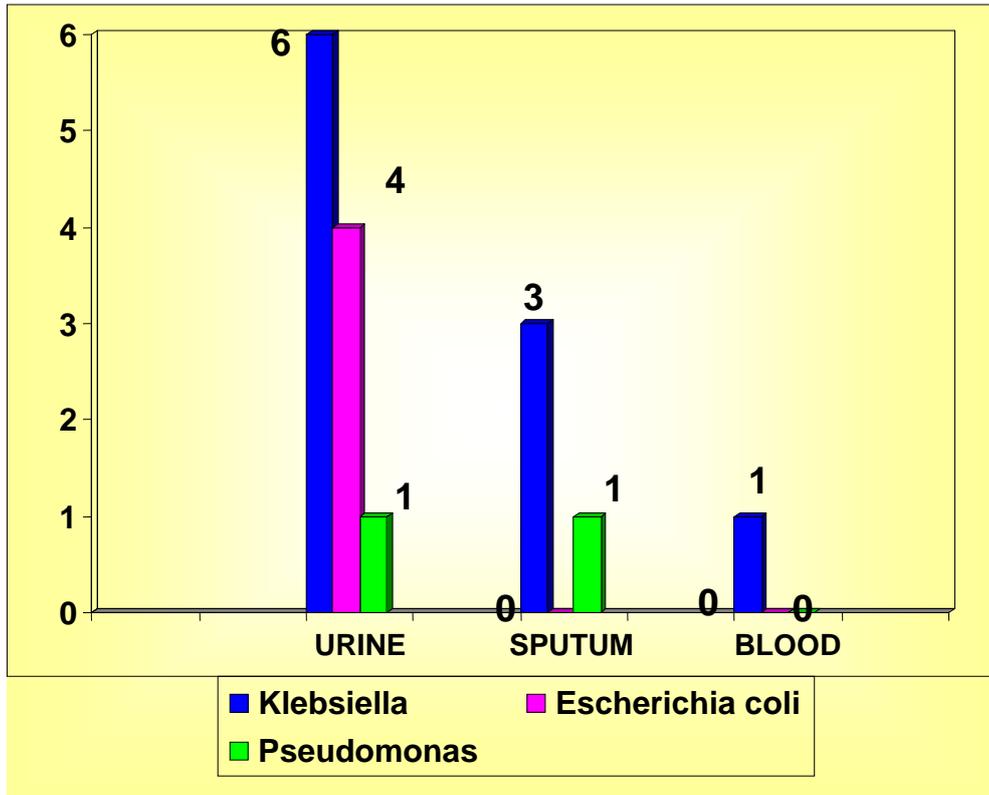
NOSOCOMICAL INFECTION AS PER VARIOUS CULTURES



ORGANISMS IN CULTURES

Urinary tract infection was caused by Klebsiella in 6 patients, E. Coli in 4 patients and pseudomonas in 1 patient. Hospital acquired pneumonia was seen in 4 patients out of which 3 were due to klebsiella and 1 due to pseudomonas. Blood stream infection was seen in 1 patient only which was caused by klebsiella. 5.5% patients had urinary tract infection, 2% patients had hospital acquired pneumonia and only 0.5% patient had hospital acquired blood stream infection. The most common organism causing nosocomial infection is Klebsiella followed by E. Coli.

ORGANISMS IN VARIOUS CULTURES



Klebsiella caused 55% ,E.coli caused 36% and Pseudomonas caused 9% of UTI.

ORGNISMS CAUSING RESPIRATORY INFECTION

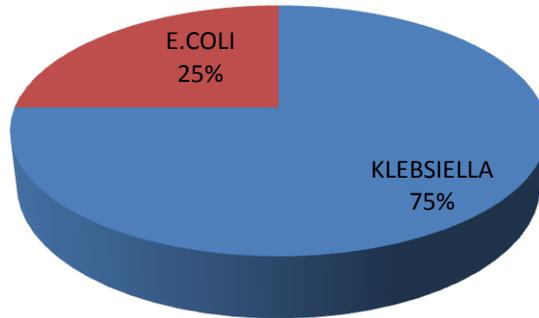


Table 4 : Organisms in various cultures

Organisms	Positive cases in							
	Urine culture		Sputum culture		Blood		Total	
	No	%	No	%	No	%	No	%
Klebsiella	6	3.0	3	1.5	1	0.5	10	5.0
Escherichia coli	4	2.0	-	-	-	-	4	2.0
Pseudomonos Aeuroginosa	1	0.5	1	0.5	-	-	2	1.0
Total positive	11	5.5	4	2.0	1	0.5	16	8.0
Negative	189	94.5	196	98.0	199	99.5	184	92.0

ORGANISMS CAUSING NOSOCOMIAL INFECTION

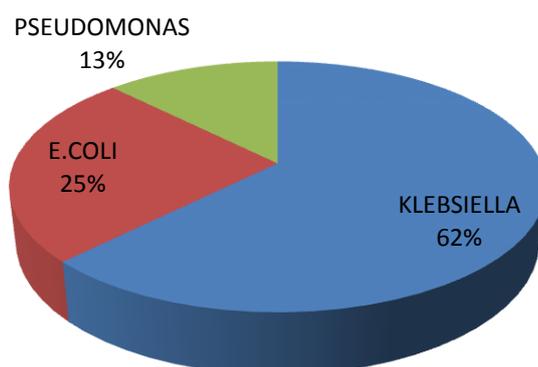


Table 6 : Nosocomial infections as per various organisms

Organisms	Positive cases		Negative cases	
	No	%	No	%
Klebsiella	10	5	190	95
Escherichia coli	4	2	196	98
Pseudomonos Aeuroginosa	2	1	198	99
Total	16	8	184	92
'p' value between				
Klebsiella and E.Coli	0.1737 Not significant			
Klebsiella and Pseudomonas	0.0402 Significant			
E.Coli and Pesudomonas	0.3426 Not significant			

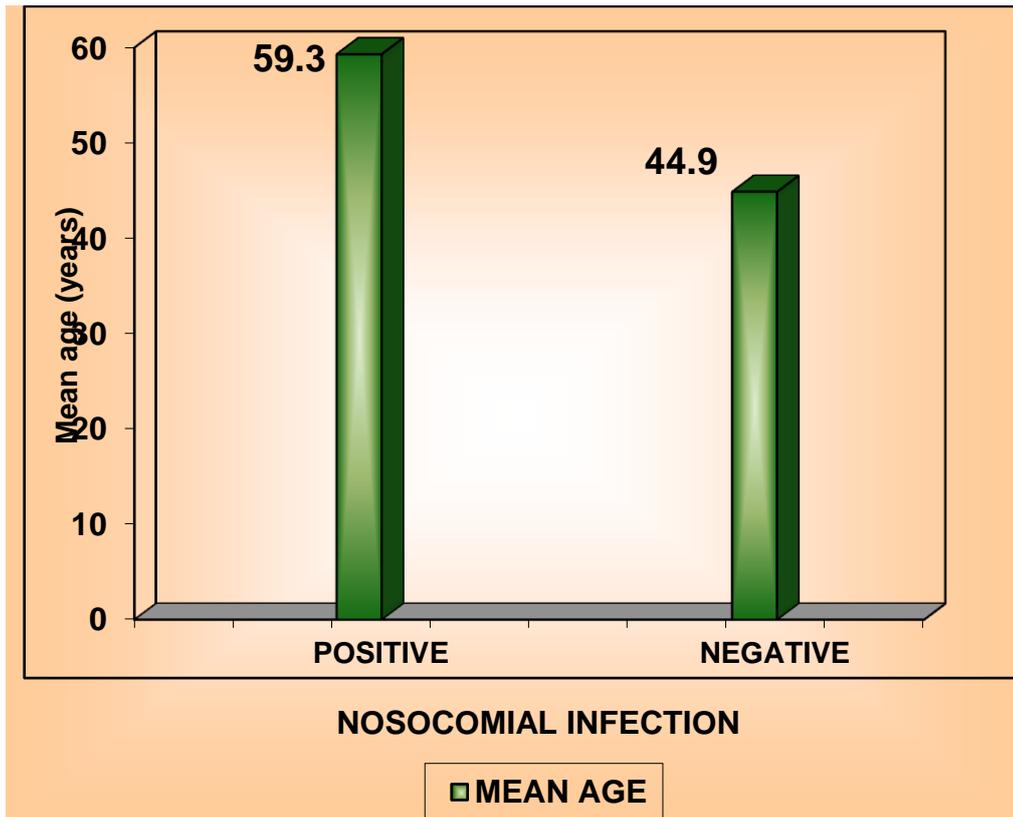
10 patients had infection with Klebsiella, 4 had E.Coli infection and only 2 had infection with Pseudomonas. Klebsiella was responsible for 62% of hospital acquired infection, Escherichia coli was responsible for 25% of infections and pseudomonas aeruginosa was responsible for 13% of infection.

Age and Nosocomial infection

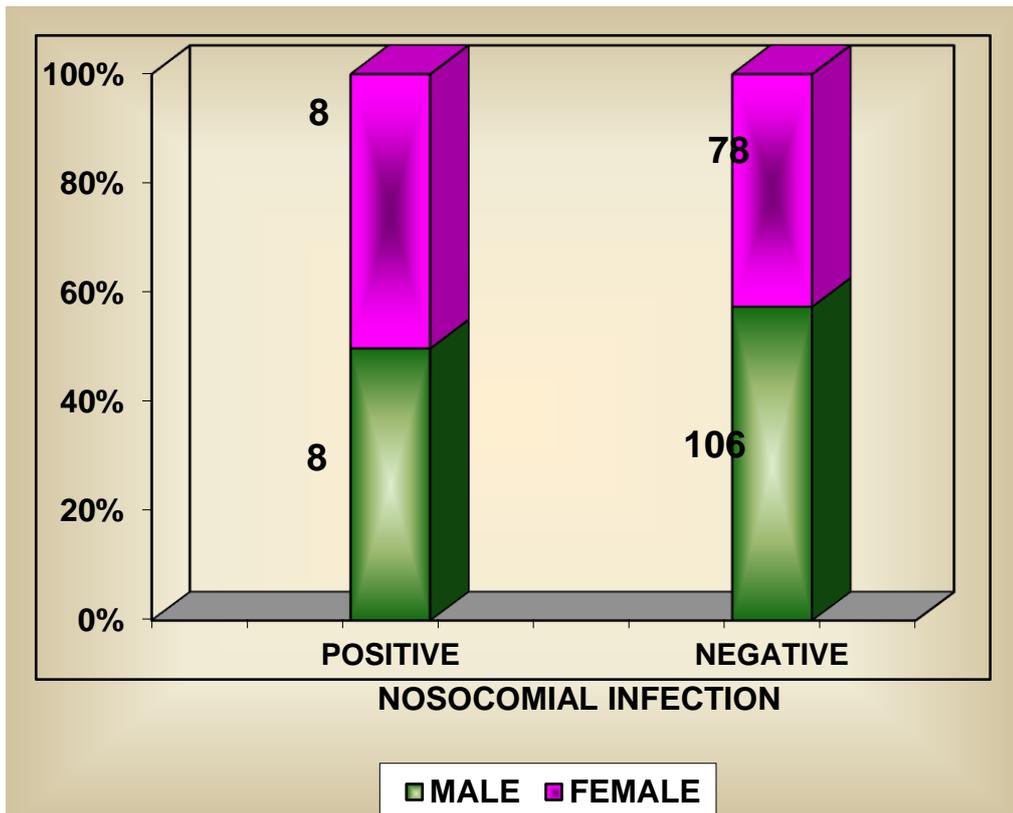
TABLE 7

Nosocomial infection	Age in years		
	Range	Mean	SD
Positive	41 – 80	59.3	9.6
Negative	13 – 79	44.9	17.3
'p'	0.0011 Significant		

All 16 infections were seen in patients aged more than 40 years of age. None of the infection was seen in patients below 40 years of age. The mean age of the patient affected with Nosocomial infection was 59.3 years. Age was a significant factor in the incidence of Nosocomial infection as per this study. The p value was 0.0011 which was highly significant.



SEX AND NOSOCOMIAL INFECTION

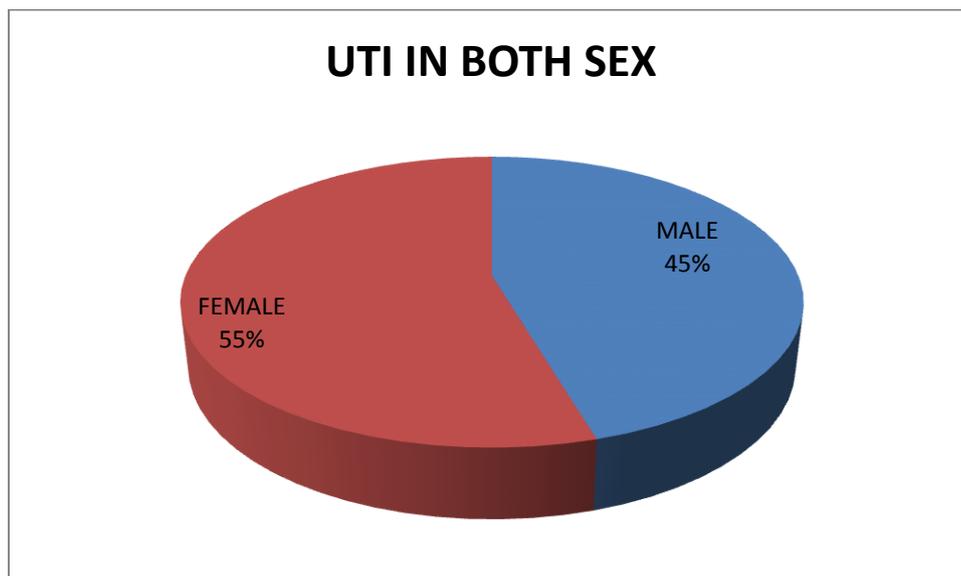


Sex and Nosocomial infection

TABLE 8

Sex	Nosocomial infection			
	Positive		Negative	
	No	%	No	%
Male (114)	8	7.0	106	93
Female (86)	8	9.3	78	90.7
'p'	0.7441 Not significant			

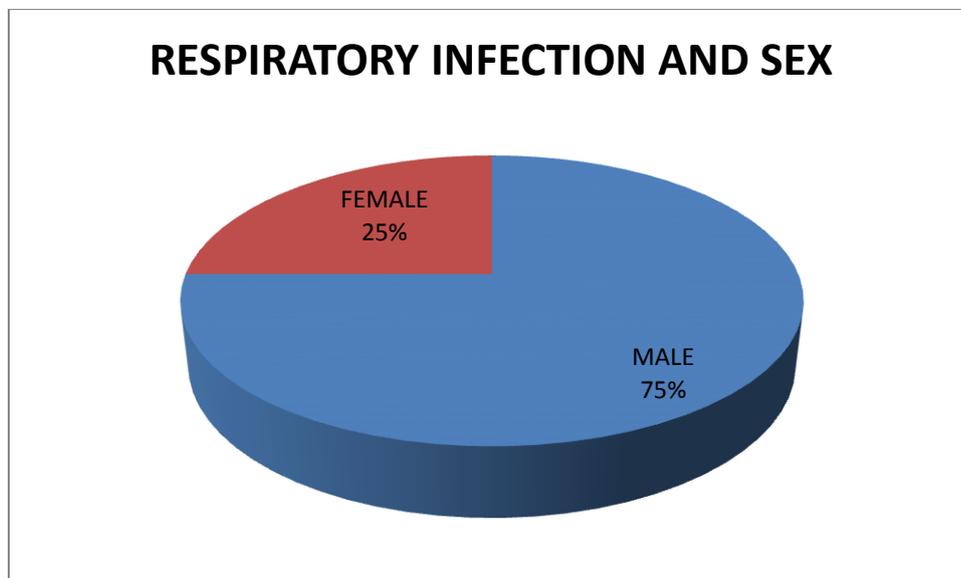
There was equal distribution of infection between male and female patients. 8 male and 8 female patients had nosocomial infection. Male patients were affected in 7% cases and female patients in 9.3% cases. There was no statistically significant difference in infection between both sex.



UTI was present in 6 females and 5 males. So the UTI was seen in 55% of females and 45% of males. So there was no significant difference in UTI between male and female.

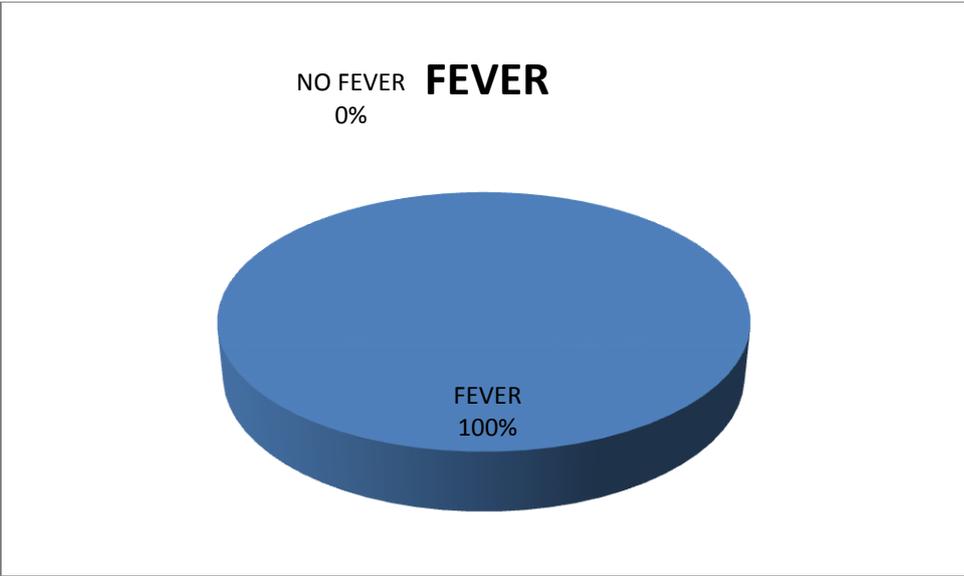
RESPIRATORY INFECTION IN BOTH SEX

Respiratory infection was seen in 4 patients of which 3 were male and only one female. But the significance of difference between both sex could not be ascertained as the total number of patients with respiratory infection is less.



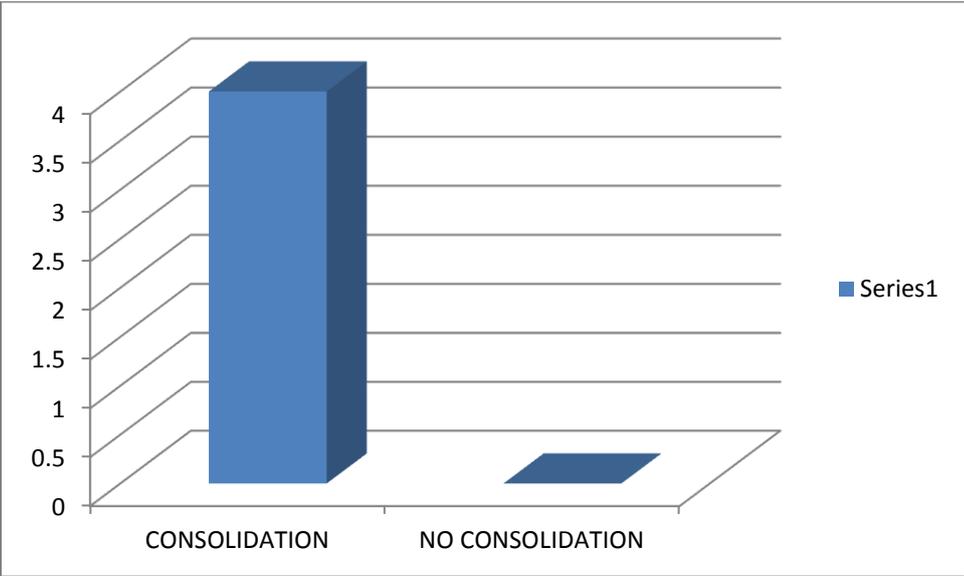
RELATION BETWEEN FEVER AND NOSOCOMIAL INFECTION.

All the 16 patients with nosocomial infection developed fever. So 100% of patients with Nosocomial infection developed fever.

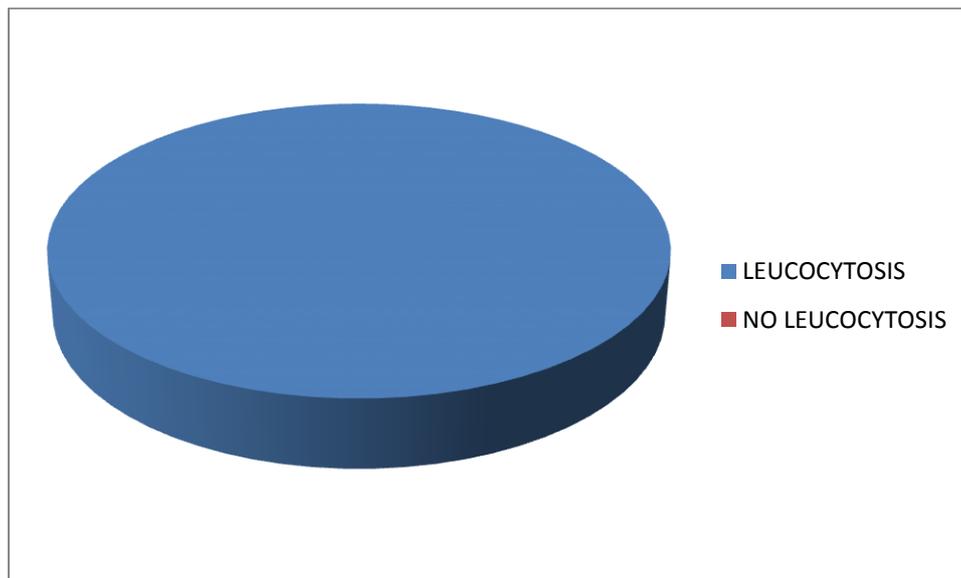


CONSOLIDATION IN CHEST XRAY AND RESPIRATORY NOSOCOMIAL INFECTION

All 4 patients with Respiratory Nosocomial infection had consolidation in CXR.



RELATION BETWEEN LEUCOCYTOSIS AND NOSOCOMIAL INFECTION.



All 16 patients with Nosocomial infection had Leucocytosis.

DISCUSSION

The incidence of Nosocomial infection in this study is 8%.The commonest infection was Urinary tract infection followed by respiratory tract infection and blood stream infection. Klebsiella was the most common organism followed by Escherichia coli and pseudomonas aeruginosa. Urinary tract infection was caused mostly by Klebsiella and Escherichia coli and pseudomonas. Respiratory infection was caused by Klebsiella and pseudomonas.Blood stream infection was caused by Klebsiella.

The age of patients admitted in IMCU was between 13 years and 85 years.Most patients were above 40 years of age i.e 63% of patients.The most comon age group was between 40 to 50 years i.e 22.5%.All nosocomial infections were in patients above 40 years old and it is statistically significant.In this study age had a significant relation to Nosocomial infection.

In this study nosocomial infection was equally distributed between male and female.There was no statistically significant correlation between sex and Nosocomial infection.

In this study out of the 16 patients with nosocomial infection 4 had diabetes mellitus i.e. 25% of the patients with nosocomial infection had diabetes mellitus.Urinary tract infection was seen in 2 diabetics and 9 non-diabetics.i.e 18% were diabetic and the remaining 82% were non diabetic.

Out of the 4 patients with respiratory infection one was a diabetic i.e 25% were diabetic and 75% non diabetic. Blood stream infection was seen in only one patient who was diabetic.

Relation with the AIM of the study:

The results in this study falls within the average range of infection in India.As per this study Urinary tract infection is the commonest nosocomial infection in Intensive Medical Care Unit.Diabetes mellitus is seen in 25% of patients with Nosocomial infection But out of 42 patients with diabetes only 4 had Nosocomial infection which was 10% only.

LIMITATIONS AND STRENGTHS:

This study has some limitations.All patients admitted in IMCU could not be included in the study because most of the patients were shifted out of the IMCU before 48 hours of admission.Sample could be obtained only one time as most of the patients were shifted to the medical ward from IMCU.Empirical antibiotics were given to all patients admitted in our IMCU.This is the reason for low incidence of Nosocomial infection in our study.The relation between duration of stay in IMCU and the incidence could not be calculated.

The sample size in this study is 200 which is relatively a large sample.The study was conducted thought the year.So there is less chance for seasonal variation in this study.

Comparison with Other studies:

Patients in IMCU are critically ill and they are more susceptible to nosocomial infection. They need invasive procedures and are in frequent contact with the health-care workers. They have disruption of barriers to infection due to endotracheal intubation, tracheostomy, urinary bladder catheterization, central venous catheterization.^[39] So patients may get highly resistant infections.

As per previous studies the most common nosocomial infection in medical ICU is urinary tract infection, pneumonia and blood stream infection.^[41] Urinary tract infection was the commonest infection in our study. The source of urinary tract infection was placement of Foleys catheter.

Richards and co reported in the national nosocomial infections surveillance system (NNIS) that 20 to 30% of nosocomial infections were due to urinary tract infection.^[41,42,43] Roser and colleagues suggested that age (>50 years) and catheterization were independent risk factors for the development of urinary tract infection. Finkelstein and co reported an incidence of 10 to 14% in 337 patients in a single Israeli ICU.^[45]

In our study urinary tract infection was 5.5%. Of these all patients were more than 40 years old. So there is a significant correlation between age and UTI. Out of the 11 patients with UTI 2 patients were known diabetic and 9 were non diabetic. In previous studies diabetic patients have slightly higher

incidence of UTI than nondiabetic patients. In our study the number of diabetic patients having UTI are less due to starting empirical antibiotics on the day of admission itself, following aseptic care during catheterization and following the correct precautions to prevent infection.

As per previous studies Nosocomial pneumonia is the second most common cause of nosocomial infection in ICU. It is the most common cause of death from infection acquired in hospital.^[47] More than 90% of patients of nosocomial pneumonia occur in patients on mechanical ventilation. More than 50% of ventilator associated pneumonia cases occur within first 4 days after intubation.^[48] Frequency of pneumonia was between 2 to 25% in different studies.^[49] In our study the incidence of pneumonia is 2%. Klebsiella was the cause in 3 cases and pseudomonas was the cause in 1 case. The low incidence of pneumonia in this study may be due to use of empirical antibiotics, use of antiseptic procedures during intubation, Good hand hygiene of hospital workers and regular suctioning of the endotracheal tube.

Blood stream infection is a common nosocomial infection in IMCU. Central venous catheterization is the most common cause for the infection. In US more than 30000 deaths annually are due to central venous catheterization.^[44] As per study by Pfaller, Jones in United States and Canada Klebsiella was the most common organism.^[50] Study by Marra, Wey also suggested that bloodstream infection with extended spectrum beta-lactamase producing Klebsiella pneumonia affected the clinical outcome in patients

admitted in ICU.^[51] In a prospective study in US in 24000 patients over a 7 year period 65% of infections were due to gram positive organisms and 25% of infections were due to gram negative organisms. Incidence of Blood stream nosocomial infection in the present study is 0.5% only. This is due to use of empirical antibiotics, following strict aseptic precautions during insertion and removal of catheters.

CONCLUSION

- The incidence of nosocomial infection in patients admitted in Intensive Medical Care Unit was 8% in the study population.
- The commonest infection was Urinary tract infection followed by respiratory tract infection and blood stream infection.
- Gram negative bacteria especially Klebsiella was the predominant organism.
- Age was a significant factor in this study. All patients who had Nosocomial infection were above 40 years old. Old age patients were more susceptible than young patients.
- There was no significant difference in incidence between male and female patients.

FUTURE STUDIES:

The future studies on Nosocomial infection in our college Hospital may focus on:

- Antibiotic resistance pattern among the causative agents for Nosocomial infection.
- Correlation between duration of stay and incidence of infection.
- Underlying Risk Factors.

BIBLIOGRAPHY

1. Mahieu LM, De Muynck AO, Ieven MM, De Dooy JJ, Goossens HJ, Van Reempts PJ. Risk factors for central vascular catheter-associated Blood stream infections among patients in a neonatal intensive care unit. *J Hosp Infect.* Jun 2001;48(2):108-16.
2. Hospital Infections Program, National Center for Infectious Diseases, CDC. Public Health Focus: surveillance, prevention, and control of nosocomial infections. *MMWR.* October 1992;41(42):783-787.
3. Hughes JM. Study on the efficacy of nosocomial infection control (SENIC Project): results and implications for the future. *Chemotherapy.* 1988;34(6):553-61.
4. Edwards JR, Peterson KD, Andrus ML, Dudeck MA, Pollock DA, Horan TC. National Healthcare Safety Network (NHSN) Report, data summary for 2006 through 2007, issued November 2008. *Am J Infect Control.* Nov 2008;36(9):609-26.
5. Wenzel RP, Edmond MB. The impact of hospital-acquired bloodstream infections. *Emerg Infect Dis.* Mar-Apr 2001;7(2):174-7.
6. Tikhomirov E. WHO programme for the control of hospital infections. *Chemioterapia.* June 1987;6(3):148-51.
7. Rosenthal VD, Maki DG, Mehta A, Alvarez-Moreno C, Leblebicioglu H, Higuera F. International Nosocomial Infection Control Consortium

- report, data summary for 2002-2007, issued January 2008. *Am J Infect Control*. Nov 2008;36(9):627-37.
- 8 Aiken AM, Mturi N, Njuguna P, Mohammed S, Berkley JA, Mwangi I, et al. Risk and causes of paediatric hospital-acquired bacteraemia in Kilifi District Hospital, Kenya: a prospective cohort study. *Lancet*. Dec 10 2011;378(9808):2021-7.
- 9 Gastmeier P, Geffers C, Brandt C, Zuschneid I, Sohr D, Schwab F. Effectiveness of a nationwide nosocomial infection surveillance system for reducing nosocomial infections. *J Hosp Infect*. Sep 2006;64(1):16-22.
- 10 Klevens RM, Edwards JR, Richards CL, et al. Estimating healthcare-associated infections in US hospitals, 2002. *Public Health Rep*. Mar 2007;122(2):160-6.
- 11 Scott RD. The direct medical costs of healthcare-associated infections in US hospitals and the benefits of prevention, 2008. CDC. Available at http://www.cdc.gov/ncidod/dhqp/pdf/Scott_CostPaper.pdf. Accessed 7/1/2009.
12. Richards MJ, Edwards JR, Culver DH, Gaynes RP. Nosocomial infections in pediatric intensive care units in the United States. National Nosocomial Infections Surveillance System. *Pediatrics*. Apr 1999;103(4):e39.
13. Grohskopf LA, Sinkowitz-Cochran RL, Garrett DO, et al. A national

- point-prevalence survey of pediatric intensive care unit-acquired infections in the United States. *J Pediatr.* Apr 2002;140(4):432-8.
14. Sohn AH, Garrett DO, Sinkowitz-Cochran RL, Grohskopf LA, Levine GL, Stover BH. Prevalence of nosocomial infections in neonatal intensive care unit patients: Results from the first national point-prevalence survey. *J Pediatr.* Dec 2001;139(6):821-7.
 15. Edward AM, Warren DK, Fraser VJ. Ventilator-associated pneumonia in pediatric intensive care unit patients: risk factors and outcomes. *Pediatrics.* May 2002;109(5):758-64.
 16. Fayon MJ, Tucci M, Lacroix J, et al. Nosocomial pneumonia and tracheitis in a pediatric intensive care unit: a prospective study. *Am J Respir Crit Care Med.* Jan 1997;155(1):162-9.
 17. Zaoutis TE, Coffin SE. Clinical Syndromes of Device-Associated Infections. In: Long SS, Pickering LK, Prober CG. *Principles and Practice of Pediatric Infectious Diseases.* 3rd ed. Churchill Livingstone; 2008:chap 102.
 18. Chastre J, Wolff M, Fagon JY, et al. Comparison of 8 vs 15 days of antibiotic therapy for ventilator-associated pneumonia in adults: a randomized trial. *JAMA.* Nov 19 2003;290(19):2588-98.
 19. Collee JG, Fraser AG, Marmion BP, Simmons A. Culture M2A7. Vol 20, No1-2, National of Bacteria. In: Mackie McCartney Practical Medical Microbiology, 14th ed, (Churchill

- Livingstone,London), 1996: 113-129.
- 20 National Committee for Clinical Laboratory Standards:Performance standards for antimicrobial susceptibility testing. 8th Information Supplement M2A7. Vol 20, No1-2, National Committee for Clinical Laboratory Standards, Villanova, Pa.
 - 21 Tam AY, Yeung CY. The changing pattern of severe neonatal staphylococcal infection: a 10-yearstudy. *AustPaediatr J*. 1988;24(5):275-9. Epub 1988/10/01.
 - 22 Tseng YC, Chiu YC, Wang JH, Lin HC, Su BH, Chiu HH. Nosocomial bloodstream infection in a neonatal intensive care unit of a medical center: a three-year review. *J MicrobiolImmunol Infect*.2002;35(3):168-72. Epub 2002/10/17.
 - 23 Lachassinne E, Letamendia-Richard E, Gaudelus J. [Epidemiology of nosocomial infections in neonates]. *Archives de pediatrie :organe officiel de la Societe francaise de pediatrie*. 2004;11(3):229-33.Epub 2004/03/31. Epidemiologie des infections nosocomiales en neonatalogie.
 - 24 Freeman J, McGowan JE, Jr. Differential risk of nosocomial infection. *The American journal of medicine*. 1981;70(4):915-8. Epub 1981/04/01.
 - 25 Freeman J, McGowan JE, Jr. Methodologic issues in hospital

- epidemiology. III. Investigating the modifying effects of time and severity of underlying illness on estimates of cost of nosocomial infection. *Reviews of infectious diseases*. 1984;6(3):285-300. Epub 1984/05/01.
- 26 Sengupta A, Lehmann C, Diener-West M, Perl TM, Milstone AM. Catheter duration and risk of CLA-BSI in neonates with PICCs. *Pediatrics*. 2010;125(4):648-53. Epub 2010/03/17.
- 27 Butler-O'Hara M, D'Angio CT, Hoey H, Stevens TP. An evidence-based catheter bundle alters central venous catheter strategy in newborn infants. *The Journal of pediatrics*. 2012;160(6):972-7 e2. Epub 2012/01/14.
- 28 Hei MY, Zhang XC, Gao XY, Zhao LL, Wu ZX, Tian L, et al. Catheter-related infection and pathogens of umbilical venous catheterization in a neonatal intensive care unit in China. *American journal of perinatology*. 2012;29(2):107-14. Epub 2011/12/02.
- 29 van der Zwet WC, Kaiser AM, van Elburg RM, Berkhof J, Fetter WP, Parlevliet GA, et al. Nosocomial infections in a Dutch neonatal intensive care unit: surveillance study with definitions for infection specifically adapted for neonates. *J Hosp Infect*. 2005;61(4):300-11. Epub 2005/10/14.

- 30 Couto RC, Pedrosa TM, Tofani C de P, Pedroso ER. Risk factors for nosocomial infection in a neonatal intensive care unit. *Infect Control Hospital Epidemiology*. 2006;27(6):571-5. Epub 2006/06/07.
- 31 Teresa C. Horan, Mary Andrus and Margaret A. Dudeck. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. *Am J Infect Control* 2008; 36: 309-332.
- 32 Harley RW, Culver DH, White JW, et al. The efficacy of infection surveillance and control programmes in preventing Nosocomial infections in US hospitals. *Am J Epidemiol* 1985; 12: 182.
- 33 Cook DJ, Walter SD, Cook RJ, Brun-Buisson C, et al. Incidence of and risk factors for ventilator-associated pneumonia in critically ill patients. *Ann Intern Med* 1998; 129: 440.
- 34 Chastre J, Fagon JY. Ventilator-associated pneumonia. *Am J Respir Crit Care Med* 2002; 165: 867–903.
- 35 Celis R, Torres A, Gatell JM, Almela M, Rodriguez-Roisin R, Agusti-Vidal A. Nosocomial pneumonia: a multivariate analysis of risk and prognosis. *Chest* 1988; 93: 318–324.
- 36 Ferrer M, Valencia M, Torres A. Management of Ventilator associated pneumonia. In: Vincent JL. 2008 Year Book of Intensive Care and

- Emergency Medicine. Verlag Berlin Heidelberg: Springer, 2008;p.353–64. 3
- 37 Craven DE, Kunches LM, Lichtenberg DA, Kollisch NR, Barry MA, Heeren TC et al. Nosocomial infection and fatality in medical and surgical intensive care unit patients. *Arch Intern Med*, 1988;148(5):1161–8
- 38 Vincent JL, Bihari DJ, Suter PM, Bruining HA, White J, Nicolas-Chanoin MH, et al. The prevalence of nosocomial infection in intensive care units in Europe. Results of The European Prevalence of Infection in Intensive Care (EPIC) Study. EPIC International Advisory Committee. *JAMA*, 1995;274:639–44
- 39 Shannon SC. Chronic critical illness. In Jesse BH, Gregory AS, Lawrence DH, eds. *Principles of Critical Care*. 3rdEd, McGraw Hill, 2005;p. 207–15.
- 40 Tablan OC, Anderson LJ, Besser R, Bridges C, Hajjeh R. Guidelines for preventing health-care-associated pneumonia, 2003: Recommendations of the CDC and the Healthcare Infection Control Practice Advisory Committee. *MMWR Recomm Rep*, 2004;53:1–36

- 41 Richards MJ, Edwards JR, Culver DH, Gaynes RP. Nosocomial infections in combined medical-surgical intensive care units in the United States. *Infect Control Hosp Epidemiol*, 2000;21:510–5. 11.
- 42 Laupland KB, Zygun DA, Davies HD, Church DL, Louie TJ, Doig CJ. Incidence and risk factors for acquiring nosocomial urinary tract infection in the critically ill. *J Crit Care*, 2002;17:50–7.
- 43 Erbay H, Yalcin AN, Serin S, Turgut H, Tomatir E, Cetin B, et al. Nosocomial infections in intensive care unit in a Turkish university hospital: a 2-year survey. *Intensive Care Med*, 2003;29:1482–8.
- 44 O'Grady NP, Alexander M, Dellinger EP, Gerberding JL, Heard SO, Maki GD. Guidelines for the prevention of intravascular catheter related infections. *Infect Control Hosp Epidemiol*, 2002;23:759–69
- 45 Finkelstein R, Rabino G, Kassis I, Mahamid I. Device associated, device-day infection rates in an Israeli adult general intensive care unit. *J Hosp Infect*, 2000;44:200–5.
- 46 Rosser CJ, Bare RL, Meredith JW. Urinary tract infections in the critically ill patient with a urinary catheter. *Am J Surg*, 1999;177:287–

- 47 Jean YF, Jean C. Nosocomial Pneumonia. In Mitchell PF, Edward A, Vincent JL, Patrick MK, (eds.) Text Book of Critical Care. 5th Ed. Elsevier 2005;p.663–77.
- 48 American Thoracic Society and Infectious Diseases Society of America. Guidelines for the management of adults with hospital acquired, ventilator-associated and healthcare associated pneumonia. Am J Respir Crit Care Med, 2005;171:388–416.
- 49 Rello J, Ollendorf DA, Oster G, Vera LM, Bellm L, Redman R, *et al.* Epidemiology and outcomes of ventilator-associated pneumonia in a large US database. Chest, 2002;122:2115–21.
- 50 Marra AR, Wey SB, Castelo A, Gales AC, Cal RG, Filho JR, *et al.* Nosocomial bloodstream infections caused by *Klebsiella pneumoniae*: impact of extended spectrum β -lactamase (ESBL) production on clinical outcome in a hospital with high ESBL prevalence. BMC Infect Dis, 2006;14(6):24.
- 51 Pfaller MA, Jones RN, Doern GV, Kugler K. Bacterial pathogens isolated from patients with bloodstream infection: frequencies of occurrence and antimicrobial susceptibility patterns from the

SENTRY antimicrobial surveillance program (United States and Canada, 1997). *Antimicrobial Agents Chemotherapy*, 1998;42:1762–70.

52. Manual of critical care by Gabrielli, Layon, Yu.

53. Harrison's Principles of Internal Medicine, 18th edition.

54. Davidson's Principles and Practice of Medicine.

55. API Text Book Of Medicine-9th edition

RS

ABDOMEN-

CNS-

Provisional Diagnosis:

INVESTIGATIONS:

CBC-

Blood Urea-

Serum Creatinine-

Serum Electrolytes-

Urine sugar-

urine albumin-

urine deposits-

CXR

BLOOD CULTURE:

URINE CULTURE:

SPUTUM CULTURE:

STOOL CULTURE:

THERAPEUTIC INTERVENTION IN IMCU:

Urinary Catheterisation:

Dialysis:

Endotracheal Intubation:

Tracheostomy:

Nasogastric tube:

Blood Transfusion:

DRUGS GIVEN:

Immuno Suppressants:

Steroids

MASTER CHART

NAME	AGE	SEX	IP Number	URINE	SPUTUM	BLOOD	HT	DM	F
RAJIAH		65 M	66008				HT	Y	
RAMEESHA		47 F	65436						
GURUVAMMAL		60 F	66019	E.COLI			HT		
MARISELVI		39 F	65314						
THAPPAN SINGH		24 M	65936						
SHANMUGATHAI		65 F	66377					Y	
KARPAGAVALLI		21 F	64034						
MUTHU KUTTY		16 M	64411						
VAIKUNDAN		70 M	65708				HT	Y	
MADASAMY		28 M	66052						
LAKSHMI		37 F	66357						
BALAJI		15 M	60786						
SUNDARAM		65 M	46463					Y	
MOOKIAH		52 M	39255		Klebs		HT	Y	
VELU		58 M	66728						
YESURAJ		25 M	66745						
APPADURAI		40 M	66711						
PANDARAM		65 M	66760					Y	
ESAKKIAMMAL		70 F	66867					Y	
AHAMED		17 M	66883						
MARIAPPAN		35 M	66876						
RAJALAKSHMI		58 F	49614	Klebs					
ULAGAMMAL		75 F	67072					Y	
KALLIAMMAL		43 F	67158						

NAME	AGE	SEX	IP NO	URINE	SPUTUM	BLOOD	HT
MUTHULAKSHMI		17 F	67258				
UTCHIMAHALI		42 F	67247				
MOOKAIYA		70 M	66626				
VALLI		50 F	67207				
CHANDRAN		55 M	67318				HT
GNAMMAL		55 F	67324				
MURUGAN		15 M	66070				
ANNALAKSHMI		46 F	68086				HT
ARUMUGAM		55 M	67750				
DHADI VEERAN		34 M	65091				
THIRUMALAIKUMAR		38 M	68101				
SOWBEEDEN		24 M	68106				
GANESAN		52 M	68085				
ESAKIPANDIAN		79 M	68313				HT
MANISHA		13 F	68658				
CHITIRAIKANI		28 F	68598				
YOGALAKSHMI		13 F	68675				
SUNDARAM		60 M	68724				
PAPPATHI		60 F	68448				
VELDAS		60 M	68439		Klebs		
SRINIVASAN		67 M	68562				
CHITRA		54 F	68748				HT
VENKATESH		47 M	68863				
BALAJI		59 M	69032				
KAVIYA		33 F	69135				

NAME	AGE	SEX	IP NO	URINE	SPUTUM	BLOOD	HT
RADHA		26 F	69237				
GAYATHRI		43 F	69383				
TAMIL		55 M	69447				
ALAGAMMAL		63 F	69542				
ULAGAMMAL		67 F	69648	Klebs			
NAGARANI		47 F	69664				
MANIKANDAN		42 M	69793				
UNNAMALAI		37 F	69854				
RAJESH		57 M	69877				
MANNAN		48 M	69894				
VEERANARAYANAN		51 M	69921				
DAMODHARAN		46 M	69937				HT
TIRUPATHI		57 M	69948				
KAMATCHI		54 M	69978				
SARASWATHI		47 F	63189				
KARTHIGA		24 F	64156				
PATTANI		55 M	62780				
KARUNAKARAPANDIAN		60 M	67276	Klebsiella			
SUBBULAKSHMI		15 F	64523				
LAKSHMANAN		61 M	66465				HT
BACKIYALAKSHMI		63 F	67134	Klebsiella			
SYED FATHIMA		47 F	66958				
SUBRAMANIYAN		55 M	66861				
MOOKAIYA DEVAR		70 M	66626				HT
KASTHURI		60 F	66724				

NAME	AGE	SEX	IP Number	URINE	SPUTUM	BLOOD	HT
ALAGAMMAL		35 F	62696				
BASKAR		40 M	67322				
MARIA AROKIAM		64 F	68790				HT
RAJENDRAN		29 M	68677				
CHELLADURAI		69 M	68336				HT
MYDEEN		60 M	68164				
MUTHU		18 M	68599				
MUTHUKRISHNAN		28 M	68794				
KANNAN		35 M	69375				
SUDALAIMANI		25 M	69366				
AYAPPAN		30 M	60507				
NELLAI APPAN		36 M	69527				
KARTHIKEYAN		26 M	69505				
NITHYAKALYANI		22 F	64775				
NANGAMUTHU		30 M	69525				
SUBRAMANIAN		53 M	69478	E.COLI			HT
SHANTHI		25 F	69354				
SUMITHA		54 F	67952				
NAGAPUTHIRAN		68 M	69912				
MUTHULAKSHMI		53 F	69184				HT
SURESH		35 M	69992				
GANESAN		49 M	71338				
UCHIMAHALI		39 F	70435				
ARUNACHALAM		65 F	66589		Pseudo		

NAME	AGE	SEX	IP NO	URINE	SPUTUM	BLOOD	HT	DM
MUTHURANI		14 F	68586					
SHANTHA		56 F	68498				HT	
ESAKIAMMAL		17 F	68703					
KANAGARAJ		78 F	67854					
PATAMUTHU		60 M	68396				HT	Y
INDIRA		62 F	68684					
SELVI		35 F	68765					
MUTHIAH DEVAR		72 M	68786				HT	
SANKARALINGAM		27 M	68864					
CHARU		56 M	47564					
RAMIAH		52 M	47654					
SHEEBA		28 F	48652					
IYAPPAN		47 M	49567					
GURUNATHAN		63 M	49638					
DEVAKI		33 F	50364					
THENMOZHI		38 F	50377					Y
MUTHURAMALINGA		50 M	50572	Klebsiella			HT	
SELVI		55 F	50648					Y
AMARAVATHI		46 F	50776					
RAMASAMY		64 M	51764				HT	
MARIAPPAN		45 M	51879					Y
NACHIMUTHU		57 M	52372					
NARAYANAN		49 M	52548					
MURUGAN		62 M	54663			KLEBS	HT	Y
AYISHA		43 F	54706					Y

NAME	AGE	SEX	IP NO	URINE	SPUTUM	BLOOD	HT	DM
SYED BEEVI		70 F	39899					Y
RAJA		48 M	39963					
KANDASAMY		50 M	39972					
GANESAN		32 M	40015					
JAKIRA BANU		29 F	40035					
RAJIAH		44 M	40091					Y
IYAMPERUMAL		40 M	40097					
MOOKAMMAL		75 F	40207					Y
MAHESWARI		21 F	40205					
DEVADAS55		55 M	40282					
SUDHA		13 F	40908					
CHELLADURAI		73 M	43792					
SUDALAIMUTHU		75 M	43656					Y
FATHIMA		65 F	49751					Y
MARIAMMAL		45 F	49763					
SUNDARAM		65 M	46463	E.COLI				
MALAYANDI		54 M	46743					
BALARAMAN		46 M	45436					
RAJA		52 M	46589					
MURUGESAN		39 M	46765					
RAJALAKSHMI		44 F	46572					
KARUTHAMMAL		53 F	46689					
VELAICHAMY		42 M	46754					
PANDI		32 M	47462					
PARVATHY		48 F	47489					

NAME	AGE	SEX	IP NO	URINE	SPUTUM	BLOOD	HT
MARIAPPAN		40 M	27008				
THIRUNAVUKARASU		65 M	27033	Klebsiella			
SARASWATHY		20 F	27135				
MOHAMED PUROSHITH		21 M	27147				
MANICKAM		20 M	27166				
KALIRAJ		36 M	27363				
RAJAM		49 F	27371				HT
MARIAPPAN		35 M	27414				
UCHIMAHALI		21 F	27428				
MUTHULAKSHMI		40 F	27441				
SUDHA		13 F	27481				
SUBBIAH		70 M	27466				
THANGARAJ		60 M	27510				
GUNASINGH		52 M	27535				HT
DURAISAMY		25 M	27564				
VEERACHIAMMAL		21 F	27586				
SYEDALI BADSHA		14 M	27605				
AMANNULAH		48 M	39448				
PETCHIAMMAL		52 M	39486				
MUTHULAKSHMI		40 F	39592				
ISAKKIAMMAL		19 F	39611				
THIVYANADAR		62 M	39654				
KANDASAMY		60 M	39693				
GANESAN		47 M	39717				
SELVASUNDARI		52 F	39776				

NAME	AGE	SEX	IP Numb	URINE	SPUTUM	BLOOD	HT
ANTHONY		54 M	25631				HT
CHELLAMAL		60 F	25620				
SANKARAN		41 M	25656				
SANKARAN		70 M	25686				
JEJAJOTHI		56 M	25238				
GANAPATHI		61 M	25898				HT
PITCHANDI		41 M	25930		Klebsiella		
MARISELVI		22 F	25946				
CHELLAMAL		55 F	26003				HT
RANI		25 F	26162				
PARVATHIAMMAL		80 F	26237	E.COLI			
UYKATTAN		70 M	25908				HT
NAGARAJ		45 M	26932				
SUSEELA		50 F	26304				HT
PETCHIAMMAL		74 F	26281				
SUDALI		46 F	26440				
JOTHI		68 F	26402	Pseudo			
MOHAMED		26 M	26472				
NAGARAJAN		45 M	26332				
MANIMEKALAI		28 F	24515				
INDU		19 F	26624				
SELVARAJ		18 M	26719				
SIVAPERUMAL		55 M	26746				
SAKUNTHALA		62 F	26154				
KADARKARAI		75 M	26804				HT

ABBREVIATIONS/KEY TO MASTER CHART

CKD-Chronic kidney disease

HT-Hypertension

DM-Diabetes Mellitus

S.Aureus-Staphylococcus aureus

E.Coli-Escherichia Coli

Spp-Species

KLEBS-Klebsiella

Pseudo-Pseudomonas aeruginosa

E.Coli-Escherichia coli

IP NO-Inpatient Number

L+ :Leucocytosis

L- :Leucopenia

CONS:Consolidation