"CLINICAL AND MICROBIOLOGICAL PROFILE OF HEALTH CARE ASSOCIATED INFECTIONS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL WARDS AND ICU".

By:

DR JITHENDRA CHAITANYA GUBBALA MBBS

Dissertation submitted to the

Sri Devaraj Urs Academy of Higher Education and Research,

Tamaka, Kolar, Karnataka,

IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE DEGREE OF

DOCTOR OF MEDICINE (M.D.)

IN

GENRAL MEDICINE

Under The Guidance Of

GUIDE:

DR. RAVEESHA. A

M.B.B.S,MD(MEDICINE)

HOD &PROFESSOR

DEPARTMENT OF GENERAL MEDICINE

SDUMC, KOLAR

CO-GUIDE:

DR.BEENA P.M

M.B.B.S, MD(MICROBIOLOGY)

HOD &PROFESSOR

DEPT OF MICROBIOLOGY

SDUMC,KOLAR

DEPARTMENT OF GENERAL MEDICINE

SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR, KARNATAKA.

April/May- 2020

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION

&RESEARCH TAMAKA, KOLAR, KARNATAKA.

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation / thesis entitled **"CLINICAL AND MICROBIOLOGICAL PROFILE OF HEALTH CARE ASSOCIATED INFECTIONS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL WARDS AND ICU"** is a bonafide and genuine research work carried out by me under the guidance of **DR. RAVEESHA A** HOD& Professor, Department Of **General Medicine** and **DR. BEENA P.M** HOD& Professor, Department of Microbiology, Sri Devaraj Urs Medical College, Kolar, Karnataka.

Date:

Place : Kolar

Dr. Jithendra Chaitanya G

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION &RESEARCH TAMAKA, KOLAR, KARNATAKA.

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"CLINICAL AND MICROBIOLOGICAL PROFILE OF HEALTH CARE ASSOCIATED INFECTIONS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL WARDS AND ICU"** is a bonafide and genuine research work carried out by **Dr. JITHENDRA CHAITANYA G** in partial fulfilment of the requirement for the degree of **DOCTOR OF MEDICINE (M.D.)**in General Medicine

Date

Place

Dr. RAVEESHA

HOD& PROFESSOR

Department of General Medicine,

Sri Devaraj Urs Medical College, Tamaka, Kolar.

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION &RESEARCH TAMAKA, KOLAR, KARNATAKA.

CERTIFICATE BY THE CO-GUIDE

This is to certify that the dissertation entitled **"CLINICAL AND MICROBIOLOGICAL PROFILE OF HEALTH CARE ASSOCIATED INFECTIONS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL WARDS AND ICU"** is a bonafide and genuine research work carried out by **Dr. JITHENDRA CHAITANYA G** in partial fulfilment of the requirement for the degree of **DOCTOR OF MEDICINE (M.D.)**in General Medicine

Date

Place

Dr. BEENA PM

HOD& PROFESSOR

Department of Microbiology,

Sri Devaraj Urs Medical

College,Tamaka, Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION &RESEARCH TAMAKA, KOLAR, KARNATAKA.

ENDORSEMENT

This is to certify that the dissertation entitled **"CLINICAL AND MICROBIOLOGICAL PROFILE OF HEALTH CARE ASSOCIATED INFECTIONS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL WARDS AND ICU"** is a bonafide research work done by **Dr. JITHENDRA CHAITANYA G** under the guidance of **Dr RAVEESHA HOD & PROFESSOR**, Department of General Medicine Sri Devaraj Urs Medical College, Kolar, in partial fulfillment of the requirement for the degree of M.D. in General Medicine

DR. RAVEESHA.A
Professor & HOD
Department of General Medicine
Sri Devaraj Urs Medical College
Tamaka, Kolar
Date :
Place : Kolar

Dr. SREERAMULU P.N
Principal
Sri Devaraj Urs Medical College
Tamaka, Kolar
Date :
Place : Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION &RESEARCH TAMAKA, KOLAR, KARNATAKA.

ETHICAL COMMITTEE CERTIFICATE

This is to certify that the Ethical committee of Sri Devaraj Urs Medical College, Tamaka, Kolar, has unanimously approved **Dr JITHENDRA CHAITANYA G** Post graduate student, in the department of **General Medicine** at Sri Devaraj Urs Medical College, Tamaka, Kolar, to take up the dissertation work titled **"CLINICAL AND MICROBIOLOGICAL PROFILE OF HEALTH CARE ASSOCIATED INFECTIONS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL WARDS AND ICU"** to be submitted to the Sri Devaraj Urs Academy Of Higher Education and Research, Kolar.

Date: Signature of Member Secretary

Place: Ethical Committee

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION &RESEARCH TAMAKA, KOLAR, KARNATAKA.

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar, Karnataka shall have the rights to preserve, use and disseminate this dissertation / thesis in print or electronic format for academic / research purpose.

Date :

Place : Kolar **Dr JITHENDRA CHAITANYA G**

SDUAHER

Sri Devaraj Urs Academy of Higher Education and Research

Certificate of Plagiarism Check for Thesis/Dissertation

Author Name	Dr. JITHENDRA CHAITANYA G
Course of Study	MD General Medicine
Name of Supervisor	Dr. Raveesha
Department	General medicine
Acceptable Maximum Limit	
Submitted By	librarian@sduu.ac.in
Paper Title	CLINICAL AND MICROBIOLOGICAL PROFILE OF HEALTH CARE ASSOCIATED INFECTIONS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL WARDS AND ICU
Similarity	10 %
Paper ID	191201095132
Submission Date	2019-12-01 09:51:32

* This report has been generated by DrillBit Anti-Plagiarism Software

Signature of Student	Signature of Supervisor, 2/12/19
	Prof. & HOD I Unit, Dept. of Medicine, R. L.J. Hospital & Research Centre, Tamaka, Kolar.
Head of the Department, Prof. & HOD I 2/12/19, Dept. of Medicine, R.L.J. Hospital & Research Centre, Tamaka, Kolar.	
University Librarian, Senior Librarian, Library and Information Centre, Sri Devaraj Urs Medical College, Tamaka, KOLAR-563 101.	Director, Of Post Graduate Studies, Director, P.G. STUDIES, Sri Devaraj Urs Medical College, Tamaka, KOLAR-563 101

ACKNOWLEDGEMENT

No academic work is single handedly accomplished. This work is no exception. Words fail me in expressing my heartfelt and humble gratitude to my guide ***Dr. RAVEESHA A,*** *M.D., HOD & Professor, Department of General Medicine, and my co-guide* ***Dr.BEENA P.M****, MD, HOD & Professor, Department of Microbiology for the guidance and encouragement all along in completing my study. His encouragement, sense of punctuality, research oriented approach, the painstaking effort to weed out errors and his affection during the entire course of study leaves me permanently indebted to him.*

I express my deep sense of gratitude and humble thanks to ***Dr. Lakshmaiah.V, Dr. B.N.Raghavendra Prasad, Dr. Prabhakar.K, Dr.Vidyasagar****,* ***Dr.Srinivas SV*** *Professors for their advice, inspiration and constant encouragement throughout the present study.*

I would like to thank all my teachers ***Dr. Thanuj Reddy, Dr.Vishwanath Reddy, Dr.Niveditha, Dr.Prasanna, Dr.Anitha, Dr.Mahesh, Dr. Phaneesh, Dr.JayaPrasad & Dr.Jalaja*** *from the Department of General Medicine for their heartfelt support at all times.*

I would like to acknowledge the opportune help and permission rendered by the Medical Director, Principal and Medical superintendent, SDUMC/RLJH, in conducting this study.

I humbly thank ***Dr. Thanuj Reddy, Dr. Rakesh Reddy, Dr.Vennela Reddy, Dr.Raghavender Reddy, Dr.Maharaj*** *my senior postgraduates and friends for their help and guidance to complete my dissertation.*

I thank all my colleagues ***Dr. Samba, Dr. Rumaisa, Dr. Minni, Dr. Deepa, Dr. Pujitha, Dr. Manoj, Dr Hamsa,*** *for their timely suggestion throughout the preparation of this manuscript.*

I thank ***Dr.Chethan,*** *Bio Statistician for his aid in Data interpretation & Statistical analysis of my Study which led to its successful completion.*

I am also thankful to all the Nursing Staff, Technical Staff and non-teaching staff for their invaluable help without which this study would not have been possible.

I thank my father ***Mr. Narayana Murthy G*** *my mother* ***Mrs. Bhavani G,*** *my sister* ***Viznani*** *and my family members for their constant source of encouragement, and support throughout the entire career.*

Last but not the least I am grateful to all those patients who were the subjects for the study, without whose cooperation this work would not have been possible.

Dr. JITHENDRA CHAITANYA G

ABSTRACT

BACKGROUND:

Infections acquired by patients while receiving treatment are called as Health Care-associated Infections (HCAI). Globally, HCAIs are the frequent adverse event in health care delivery. Each year, numerous patients are affected by infections, causing significant mortality and financial losses for health systems. At any time, of the hundred patients that are hospitalized, about seven patients in developed countries and ten patients in developing countries will contact at least one HCAI. Also, the endemic load of HCAI is higher in developing countries as compared to high-income developed countries, especially in patients admitted to Intensive Care Units (ICUs) and in neonates. The review of the literature of various studies revealed a fragmented picture of endemic burden of Hospital acquired infection in the developing countries. For most countries (66%), no data is available and for some regions only very meagre information was available. In ICUs, HCAI acquiring risk is very much high with approximately 30 out 100 patients being affected by at least one episode of HCAI with significantly associated morbidity leading to high DALY and mortality. A report on Device-associated Infections (DAIs) from 25 countries across globe in 173 ICU's states , crude excess mortality in adult patients was 18.5%, for Catheter-related Urinary Tract Infection (CRUTI), 23.6%, for Catheter-related Bloodstream Infection (CR-BSI), and 29.3% for Ventilator-associated Pneumonia (VAP). There are only few studies focusing on profiles of HCAI in medical wards in developing countries like India. Our study proposes to be a descriptive study of clinical and microbiological profiles of HCAIs in patients admitted in medical ward and ICU.

OBJECTIVES:

1. To record and isolate the pathogen responsible for hospital acquired infections.

2. To determine the Clinical profile of Hospital care acquired infections in medical wards and ICU.
3. To determine the antibiotic sensitivity of isolated organism.

MATERIALS AND METHODS:

The study included 69 patients admitted in the medical wards and ICU under the Department of Medicine at RL JALAPPA HOSPITAL, TAMAKA, KOLAR who met the inclusion criteria and who

provided informed written consent for being part of our study. It was a prospective observational study

carried out between AUGUST 2018 and JULY 2019. The study protocol was approved by the

institutional ethical committee.

Detailed history was taken and clinical examination was done. Case records were reviewed for the initial investigations, diagnosis and treatment. The invasive devices like Foleys catheter, ET tube, IV cannula and central vein catheter used and procedures done were recorded along with the duration of each device.

RESULTS

Our study included 69 patients admitted in the medical wards and ICU under the Department

of Medicine at RL JALAPPA Hospital, Tamaka, Kolar who met the inclusion criteria and who provided informed written consent for being part of our study. Our study included 42 patients (60.9%) from ICU and 27 (39.1%) of the subjects were ward patients. 42(60.9%) of the patients were male and 27(39.1%) of the patients were female.

Among ICU patients most common HAI was VAP, it was present in 90.5% of ICU patients followed by 14.3% of ICU patients had UTI, 14.3% of ICU patients had Blood stream infection, 11.9% of ICU patients had Phlebitis.

Among ward patients most common HAI was Phlebitis, it was present in 81.5% of ward patients followed by Venous infection was present in 7.4% of ward patients, 7.4% of ward patients had Blood stream infection and 3.7% of ward patients had UTI.

In patients of medical ward who developed HAI, device days >4 days ,was the most common predisposing factor followed by prior antibiotic use, prolonged hospital stay(>5 days) , immunosuppressed states. Immunosuppressed states included patients with chronic liver disease , chronic kidney disease, connective tissue disorders and Tuberculosis. In ICU patients who had HAI, most common predisposing factors were device days >4 days , followed by respiratory disorders, prolonged hospital stay, prior antibiotic use. Patients with poor GCS (<9) accounted for 33.33%. Four patients who have developed VAP with Acinetobacter isolated in 3 patients and Klebsiella isolated in 1 patient showed pan resistant to all antibiotics. These patients were started on Colistin. Outcome of these patients was that all patients succumbed due to complications of underlying diseases.

CONCLUSION:

From this study, we concluded that patients admitted to medical ward and ICU are at a risk of acquiring healthcare associated infection. In medical ward, most common HAI was PHLEBITIS followed by BSI, CA-UTI. Most common HCAIs in ICU patients were VAP followed by BSI and UTI. Acinetobacter was the most common organism followed by Klebsiella. In fungi, Candida was the most common organism. More stringent aseptic measures should be taken in medical wards and ICU to reduce

incidence of HAIs. Guidelines for empirical antibiotics should be formed in hospital according to the sensitivity and resistance patterns. Indiscriminate use of antibiotics should be avoided.

KEY WORDS:

HCAI, VAP, OHLEBITIS

ABBREVIATIONS

BSI-	BLOODSTREAM INFECTION
CA-UTI-	CATHETER ASSOCIATED INFECTION
CDC-	CENTERS FOR DISEASE CONTROL AND PREVENTION
CKD-	CHRONIC KIDNEY DISEASE
CLD-	CHRONIC LIVER DISEASE
CRBSI-	CATHETER RELATED BLOODSTREAM INFECTION
CR-UTI-	CATHTER RELATED ASSOCIATED INFECTION
CVA-	CEREBROVASCULAR ACCIDENT
DM-	DIABETES MELLITUS
E.COLI-	ESCHERICHIA COLI
ESBL-	EXTENDED SPECTRUM - LACTAMASE
ET TUBE-	ENDOTRACHEAL TUBE
EVD-	EXTERNAL VENTRICULAR DRAIN
GCS-	GLASGOW COMA SCALE
HC AI-	HEALTHCARE ASSOCIATED INFECTION
MDR-	MULTIDRUG RESISTANT
MRCONS-	METHICILLIN RESISTANT COAGULASE NEGATIVE STAPHYLOCOCCUSAUREUS
MRSA-	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS
MV-	MECHANICAL VENTILATION
NHSN-	NATIONAL HEALTHCARE SAFETY NETWORK
NNIS-	NATIONAL NOSOCOMIAL INFECTIONS SURVEILLANCE
NP-	NOSOCOMIAL PNEUMONIA
ORSA-	OXACILLIN RESISTANT STAPHYLOCOCCUS AUREUS
PCNL-	PERCUTANEOUS NEPHROLITHOTOMY
PNU1-	CLINICALLY DEFINED PNEUMONIA
PUO-	PYREXIA OF UNKNOWN ORIGIN

R-	RESISTANT
S-	SENSITIVE
SSI-	SURGICAL SIT E INFECTION
TPN-	TOTAL PARENTERAL NUTRITION
VRE-	VANCOMYCIN RESISTANT ENTEROCOCCUS
WHO-	WORLD HEALTH ORGANISATION

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

INTRODUCTION

Infections acquired by patients while receiving treatment are called as Health Care-associated Infections (HCAI).[1] Initially, HCAI was referred to those infections connected with admission to an acute-care hospital which earlier was called as a nosocomial infection, but now the term includes infections developed in various settings where patients obtain health care that includes long-term care, family medicine clinics, ambulatory care and home care. HCAIs are infections that first appear 48 hours or more after hospitalization or within thirty days post receiving health care.[2]

Globally, HCAIs are the frequent adverse event in health care delivery. Each year, numerous patients are affected by infections, causing significant mortality and financial losses for health systems. At any time, of the hundred patients that are hospitalized, about seven patients in developed countries and ten patients in developing countries will contact at least one HCAI. Also, the endemic load of HCAI is higher in developing countries as compared to high-income developed countries, especially in patients admitted to Intensive Care Units (ICUs) and in neonates.[3]

WHO report (results of systematic reviews of the literature on endemic HCAI from 1995 to 2010) of published studies, states pooled HCAI prevalence in mixed patient populations was 7.6% in higher income countries. European Centre for Disease Prevention and Control (ECDC) estimates, 4,131,000 patients are affected by ~4,544,100 episodes of HCAI every year in Europe. In 2012, the estimated incidence rate of HCAI in the United States of America was 4.5%, equalling to 9.5 patients affected with infections per thousand patient-days and with overall 1.7 million patients affected.[4]

The review of the literature of various studies revealed a fragmented picture of endemic burden of Hospital acquired infection in the developing countries. For most countries (66%), no data is available and for some regions only very meagre information was available. Multiple studies that were conducted in resource limited health care setting reported HCAI

rates are raised as compared with those of developed countries. Rate of prevalence of HCAI has varied from 5.7- 19.1% with a average rate of prevalence of 10.1%. The pooled HCAI prevalence rate was fundamentally higher in high-quality studies as compared to low-quality studies (15.5% *vs*. 8.5%, respectively). Surgical Site Infection (SSI) is the most common type of hospital acquired infection in developing countries with rates of overall incidence varying from 1.2-23.6 per hundred surgical procedures and a pooled incidence rate of 11.8%. In contrast, SSI rates vary between 1.2% and 5.2% in developed countries.[4]

In ICUs, HCAI acquiring risk is very much high with approximately 30 out 100 patients being affected by at least one episode of HCAI with significantly associated morbidity leading to high DALY and mortality. In patients with high risk, pooled cumulative incidence density was seventeen episodes per thousand patient-days in industrialized world as compared to the incidence rates of ICU-acquired infection among adult patients in developing countries which ranged from 4.4-88.9% and pooled cumulative incidence density was 42.7 episodes per 1000 patient-days.[4]

The adverse effects of HCAI include prolonged continuous stay in hospital, high disability, rise of resistance to antimicrobials, a heavy financial loss for health systems, high cost of expenditure for patients and patient families, overall increased deaths. In Europe, HCAIs cause sixteen million extra-days of hospital stay, thirty seven thousand attributable deaths, and contribute to an additional one million deaths annually. In the United states, ~99,000 deaths were attributed to HCAI in 2012. Information is again very scarce and minimal from developing world and no data are available at national or local levels. A report on Device-associated Infections (DAIs) from 25 countries across globe in 173 ICU's states , crude excess mortality in adult patients was 18.5%, for Catheter-related Urinary Tract Infection (CRUTI), 23.6%, for Catheter-related Bloodstream Infection (CR-BSI), and 29.3% for Ventilator-associated Pneumonia (VAP). A review of many studies demonstrated that increased time of hospital stay associated with HCAI varied between 5 and 29.5 days. Although worldwide estimates of HCAI are not yet readily available, by integrating data

different published studies, there is clear cut evidence that numerous patients are affected every year globally, with very high percent of HCAI seen in low-income and middle-income countries.[4] There are only few studies focusing on profiles of HCAI in medical wards in developing countries like India. Our study proposes to be a descriptive study of clinical and microbiological profiles of HCAIs in patients admitted in medical ward and ICU.

OBJECTIVES

AIMS AND OBJECTIVES

1. To record and isolate the pathogen responsible for hospital acquired infections.
2. To determine the Clinical profile of Hospital care acquired infections in medical wards and ICU.
3. To determine the antibiotic sensitivity of isolated organism.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

REVIEW OF LITERATURE

For National Healthcare Safety Network [NHSN] surveillance purpose in the acute health care settings, the Centers for Disease Control and Prevention (CDC) defines an HCAI as a localized or systemic condition resulting from an adverse effect and reaction to the presence of an infectious agent(s) or its toxin(s). At the time of admission, there should be no evidence that the infection was incubating or present in the acute care setting. HCAIs can be developed due to infectious organisms from endogenous or exogenous sources. Endogenous sources are body sites, like the skin, mouth, nose, GI tract, or vagina that are normally inhabited by micro-organisms. Exogenous sources are those external to the patient, such as patient care personnel, patient care equipment, visitors, surgical and medical equipment, or from the health care environment.[5]

The US CDC identifies that ~1.7 million hospitalized patients annually acquire HCAIs while being treated for other health problems and more than 98,000 of these patients (one in 17) die due to HCAIs. Among hundred patients that are hospitalized, seven patients in advanced countries and ten patients in emerging countries acquire an HCAI. Many studies that are conducted in high-income countries found that 5% to 15% of the patients acquire HCAIs, which can affect from 9% to 37% of those patients that are admitted to the ICUs.[6]

Around 12–17 microorganisms cause 80–87% of HCAIs that include *Staphylococcus aureus*, *Enterococcus* species (e.g., *faecalis, faecium*), *Escherichia coli*, coagulase-negative *staphylococci*, *Candida* species (e.g., *albicans, glabrata*), *Klebsiella pneumoniae*, *K. oxytoca*, *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Enterobacter* species, *Proteus* species, Yeast NOS, *Bacteroides* species, and other pathogens.[6]

A Singapore study reported 11.9% (646) patients with HCAIs, primarily undetermined clinical sepsis, and pneumonia caused mainly by *S. aureus* and *P. aeruginosa*. This Study also

reports that the *Acinetobacter* species and *P. aeruginosa* were extremely resistant to carbapenem.[6]

An European study found that 2,609,911 new patients were diagnosed with HCAIs yearly in the European Union and European Economic Area. Study demonstrated that for every twenty patients that are hospitalized , minimum one patient acquired an HCAI that was preventable. *K. pneumoniae* and *Acinetobacter* species were very much resistant to multiple antimicrobials, and the lack of new antimicrobials increases the burden in this settings.[6]

Systematic review with meta-analysis of several studies for HCAIs in Asian countries found an average prevalence rate of 9.1 per 100 patients with the commonest infectious microorganisms being *Pseudomonas aeruginosa*, followed by *Klebsiella* species, and *A. baumannii*.[6]

Devrajani BR, et al., conducted a hospital-based descriptive study of 4 months including 50 patients with nosocomial infection. Majority of the patients, 38 (76%), developed febrile illness during the admission period/hospital stay in ward. The reasons of hospital acquired infection were UTI in 34 (68%) patients, followed by blood stream infection (BSI) in 19 (38%), pneumonia in 9 (18%) patients, sinusitis in 4 (8%) patients, otitis in 3 (6%) patients, bronchitis in 7 (14%) patients and tracheitis in 3 (6%) patients. Skin infection and soft tissue infection was identified in 8 (16%) patients. Gastrointestinal infection was noted in 12 (36%) patients, and 6 (12%) patients developed fever post medical-specific procedures (peritoneal paracentesis, bone marrow and liver biopsy).[7]

The blood culture was taken from two different sites which showed positive result in 19 (38%) patients and this is due to BSI. The chest radiograph showed the pneumonic patch (consolidation) in 16 (32%) patients, in which 7 (14%) were unconscious, 2 (4%) were semiconscious and 10 (20%) were conscious. On the results of pleural fluid examination of the study, the reason for hospital-acquired pneumonia in such patients was infection with gram-negative bacilli *E. coli* in 6 (12%) patients, *Pseudomonas* in 3 (6%) patients,

Enterobacter in 1 (2%) patient, *Klebsiella* in 2 (4%) patients, *S. aureus* in 3 (6%) patients and anaerobes in 4 (8%) patients. Out of 12 (36%) patients of hospital-acquired GI infection, 8 (16%) were infected with *Clostridium difficile*, 3 (6%) had stool culture positive with gram-negative organisms, 1 (2%) patient with acquired hepatitis A and 1 (2%) patient with acquired hepatitis E infection during admission period, and 4 (8%) patients developed malaria during hospitalization.[7]

Donowitz LG, et al., conducted a study on HCAI's in ICU patients. This study states that, patients admitted to the ICU have a high risk of HCAI as compared to other patients in wards. The general medical/surgical ward patients have an overall risk of 6% of acquiring an HCAI, whereas severely ill patients admitted in ICU have an 18% risk ($p<0.001$). During this 2-year study, 440 of 2441 patients admitted to the ICU developed hospital acquired infections. Patients with prolonged ICU stays and patients in the obstetrics and gynecology, orthopedics, and general surgery departments were more likely to become infected. The common bloodstream pathogens were *S. epidermidis*, *S. aureus*, and *Serratia* and *Pseudomonas* species.[8]

Habibi S, et al., conducted an observational propspective study which describes the rates of nosocomial infections, the sites of infection, the pathogens involved, their antibiogram and the risk factors at a tertiary care hospital in India. Ninety five episodes of HCAI's were recorded in 62 patients with an incidence rate of 28.6/1000 person days): pneumonia 77%; UTI 24% and BSI 24%. All isolates of *Acinetobacter*, *Pseudomonas* and *Klebsiella* and 83.3% of *E. coli* were resistant to the third generation cephalosporins. A prolonged duration of the time spent in ICUs and days of intervention were associated with increased rate of incidence of HCAI's.[9]

HCAI's are major problem in the ICU because of increased frequency, morbidity, and high incidence of death. The commonest ICU infections are pneumonia, BSI and UTI, most of which are device related.[10]

Catheter associated Urinary Tract Infection (CA-UTI):

Worldwide, Catheter associated-bacteriuria is the most common HCAI, accounting for up to 40% of hospital-acquired infections. In public hospitals, 15–25% of patients have a urethral catheter inserted at some time during their hospital stay, and the rate of usage catheter appears to be raising. Most patients are catheterized for 2–4 days only, but some are catheterized for longer durations during stay in hospital. The incidence rate of bacteriuria in relation to catheterization is three to eight percent per day, and the duration of catheter is the most important and critical risk factor for the development of Catheter associated-bacteriuria. Other factors related to risk of developing CA-bacteriuria include not receiving systemic antimicrobial therapy, female gender, positive urethral meatal culture results, colonization of the microbes in drainage bag, catheter insertion outside the operating room, catheter care violations, rapidly fatal underlying illness, geriatric patents, diabetes mellitus, and increased serum creatinine levels at the time of catheterization.[11]

Pneumonia:

VAP is the pneumonia that developes 48 hours or more post endotracheal intubation and Mechanical Ventilation (MV) that was not intubating at the time of admission, also including pneumonia developing post extubation. Pneumonia is the second commonest ICU-acquired infection, and 86% of nosocomial pneumonias are VAP. Around 10–20% of patients on MV for >48 hours develop VAP. Early-onset VAP, which occurs during the first 4 days of MV, is less severe usually and is associated with a better prognosis and more likely caused by antibiotic-sensitive bacteria. Late-onset VAP, which develops 5 or more days after initiation of MV, is caused by Multidrug Resistant (MDR) organisms and these are associated with high rates of mortality and morbidity. The commonest pathogens causing VAP include aerobic gram-negative rods such as *P. aeruginosa*, *Acinetobacter* species, *K. pneumoniae* and *E. coli*.[12]

Multivariate analyses have shown a significant relation between previous antibiotic exposure, MV lasting >7 days, and *P. aeruginosa* isolates. The American Thoracic Society and a further report from France described the distribution of causative organisms for VAP according to easily identifiable risk factors. These studies suggested that classifying patients according to previous duration of MV and previous exposure or no exposure to antibiotics provided a rational basis for expecting the pathogens.[13]

Rit K, et al., conducted a study called 'VAP in a tertiary care hospital in India: Incidence, etiology, risk factors, and role of multidrug resistant pathogens,' which included 140 adult patients. The incidence density rate of VAP was 21.875 per thousand ventilator days. Most of the patients had late-onset VAP (60.7%) with an average number of days for onset around 8 days. *Pseudomonas* spp. and *Acinetobacter* spp. are significantly associated with late-onset VAP.[12]

According to study conducted by Akkoyunlu Y, et al., risk factors for Nosocomial Pneumonia (NP) in ICUs of a University Hospital including 304 patients, NP developed in 78 patients (25.6%) with a density rate of 23.1 cases per 1000 patient-days. Overall, 11.5% of 304 patients developed VAP. Thereby, VAP accounts for 44.9% (n=35) of all patients with NP during the study period. VAP density rate was 28.7 cases of NP per 1000 ventilator-days. The mean time for NP occurrence was 9.1 ± 6.0 days following hospital admissions. The mean time for VAP occurrence was 5.2 ± 3.1 days after MV. Non-invasive MV (NIMV) accounted for 10.3% of all patients with NP, and 9% of control patients (p=0.75).[14]

Bloodstream Infections (BSIs):

BSIs are important casuse of morbidity and mortality in the hospitalized patients. On the basis of data from death certificates, BSI's are tenth leading cause of death in USA with the age-adjusted death rate has risen by 78% over the past 2 decades. Studies reported that BSIs incidence to be between 1% in ICU patients and thirty six percent in bone-marrow recipients. The death rate ranged from twelve percent in total hospitalized patients to eighty percent in

ICU patients. In recent studies of ICU patients, crude death rates varied from 35–53%. The rate of mortality due to blood stream infections in these patients was estimated to be 16–40%. Among ICU patients with BSI, the length of hospital stay was increased by 7–25 days and the total hospitalization time by 4.5–32 days. Inappropriate empirical antibiotics is a crucial death predictor in such patients.[15]

CR-BSI can be defined as presence of bacteraemia originating from an Intravenous (IV) catheter. It is one of the commonest, lethal, and costly complications of central catheterization and also the most common cause of nosocomial bacteraemia. Majority of CR-BSIs are associated with Central Venous Catheters (CVCs), and in many prospective studies, the relative risk for CR-BSI is up to sixty four times higher with CVCs than with peripheral venous catheters. Incidence of reported CR-BSI varies between countries and differs even from one hospital to other hospital. Meta-analysis study done at The Johns Hopkins University showed that BSIs are the third most leading cause of HCAI's leading to increased morbidity and mortality. BSI's have a high attributable mortality rate of 12–25%. The risk factors for CR-BSI include underlying disease, catheter insertion method, catheter insertion site and duration, and purpose of catheterization. The parenteral nutrition administration through intravascular catheters increases CR-BSI risk. Local risk factors like poor personal hygiene, occlusive dressing, moisture around the insertion site, *S. aureus* nasal colonization, and adjoining infections support the role of bacterial colonies formation in the pathogenesis of CR-BSI. Major risk factors for dialysis CR-BSI include dialysate contamination or equipment contamination, improper water treatment, re-using dialyzer, old age, higher intravenous iron dose, increased human erythropoietin dose, low hemoglobin level, low serum albumin level, diabetes mellitus, atherosclerosis, and recent hospitalization for surgery. CR-BSI rate varies significantly in different studies.[16]

Infection related to IV devices results in increase in hospital costs for patients, increased hospitalization duration, and high patient morbidity. In a meta-analysis of 2573 CR-BSI, the case-fatality rate was fourteen percent , and among these 19% of the deaths were related to

catheter-related infection. The death rates attributed to catheter-related *S. aureus* bacteremia (8.2%) is significantly higher than the rates for other infectious pathogens ($p<0.001$), whereas the deaths secondary to coagulase-negative staphylococcal catheter-related bacteremia (0.7%) was significantly lower compared to other pathogens.[17]

Moreno CA, et al., conducted a Columbian study for 3 years including 2172 patients hospitalized in an ICU for an aggregate duration of 14,603 days who acquired 266 Device Associated Infections (DAIs), for an overall DAI rate of 12.2%, or 18.2 DAIs per thousand patient-days. This study states that, CVC-related BSI occurred in 47.4% of all DAIs, VAP represented 32.3%, and CAUTI represented 20.3%. The crude attributable mortality was 18.5 among those with CVC-associated BSI, 16.9% among patients with VAP; and 10.5% among those with CAUTI.[18]

Aa per one Indian study by Mehta A, et al., including 10,835 patients hospitalized for 52,518 days acquired 476 HCAIs. CVC-BSI rate was 7.92 per thousand catheter-days; the VAP rate was 10.46 per thousand ventilator-days; and the CAUTI rate was 1.41 per thousand catheter-days.[19]

Since 1970, CDC's NNIS-National Nosocomial Infection Surveillance System has been collecting data about the incidence and aetiologies of hospital-acquired infections, including CVC-associated BSIs in a group of nearly 300 US hospitals. The majority of BSIs acquired in hospital are associated with the increased use of a CVC, with BSI rates being predominant among patients with CVCs than those without CVCs. Incidence rates of CVC-associated BSI vary considerably by hospital size, hospital service, and types of CVC. During 1992–2001, NNIS hospitals reported ICU rates of CVC-associated BSI ranging from 2.9 (in a cardiothoracic ICU) to 11.3 (in a neonatal nursery for infants weighing <1000 g) BSIs per 1000 CVC days.[20]

As per the study, 'The Influence of Inadequate Antimicrobial Treatment of Bloodstream Infections on Patient Outcomes in the ICU Setting' conducted by Ibrahim EH, et al., in

critically ill patients with a BSI who received inadequate antimicrobial treatment were significantly more likely to die during their hospitalization compared with similar patients with BSIs receiving adequate antimicrobial treatment. Study also identified various risk factors for the administration of inadequate antimicrobial treatment. These factors included the presence of a BSI caused by *Candida* species, prior antibiotic therapy during the same hospitalization, longer durations of central vein cannulation, and low serum albumin concentrations at the time of ICU admission. In addition, BSI caused by antibiotic-resistant pathogens (*Candida* species, vancomycin-resistant enterococci, ORSA, and coagulase-negative *staphylococci*) are due to usage of inadequate antimicrobial treatment. Study further demonstrated a significant direct association between the inadequate antimicrobial treatment for specific pathogens and their associated rates of hospital mortality. Nonetheless, a few pathogens, such as *E. coli, P. aeruginosa*, are found to associate with relatively minimal rates of inadequate antimicrobial treatment yet had observed hospital mortality rates >30%. Prior treatment during the same hospitalization with antimicrobial agents are one of the important risk factor for the later occurrence of an antibiotic-resistant infection. In addition, the overuse of specific antimicrobial agents or classes of antibiotics can predispose to higher rates of resistance to those drugs among both community-acquired pathogens and hospital-acquired pathogens. Likewise, the prolonged presence of invasive medical devices, particularly intravascular catheters and equipment was associated with the emergence of antibiotic resistance. In addition to being a marker of greater severity of illness, these devices are frequently associated with biofilm formation on their surfaces. Penetration of antibiotic into biofilms is usually diminished, allowing sequestered pathogens colonizing these devices within the biofilms to be exposed to subtherapeutic concentrations of antibiotics. Presence of such an environment favors the emergence of antibiotic-resistant micro-organisms.[21]

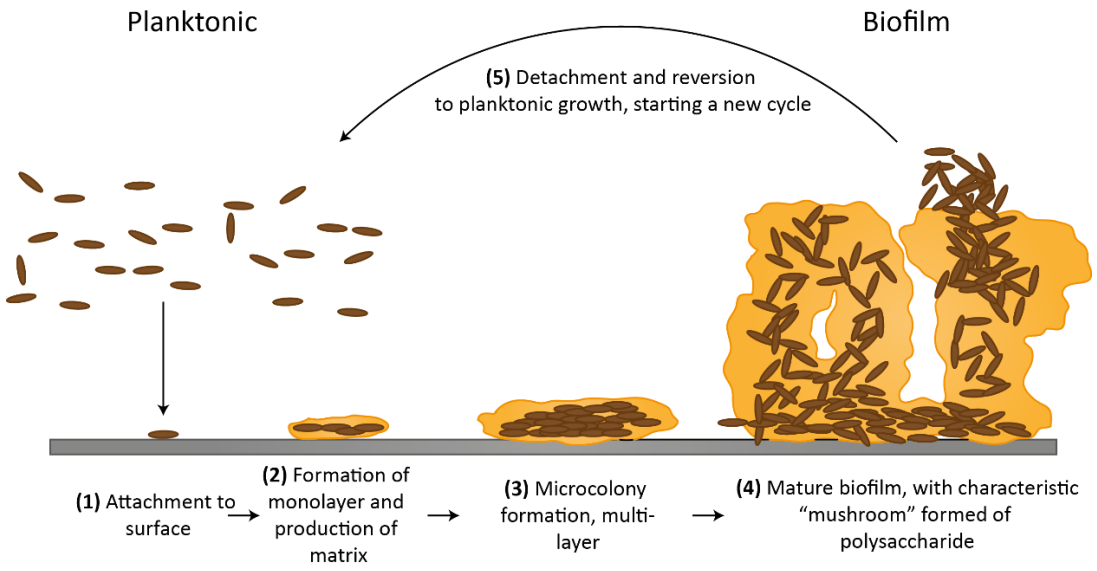


Fig 1:Representation of biofilm formation

Datta P, et al., conducted the study called 'Healthcare associated infections: Risk factors and epidemiology from ICU in Northern India,' including 679 patients. The overall infection percentage was 24.44% and infection rate was 29.1%. Central-line-associated BSI (13.50%) was the commonest HCAI followed by CA-UTI (10.75%) and Ventilator associated Pneumonia (6.15%). Among the 166 patients diagnosed with DAIs, 81 died (48.7%), whereas 162 patients out of 513 (31.5%) died among the group not having DAIs. All of the 679 patients had indwelling urinary catheter and total number of Foley's catheterization days was 8039. Number of UTI episodes are found to be seventy three (10.75%) among the ICU patients who had indwelling urinary catheter. Additionally, CAUTI was 9.08 per thousand catheter days. *K. pneumoniae* was the most commonly isolated organism from BSIs among ICU patients.[22]

Parameswaran R, et al., conducted a study named 'Intravascular catheter-related infections in an Indian tertiary care hospital.' The mean age among controls was 43.53years and that among patients with local catheter infections and CR-BSI were 42.1 and 42.84, respectively. The commonest premorbidity among the controls and patients with CR-BSI was renal failure (35.5% and 40%, respectively) while that among the patients with local catheter infections was diabetes (34.9%). Local signs of inflammation such as erythema, warmth, induration,

tenderness, and purulence at the exit site were seen among all patients with CR-BSI and among the majority of patients with local catheter infections (96.4%). The commonest indication for central venous cannulation was for IV fluids and antibiotic administration in the controls, local catheter infections and CR-BSI (57% *vs*. 77% *vs*. 64%, respectively). Hemodialysis was the indication in 44% of controls and 29% in patients with local catheter infections and 40% in CR-BSI, respectively. Chemotherapy as an indication was in 3% of central venous catheters among controls and 4% of patients with local catheter infection.[23]

Venous infections:

Phlebitis has long been recognized as a risk for infection. For adults, lower extremity insertion sites have a higher risk for infection than are upper extremity sites. Additionally, hand veins have a lower risk for phlebitis than do veins on the wrist or upper arm. The density of flora over skin at the catheter insertion site is the commonest and major risk factor for CR-BSI. Authorities recommend that CVCs be placed in a subclavian site instead of a jugular site or femoral area in order to reduce the risk for infection. No randomized study has satisfactorily compared infection rates for jugular site, subclavian sites and femoral site catheters. Internal jugular vein catheterisation was associated with higher risk for infection than subclavian or femoral vein catheterisation. Femoral catheterisation has comparatively high floral colonization rates when used in adults. Femoral catheters should be avoided because of the high risk of developing deep venous thrombosis than compared with internal jugular or subclavian catheters, and also because of a presumption that such femoral catheters are more likely to become infected. However, studies in paediatric patients have shown that femoral catheters have a low incidence of mechanical complications and might have an equivalent infection rate to that of non-femoral catheters.[20]

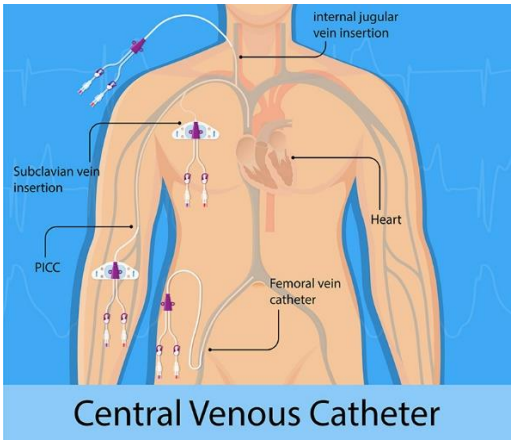


Fig 2: CVC sites

Healthcare-associated fungal infections:

Exposure to airborne fungal pathogens such as *Aspergillus* spp within the hospital environment, particularly during construction, has caused outbreaks of nosocomial aspergillosis in severely reduced immunity patients such as who underwent Hematopoietic Stem Cell Transplantation (HSCT).[24]

The primary fungal infections of concern in HSCT recipients are candidiasis and aspergillosis. Infections caused by *Candida* species fall into the distinct syndromes of acute BSI and chronic infection or hepatosplenic candidiasis. In both the cases, *Candida* species are primarily acquired from the GI tract. *Candida parapsilosis* is the exception to this rule, as this organism is acquired via the IV catheter route, probably in association with infected infusates. Aspergillosis is exogenously acquired, via aerosolization.[25]

Antifungal prophylaxis in critically ill non-immunocompromised patients could be considered for selected groups of patients in whom the incidence of candidiasis is expected to be >10%.[26]

Yang SP, et al., conducted a study for risk factor analysis of healthcare-associated fungal infections in an intensive care unit: a retrospective cohort study. Five independent risk factors were recognised for all sites of ICU fungal HCAI by the multivariable analysis, which included Total Parenteral Nutrition (TPN) use, sepsis, surgical patients, mechanical

ventilation and an indwelling urinary catheter. The mortality rate of patients with ICU fungal HCAIs was observed to be twice that of patients without ICU HCAIs. Candidemia has resulted in high mortality rates of upto 50–70%. As documented Candiduria precedes candidemia in the ICU, and candiduria itself has a higher death rate of 31.3% . The rates and species distribution of fungal HCAIs have differed geographically and institutionally, and strains of *Candida* species resistant to antifungals have been increasing.[27]

Candida species are the commonest fungal organisms causing serious HAIs, especially in ICU admitted patients. A study using the National Hospital Discharge Survey estimated that the incidence of invasive candidiasis to have increased from 23 per 100,000 US populations in 1996 to 29 per 100,000 in 2003. The corresponding incidence rate of invasive candidiasis per 10,000 hospital discharges increased from 20 in 1996 to 24 in 2003. Among invasive candidiasis, candidemia is seen to affect 2-8 infections per 10,000 hospital discharges in recent studies from the Europe and US. The exact incidence rate of health care–associated candidemia is estimated to be higher because of the relatively poor diagnostic yield (approximately 50%) of positive blood culture results in patients with disseminated candidiasis with implicit candidemia[27].

As per the study done by Lundstrom T, et al., fungal UTI's encompass a broad variety of fungi, including the endemic mycosis *Cryptococcus* species and opportunistic pathogens such as *Aspergillus* species. However, majority of fungal urinary tract infections are caused by *Candida* species, and they mainly present as complicated hospital acquired infections. Only rarely we encounter candiduria as a community-acquired infection in a structurally normal urinary tract. This study focused on hospital acquired *Candida* species UTIs or candiduria. Incidence of *Candida* species increased recently in humans due to the increased use of invasive devices, immunosuppressive therapy and higher antimicrobial agents. The recovery of *Candida* species from urine samples of patient creates a dilemma in the doctors, because the presence of *Candida* species can signal colonization, which may not require treatment;

lower tract infection; or upper UTI, including both ascending pyelonephritis and renal candidiasis, which need treatment.[28]

A study performed by Platt, et al., showed that 26.5% of all urinary infections related to catheters were caused by fungi. Rivett, et al., found that 2% of urine specimens submitted to a hospital microbiology laboratory tested positive for yeast *vs*. 11% of the urine samples obtained from patients in immunocompromised state like leukaemia, transplantation. Currently, 10–15% of nosocomial UTIs are caused by *Candida* species. Candiduria is a relatively rare finding in otherwise healthy people. Guze and Harley found funguria in only 15 of 1500 patients; >50% of these 15 patients had diabetes mellitus and were receiving antibiotics. In another study, the results of urine cultures were positive for 10 of 440 healthy adults, but these culture results turned back to negative when clean-catch techniques were used; whereas, the incidence of fungal UTIs, and specifically candiduria, has dramatically increased among hospitalized patients, particularly among those patients with indwelling drainage devices.[28]

Diabetic patients are at increased risk of developing UTIs, including UTIs caused by fungi. Diabetes may predispose patients to fungal candiduria by predisposing them to *Candida* colonization of the vulvovestibular area (in women) by enhancing urinary fungal growth in the background of glycosuria, by lowering host resistance to invasion by fungi as a consequence of impaired phagocytic activity, and by promoting stasis of urine in a neurogenic bladder. Ultimately, patients with diabetes are more likely to undergo urinary tract instrumentation and to receive antibiotics. *Candida* species colonization of the GI tract is present in ~30% of normal adults. However, among them who are receiving antibiotics, colonization rates approach 100%. As there is little evidence that systemic antibiotics directly influence *Candida* species proliferation or virulence, it is likely that antibiotics contribute to colonization by *Candida* species by suppressing endogenous bacterial flora, primarily in the gastrointestinal and genital tract and in areas adjacent to the urethral meatus. It is apparent

that all the aforementioned risk factors for nosocomial candiduria also predispose a patient to bacteriuria.[28]

Patients with blood neoplasms form a group at high risk for invasive mycoses, of which invasive aspergillosis is commonest. Infections are normally located in the lower respiratory tract and nasal sinuses. Fungal spores are ubiquitously distributed in both outdoor and indoor environments and can easily enter the respiratory tract through inhalation. Depending on the number of conidia to which patients are exposed and the efficiency of the host defence, particularly the activity of alveolar macrophages and neutrophils the development of mycoses depends. Impaired immunological response, including periods of severe neutropenia, is a typical disorder noticed in patients with blood neoplasms and cytotoxic and immunosuppressive chemo- and corticosteroid therapies supporting that health state. Immunological response disorders are regarded as the main reason for frequent invasive aspergillosis in this group of patients. Despite the new antifungal drugs introduced in the last decade, mortality from invasive mycoses is high and ranges from 50–80%.[29]

Aspergillus species are the most common causes of invasive mold infection globally, particularly in severely immunosuppressed persons. The presence of *Aspergillus* species in the environment is very critical risk factor for nosocomial invasive aspergillosis, and outbreaks of infection have been associated with hospital construction or renovation activities, contaminated air-handling systems, and other environmental reservoirs. Inhalation and direct inoculation of tissue by spores are believed to be the most common routes of infection. Person-to-person transmission of *Aspergillus* species has not been reported elsewhere, with the exception of reports of direct donor-to-recipient transmission.[30]

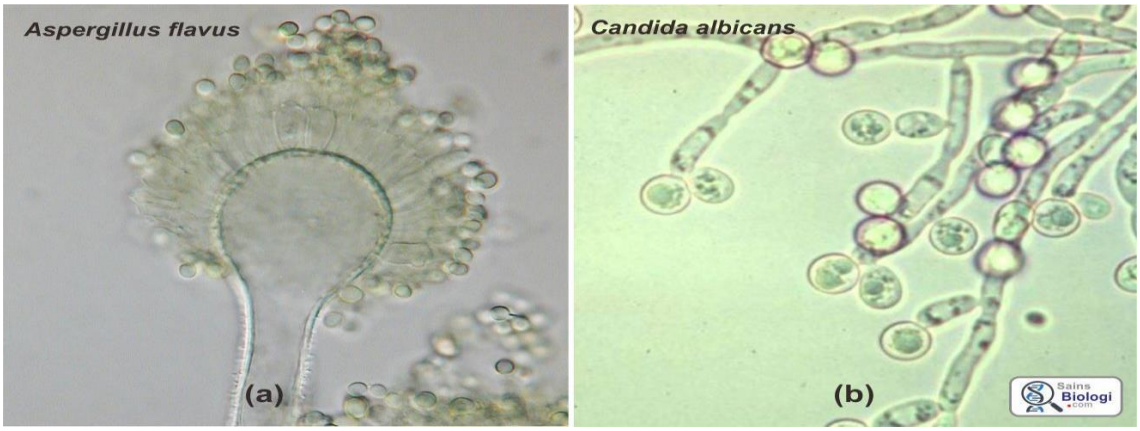


Fig 3: A)Aspergillus sp. B) Candida albicans

Risk factors for HCAIs:

High frequency of infection is associated with the use of invasive devices, particularly central lines, urinary catheters and ventilators. In a report from the USA NNIS system, 83% of episodes of hospital-acquired pneumonia were associated with MV, 97% of UTIs occurred in catheterized patients, and 87% of primary BSI in patients with a central line. Among adult ICU patients in high-income countries, pooled cumulative incidence densities of CR-BSI, urinary CR-UTI and VAP were 3.5 (95% CI 2.8-4.1) per 1000 CL-days, 4.1 (95% CI 3.7-4.6) per 1000 urinary catheter-days, and 7.9 (95% CI 5.7-10.1) per 1000 ventilator-days, respectively. [4]

Infections with *Aspergillus* spp. appear to be the result of both host susceptibility and environmental exposure to the fungus. The major risk factor related to the host is severe granulocytopenia. The longer the duration of severe granulocytopenia (<1,000 polymorphonuclear leukocytes/mm^3), the greater the risk of invasive disease. Patients receiving solid organ transplants also are at risk for invasive aspergillosis as a result of immunosuppression by corticosteroid therapy. However, the use of cyclosporine and FK506 has helped to decrease the degree of immunosuppression in these patients. Attempts to establish the efficacy of decreasing the period of severe granulocytopenia in these patients by

administration of granulocyte-stimulating factor or cytokines are underway. Additionally, the role of prophylactic intranasal, oral, or systemic antifungal drugs is being evaluated.[31]

Prior antibiotic therapy, hospitalization for 5 days or, MV for ≥5 days ($p<0.0001$), supine head position, reintubation, and impaired consciousness are significant risk factors for VAP.[12]

As per the study done by Mehta A, et al., an overall rate was 4.4%, and 9.06 HCAIs per 1000 ICU-days. The CVC-BSI rate was 7.92 per 1000 catheter-days; the VAP rate was 10.46 per 1000 ventilator-days; and the CAUTI rate was 1.41 per 1000 catheter-days. Overall, 87.5% of all *S. aureus* HCAIs were caused by methicillin-resistant strains, 71.4% of *Enterobacteriaceae* were resistant to 2nd gen cephalosporins; 28.6% of the *P. aeruginosa* strains were resistant to florquinolones, 64.9% to 3rd generation cephalosporins and 42.0% to imipenem. Length of stay of patients was 4.4 days for those without HCAI, 9.4 days for those with CVC-BSI, 15.3 days for VAP and 12.4 days for those with CAUTI.[19]

Microbiological profiles of HCAI:

A review of microbiological patterns of HCAI from 28 different studies conducted in low income and middle income countries reported gram-negative rods as the most common hospital acquired isolates, both in mixed patient populations and in high-risk patients. The most common pathogens isolated were *S. aureus* in mixed patient populations, and *Acinetobacter* spp. in high-risk patients. Acinetobacter was the second most common organism identified for VAP (24.0%) and, unexpectedly, for BSI (17.7%). *S. aureus* was the most frequent cause of both SSI and BSI.[4]

The U.S. National Healthcare Safety Network (NHSN) indicates that gram-negative bacteria are responsible for >30% of HCAIs, and these bacteria also predominate in cases of VAP (47%) and UTIs (45%). In ICUs, about 70% of the infections are caused solely by gram-negative bacteria. A range of gram-negative organisms are responsible for hospital-acquired infections, the *Enterobacteriaceae* family being the most commonly identified group overall. Unfortunately, MDR organisms, including *P. aeruginosa*, *A. baumannii*, and extended-

spectrum β-lactamase (ESBL)–producing or carbapenemase-producing *Enterobacteriaceae*, are increasingly being reported globally.[32]

According to annual report by NHSN about antimicrobial resistance patterns among pathogens causing device-associated and procedure-associated HCAIs. As much as 16% of all HCAIs were associated with the following MDR pathogens: MRSA (8% of HCAIs), vancomycin-resistant *E. faecium* (4%), carbapenem-resistant *P. aeruginosa* (2%), extended-spectrum cephalosporin-resistant *K. pneumoniae* (1%), extended-spectrum cephalosporin-resistant *E. coli* (0.5%), and carbapenem-resistant *A. baumannii*, *K. pneumoniae*, *K. oxytoca*, and *E. coli* (0.5%). Nationwide, the majority of units reported no HCAIs due to these antimicrobial-resistant pathogens.[33]

As per study done by Gopalakrishnan R, et al., according to which antimicrobial resistance is an increasing problem globally as well as in Indian hospitals. An increasing prevalence of MDR-O (multi-drug resistant organisms) such as MRSA, ESBL (extended spectrum beta lactamase) producing *Enterobacteriaceae* and carbapenem-resistant *Pseudomonas* and *Acinetobacter* has been reported from various Indian centres. The study demonstrated that gram-negative pathogens such as ESBL-producing *Enterobacteriaceae* and carbapenem-resistant *Pseudomonas* and *Acinetobacter* are common in Indian hospitals. Such a scenario appears likely to persist in the near future, with vital implications for antimicrobial selection and infection control policies. For instance, empirical coverage for any hospital acquired infection should include ESBL coverage, drastically restricting the number of antibiotic choices available. Carbapenem resistance in *Klebsiella* has also worryingly begun to emerge from 2005. Among gram-positive pathogens, MRSA is a significant pathogen while vancomycin-resistant *Enterococci* and *C. difficile* remain very uncommon, in sharp contrast to the situation in western hospitals. CR-BSI are usually caused by gram-negative bacteria with MRSA contributing a small proportion only.[34]

METHODOLOGY

MATERIALS AND METHODS

The study included 69 patients admitted in the medical wards and ICU under the Department of Medicine at RL JALAPPA HOSPITAL, TAMAKA, KOLAR who met the inclusion criteria and who

provided informed written consent for being part of our study. It was a prospective observational study

carried out between AUGUST 2018 and JULY 2019. The study protocol was approved by the

institutional ethical committee

INCLUSION CRITERIA

All patients aged > 18yrs admitted in the medical ward and ICU developing clinical evidence of infections that do not originate from a patient's original diagnosis after at least 48 hours of admission.

EXCLUSION CRITERIA

- Postoperative patients transferred in from surgical wards.

Sampling procedure:

- An observational prospective study in the department of medicine RLJH was conducted from April 2018 to March 2019. After obtaining approval from the ethical committee board and informed consent from patients and from patient's close relatives in case if patient is comatose, all patients who met inclusion and exclusion criteria were enrolled.

METHODS

Detailed history was taken and clinical examination was done. Case records were reviewed for the initial investigations, diagnosis and treatment. The invasive devices like Foleys catheter, ET tube, IV cannula and central vein catheter used and procedures done were recorded along with the duration of each device.

Clinical examination

Detailed clinical examination was done examining the following points:

1. Level of consciousness: Glasgow Coma Scale

2. Vitals:

    a. Pulse (beats per minute)

    b. Blood pressure (systolic/diastolic mmHg)

    c. Respirations (rate per minute)

    d. Rectal temperature (oC)

3. Pallor

4. Icterus

5. Cyanosis

6. Clubbing

7. Pedal edema

8. Hydration

9. Neck glands & neck veins

10. Build & nutrition

11. Respiratory system examination

12. Cardiovascular system examination

13. Central nervous system examination

14. Per abdomen examination

Collection and processing of clinical specimens

The following clinical specimens were collected for microbiological investigations:

* Blood culture: Venipuncture site was prepared with alcohol and povidone iodine and 5 mL of blood was collected per blood culture bottle using aseptic precautions. The blood was immediately dispensed into a biphasic brain-heart infusion blood culture bottle or in a BactAlert culture bottle. They were incubated at 37°C and vented for the first 2–3 days. For fungal culture, after 48 hrs, the bottles were slanted daily for 30 min, so that the BHI broth completely covered the BHI agar slant, after which they were again incubated upright. The slants were observed regularly for any fungal growth for 6 weeks.

* Urine: Early morning mid-stream, clean-catch urine sample was collected under aseptic precautions. Semi quantitative method using stand loop technique was used for gram stain.

Other samples such as sputum, CSF, pus, IV cannula tip culture, etc., for bacteriological and fungal cultures were collected where clinically indicated in the following methods.

* Sputum: Freshly expectorated sputum specimens were collected in sterile, wide-mouthed, screw-capped glass bottles from patients after thoroughly rinsing the oropharyngeal cavity with sterile distilled water. An adequate sputum sample was defined as having less than 10 squamous epithelial cells per low power field (100x) and 25 or more polymorphonuclear

leukocytes per high power field (1000x). They were homogenized by adding 10-20 sterile glass beads (diameter -2mm) and 3-5 mL sterile water, followed by shaking on a vortex mixer for 2 min. A loopful of each specimen was digested with 10% potassium hydroxide and examined microscopically for fungal elements

* CSF: Generally 5-6 mL of cerebrospinal fluid (CSF) was collected from patient in a sterile, screw-capped bottle by lumbar puncture which was carried out under complete aseptic precautions. CSF was centrifuged at 1500 rpm for 10 minutes and the sediment was separated to be used for microscopy and culture.

All of the collected clinical specimens were transported immediately to the laboratory for prompt microbiological investigations. If a delay in transport or processing was anticipated, the vials were left at room temperature.

Drug sensitivity and resistance of bacteria was tested using Kirby-Bauer Disk Diffusion Susceptibility Test

A symptomatic urinary tract infection must meet at least one of the following criteria:

1. Patient has at least 1 of the following signs or symptoms with no other recognized cause: fever (38.8C), urgency, frequency, dysuria or suprapubic tenderness and patient has a positive urine culture, that is, $>10^5$ microorganisms per cc of urine with no more than 2 species of micro-organisms.

2. Patient has at least 2 of the following signs or symptoms with no other recognized cause: fever (38.8C), urgency, frequency, dysuria or suprapubic tenderness and at least 1 of the following:

a. Pyuria (urine specimen with >10 white blood cell [WBC]/mm^3 or >3 WBC/high power field of unspun urine)

b. Organisms seen on Gram's stain of unspun urine

c. At least 2 urine cultures with repeated isolation of the same uropathogen (gram-negative bacteria or *Staphylococcus saprophyticus*) with $>10^2$ colonies/mL in non-voided specimens

d. $<10^5$ colonies/mL of a single uropathogen (gram-negative bacteria or *S. saprophyticus*) in a patient being treated with an effective antimicrobial agent for a urinary tract infection

e. Physician diagnosis of a urinary tract infection

f. Physician institutes appropriate therapy for a urinary tract infection.

Pneumonia: There are 3 specific types of pneumonia: clinically defined pneumonia (PNU1), pneumonia with specific laboratory findings (PNU2), and pneumonia in immunocompromised patients (PNU3)

Pneumonia 1: PNU1

Radiological findings: Two or more serial chest radiographs with at least one of the following:

1. New or progressive and persistent infiltrate

2. Consolidation

3. Cavitation

Signs/symptoms: FOR ANY PATIENT, at least one of the following:

1. Fever (38.8C or 100.48F) with no other recognized cause

2. Leukopenia (<4000 WBC/mm^3) or leukocytosis (>12,000 WBC/mm^3)

3. For adults >70 years old, altered mental status with no other recognized cause and

At least two of the following:

1. New onset of purulent sputum or change in character of sputum or increased respiratory secretions or increased suctioning requirements

2. New onset or worsening cough, or dyspnea, or tachypnea

3. Rales or bronchial breath sounds

4. Worsening gas exchange (e.g., O2 desaturation [e.g., PaO2/FiO2 #240], increased oxygen requirement, or increased ventilator demand)

Pnemonia2: PNU2

Radiological findings: Two or more serial chest radiographs with at least one of the following:

1. New or progressive and persistent infiltrate

2. Consolidation

3. Cavitation

Signs/Symptoms: At least one of the following

1. Fever (38.8C or 100.48F) with no other recognized cause

2. Leukopenia (4000 WBC/mm^3) or leukocytosis (>12,000 WBC/mm^3)

3. For adults >70 years old, altered mental status with no other recognized cause and at least one of the following:

1. New onset of purulent sputum or change in character of sputum or increased respiratory secretions or increased suctioning requirements

2. New onset or worsening cough or dyspnea or tachypnea

3. Rales or bronchial breath sounds

4. Worsening gas exchange (e.g., O2 desaturation [e.g., PaO2/FiO2 #240], increased oxygen requirement, or increased ventilator demand)

Laboratory: At least one of the following:

1. Positive growth in blood culture not related to another source of infection

2. Positive growth in culture of pleural fluid

3. Positive quantitative culture from minimally contaminated LRT specimen (e.g., BAL or protected specimen brushing)

4. >5% BAL-obtained cells contain intracellular bacteria on direct microscopic exam (example, Gram stain)

Pneumonia 3: PNU3

Radiological findings: Two or more serial chest radiographs with at least one of the following:

1. New or progressive and persistent infiltrate

2. Consolidation

3. Cavitation

Signs/symptoms: At least one of the following:

1. Fever (38.8C or 100.48F) with no other recognized cause

2. Leukopenia (<4000 WBC/mm^3) or leukocytosis (>12,000 WBC/mm^3)

3. For adults >70 years old, altered mental status with no other recognized cause and at least one of the following:

1. New onset of purulent sputum or change in character of sputum or increased respiratory secretions or increased suctioning requirements

2. New onset or worsening cough or dyspnea or tachypnea

3. Rales or bronchial breath sounds

4. Worsening gas exchange (e.g., O2 desaturation [e.g., PaO2/FiO2 #240], increased oxygen requirements, or increased ventilator demand)

Bloodstream infection must meet at least one of the following criteria:

1. Patient has a recognized pathogen cultured from 1 or more blood cultures and organism cultured from blood is not related to an infection at another site.

2. Patient has at least one of the following signs or symptoms: fever (38.8C), chills, or hypotension and signs and symptoms and positive laboratory results are not related to an infection at another site and common skin contaminant.

Arterial or venous infection must meet at least one of the following criteria:

Criterion 1: Patient has at least one of the following signs or symptoms with no other recognized cause:

Fever (38° C), pain, erythema, or heat at involved vascular site and more than 15 colonies cultured from intravascular cannula tip using semiquantitative culture method and blood culture not done or no organisms cultured from blood.

Criterion 2: Patient has purulent drainage at involved vascular site and blood culture not done or no organisms cultured from blood.

RESULTS

OBSERVATION AND RESULTS

RESULTS

Our study included 69 patients admitted in the medical wards and ICU under the Department
of Medicine at RL JALAPPA Hospital, Tamaka, Kolar who met the inclusion criteria and who provided informed written consent for being part of our study. Our study included 42 patients (60.9%) from ICU and 27 (39.1%) of the subjects were ward patients. 42(60.9%) of the patients were male and 27(39.1%) of the patients were female.

Table 1:- Distribution of subjects according to age group

AGE GROUP	Frequency	Percent
21-30yrs	9	13.0
31-40yrs	13	18.8
41-50yrs	9	13.0
51-60yrs	18	26.1
>60yrs	20	29.0
Total	69	100.0

Majority of the subjects 29% were >60yrs age group followed by 26.1% of the subjects were in 51-60yrs age group , 18% of the subjects were in 31-40yrs age group, 13% of the subjects were in 21-30yrs age group and 41-50yrs each . Minimum age was 23yrs and maximum was 86yrs with mean age 52.26yrs .

Figure 4:- Graph showing Distribution of subjects according to age group

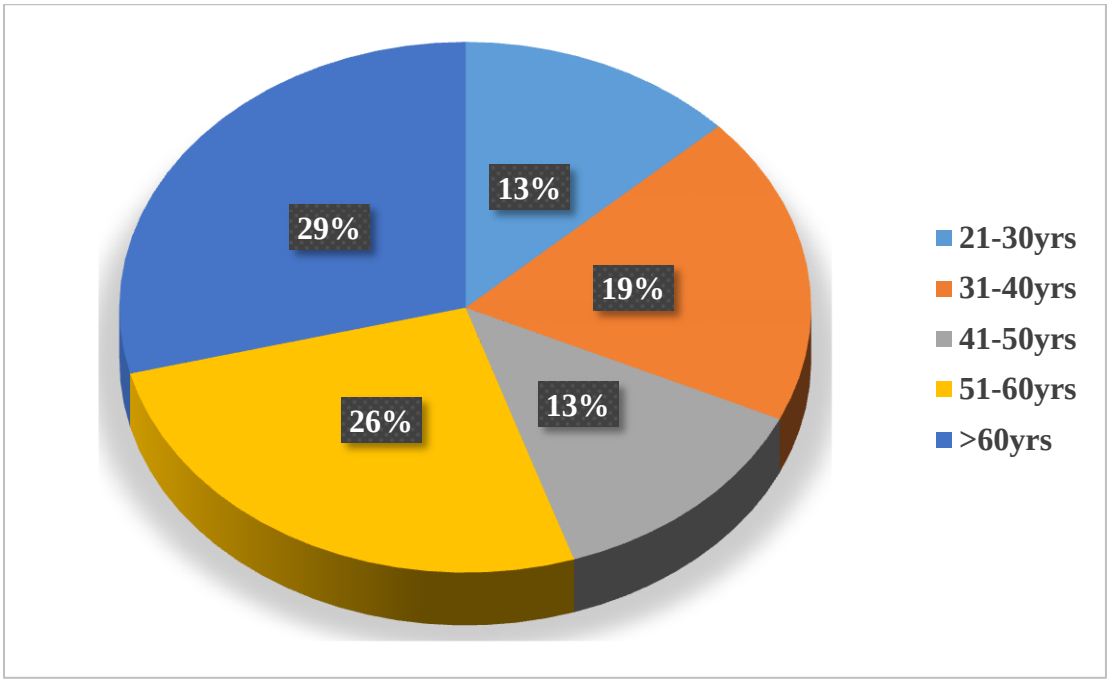


Table 2:- Distribution of subjects according to sex

	Frequency	Percent
Female	27	39.1
Male	42	60.9
Total	69	100.0

60.9% of the subjects were male and 39.1% of the subjects were female

Figure 5:- Graph showing Distribution of subjects according to sex

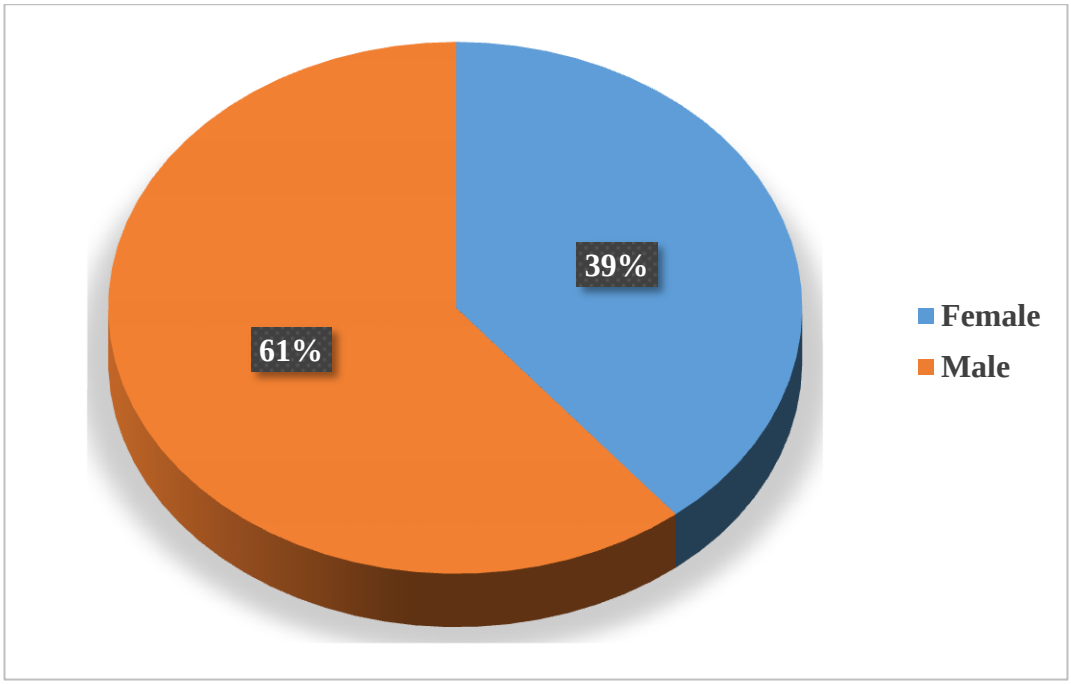


Table 3:- Distribution of subjects according to ICU/Wards

	Frequency	Percent
ICU	42	60.9
Wards	27	39.1
Total	69	100.0

60.9% of the subjects were ICU patients and 39.1% of the subjects were ward patients

Figure 6:- Graph showing Distribution of subjects according to ICU/Wards

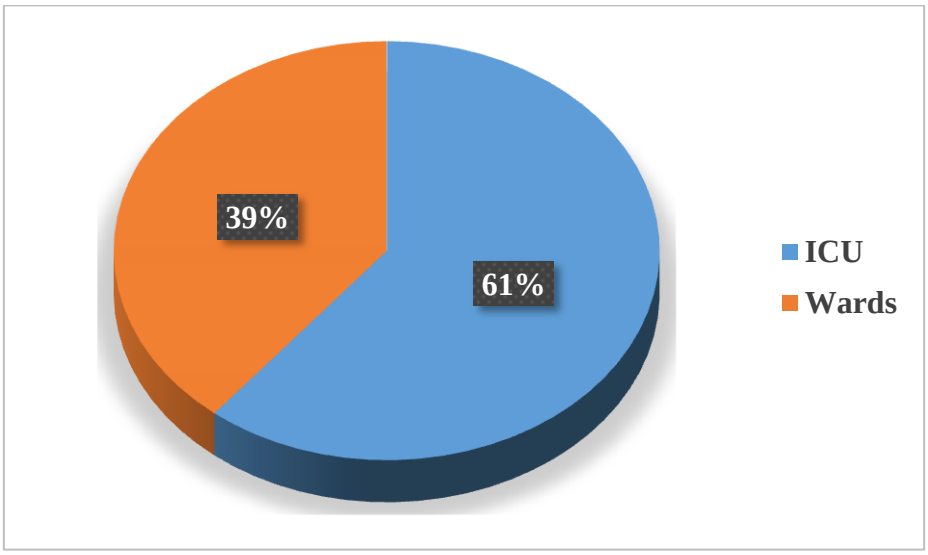


Table 4:- Distribution of subjects according to ICU/Wards and day on which symptoms of HAI developed

	ICU patients		Ward patients	
	N	%	N	%
≤ 5days	28	66.7%	18	66.7%
6-10days	12	28.6%	8	29.6%
>10days	2	4.8%	1	3.7%

P value 0.976,there was no statistically significant difference found between ICU and Ward patient with respect to day on which symptoms of HAI developed. In ICU Patients symptoms of HAI developed less than or equal to 5days in 66.7% followed by 28.6% between 6-10days and only 4.8% developed symptoms of HAI more than 10days In ward Patients symptoms of HAI developed less than or equal to 5days in 66.7% followed by 29.6% between 6-10days and only 3.7% developed symptoms of HAI more than 10days Overall among all subjects Day on which symptoms of HAI developed Minimum was 3days and maximum was19days. With average 5.43days to developed symptoms of HAI.

Figure 7:- Graph showing Distribution of subjects according to ICU/Wards and day on which symptoms of HAI developed.

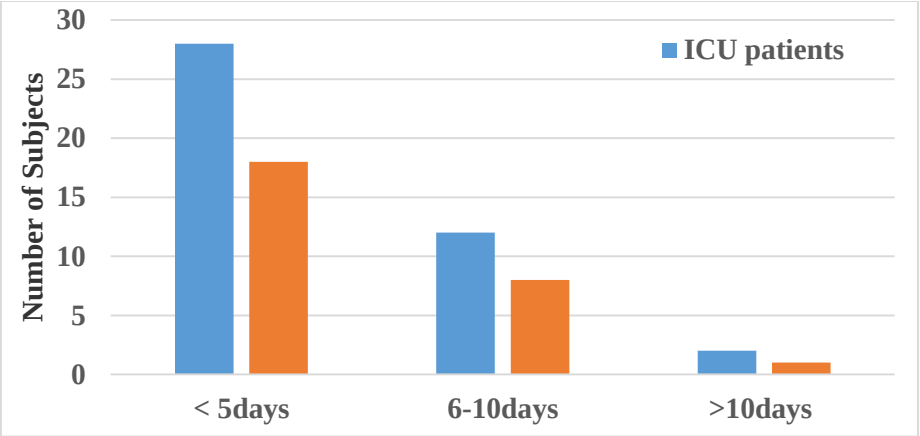


Table 5:- Frequency distribution of different HAI among ICU patients and Wards patients

	ICU patients		Ward patients	
	N	%	N	%
UTI	6	14.3%	1	3.7%
VAP	38	90.5%	0	0%
CLABSI	0	.0%	2	7.4%
Blood stream infection	6	14.3%	2	7.4%
Phlebitis	5	11.9%	22	81.5%

Among ICU patients most common HAI was VAP, it was present in 90.5% of ICU patients followed by 14.3% of ICU patients had UTI, 14.3% of ICU patients had Blood stream infection, 11.9% of ICU patients had Phlebitis.

Among ward patients most common HAI was Phlebitis, it was present in 81.5% of ward patients followed by Venous infection was present in 7.4% of ward patients, 7.4% of ward patients had Blood stream infection and 3.7% of ward patients had UTI.

Figure 8:- Graph showing Frequency distribution of different HAI among ICU patients and Wards patients.

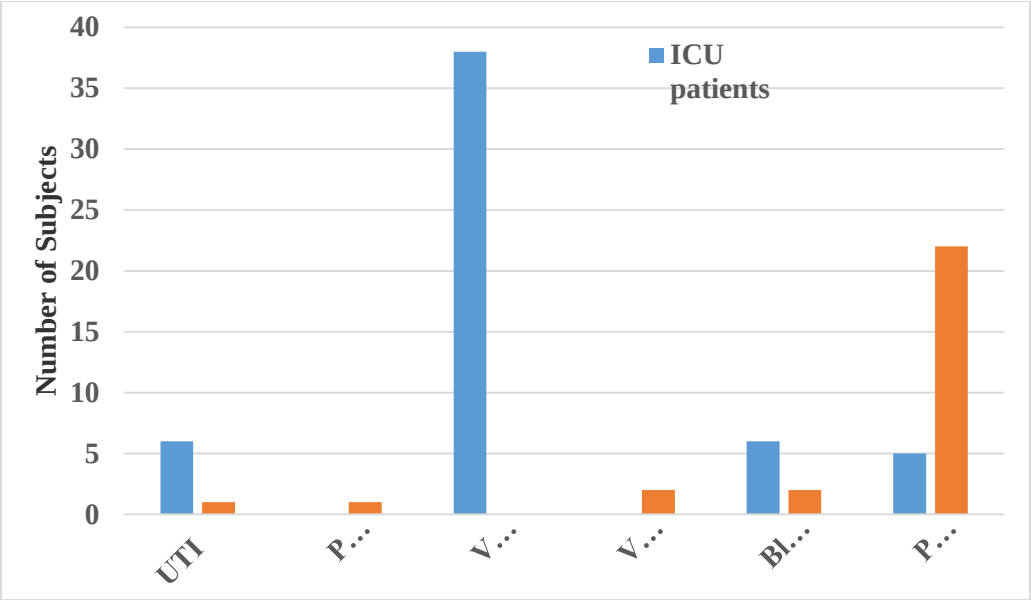


Table 6:-Comparison of mean Day on which symptoms of HAI developed among ICU patients and Wards patients

	ICU patients	Ward patients
	Mean days	Mean days
UTI	7.33	5.00
VAP	5.42	.
CLABSI	.	11.00
Blood stream infection	6.17	6.50
Phlebitis	4.60	5.45

Figure 9:- Comparison of mean Day on which symptoms of HAI developed among ICU patients and Wards patients.

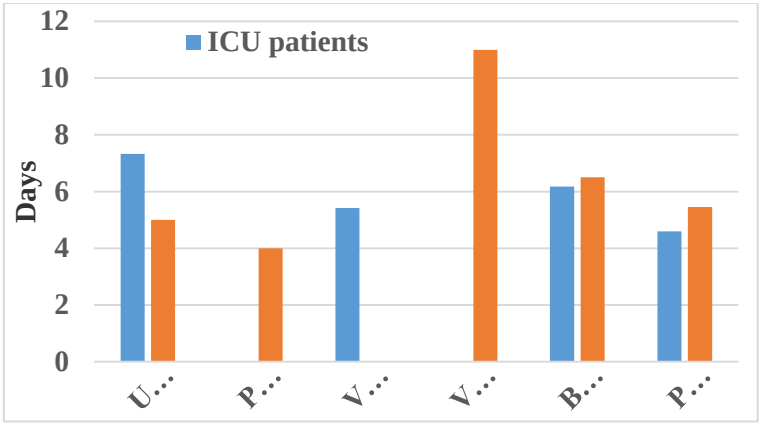
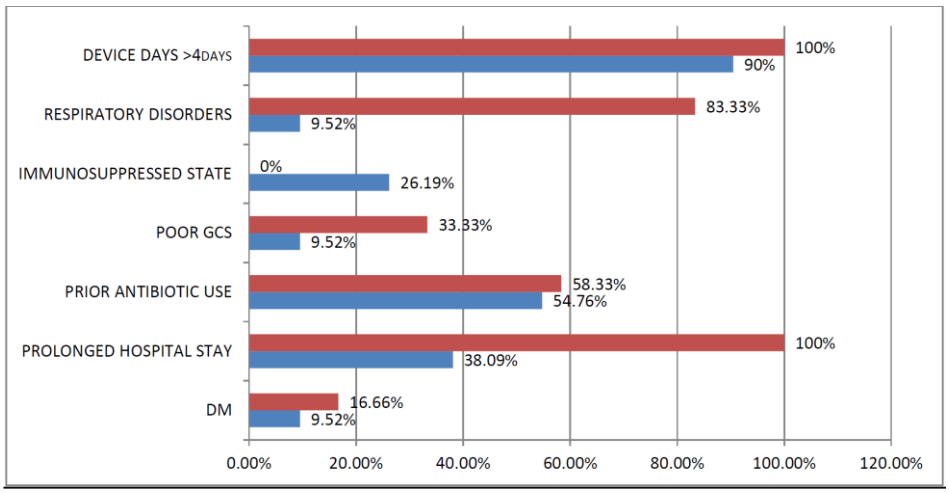


figure 10: predisposing factors for development of HCAI in medical ward and ICU



RED - ICU BLUE- WARD

In patients of medical ward who developed HAI, device days >4 days ,was the most common predisposing factor followed by prior antibiotic use, prolonged hospital stay(>5 days) , immunosuppressed states. Immunosuppressed states included patients with chronic liver disease , chronic kidney disease, connective tissue disorders and Tuberculosis. In ICU patients who had HAI, most common predisposing factors were device days >4 days , followed by respiratory disorders, prolonged hospital stay, prior antibiotic use. Patients with

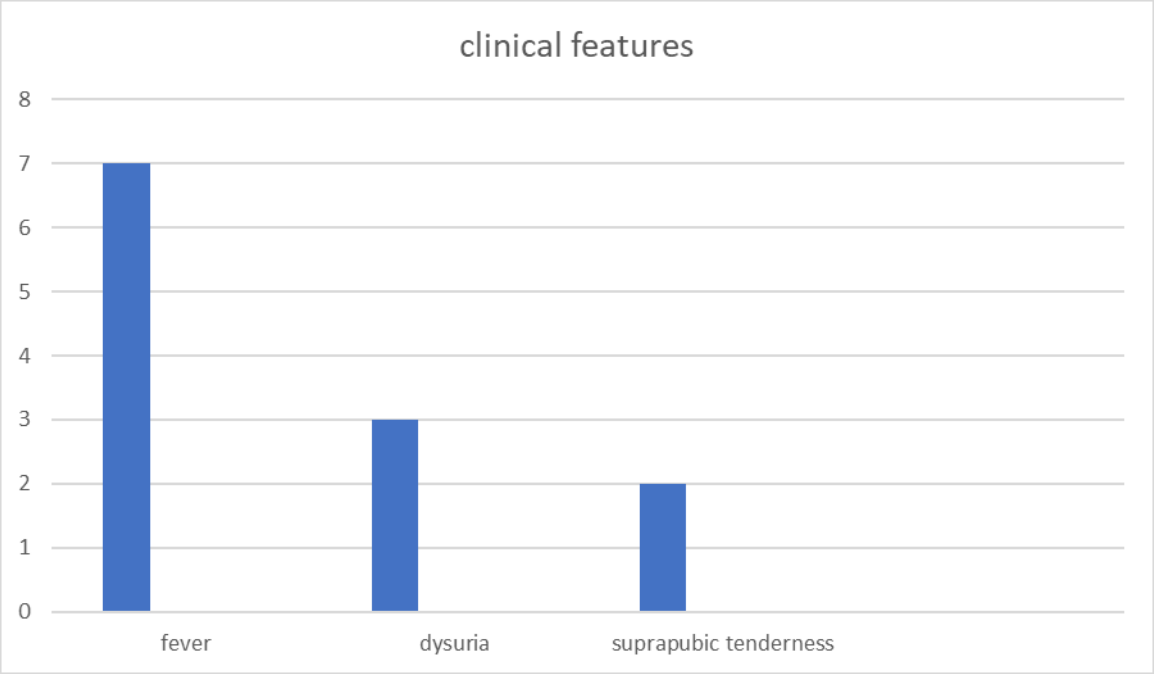
poor GCS (<9) accounted for 33.33%.

Table 7:-Comparison of clinical features among various HAI

	Fever	Dysuria	suprapubic tenderness	Increased tracheal secretions	Cough and expectoration	Pain/ erythema at cannula site
UTI	7(100%)	3(42.9%)	2(28.6%)	0	0	0
VAP	37(97.4%)	0	0	36(94.7%)	5(13.2%)	0
CLABSI	2(100%)	0	0	0	0	1(50%)
Blood stream infection	8(100%)	0	0	0	0	1(12.5%)
Phlebitis	19(70.4%)	0	0	0	1(3.7%)	24(88.9%)

Clinical features in UTI patients: - All patients had fever, 42.9% had dysuria, and 28.6% had suprapubic tenderness.

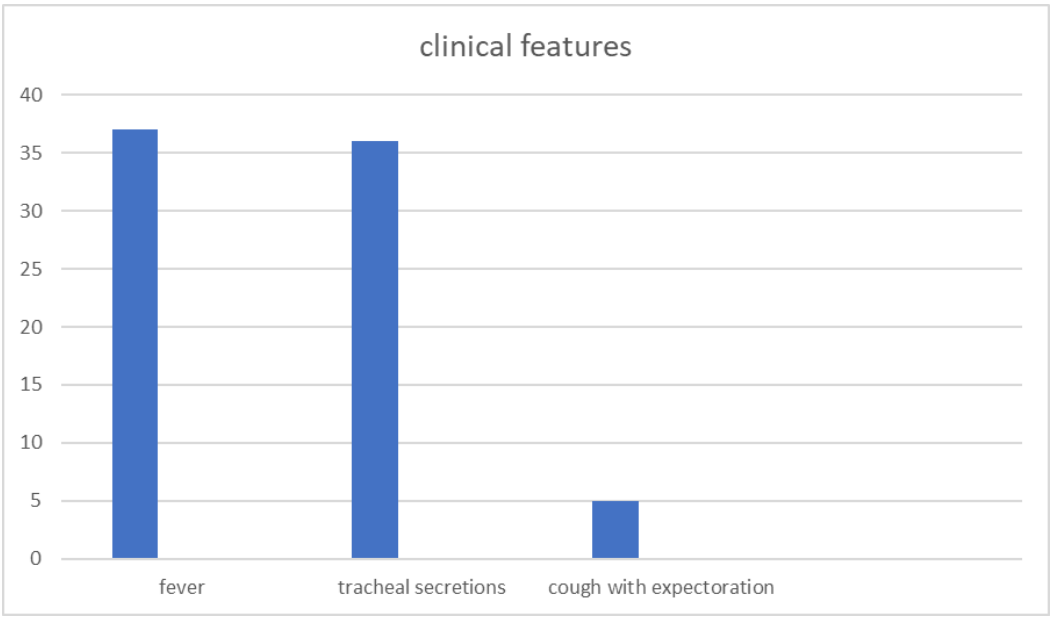
Figure 11: clinical features of UTI patients



Clinical features in HAP patients:-97.3% had fever, 91.9% had increased tracheal secretions, and 16.2% had Cough and expectoration.

Clinical features in VAP patients: - 97.4% had fever, 94.7% had increased tracheal secretions, and 13.2% had Cough and expectoration.

Figure 12: clinical features of VAP patients



Clinical features in venous infection patients:- All patients had fever and 50% Pain/ erythema at cannula site.

Clinical features in Blood stream infection patients:- All patients had fever, 62.5% had increased tracheal secretions

Clinical features in Phlebitis infection patients: - 88.9% had Pain/ erythema at cannula site, 70.4% had fever, 18.5% had increased induration and palpable venous cord.

Figure 13: clinical features of phlebitis

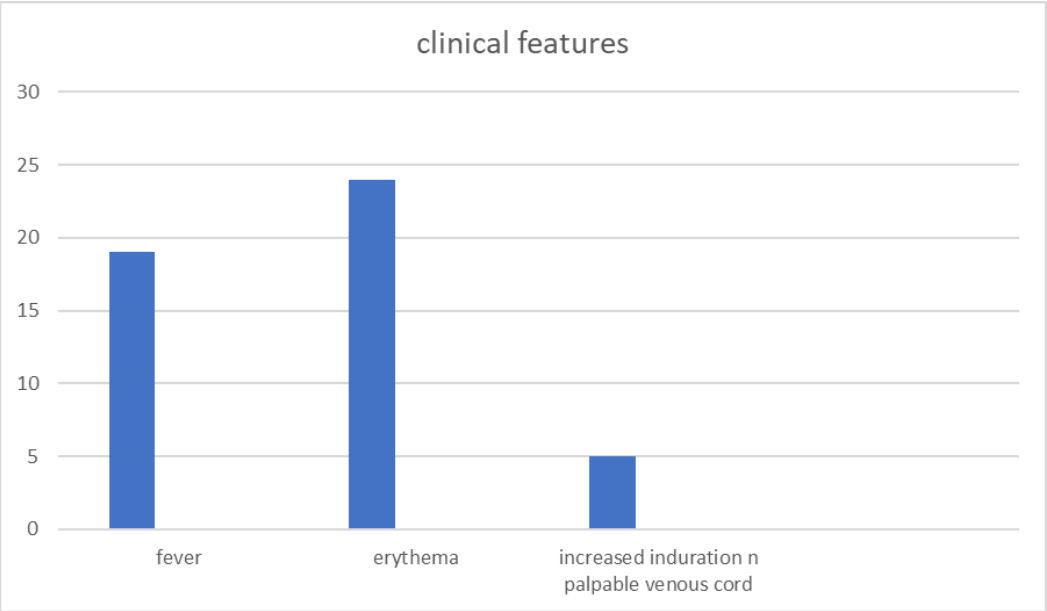


Table 8:-Comparison of devices used and related HAI

	IV cannula	Foleys	ET tube	Central line
	Count	Count	Count	Count
UTI	7	7	6	1
VAP	38	37	38	11
Venous infection	2	1	0	0
Blood stream infection	8	7	5	1
Phlebitis	27	6	5	1

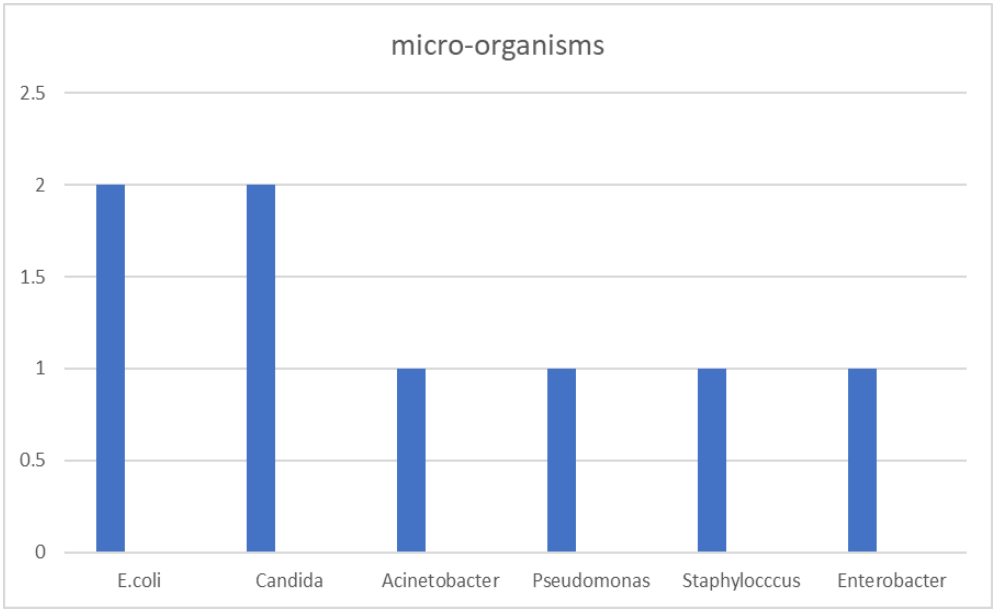
All the patients had IV cannula, 27 out of 69 patient had Phlebitis, 2 out of 69 patients had venous infection, 8 out of 69 patients had Blood stream infection. 46 patient had Foleys 7 out of 46 patients had UTI, 42patient had ET tube 38 had VAP, 1 out of 11 patients who had central line had blood stream infection

Table 9:- Microbiological profile of various HAI

Organism	UTI	VAP	CLABSI	Blood stream infection	Phlebitis
	Count	Count	Count	Count	Count
Acinetobacter	1	19	1	2	1
Klebsiella	0	15	2	3	3
Pseudomonas	1	4	0	1	1
Staphylococcus	1	4	0	2	1
Candida	2	3	0	0	0
E.coli	2	2	0	0	0
Enterobacter	1	5	0	3	1

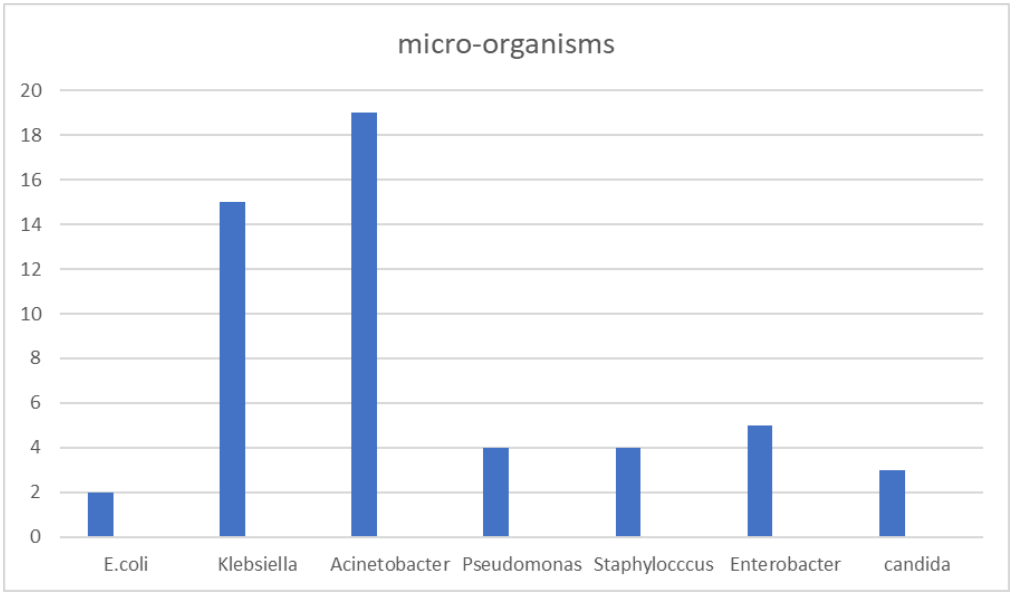
Among UTI patients culture showed positive for following organism , Ecoli were in 2 patients , Candida were in 2 patients, Acinetobacter were in 1 patients, Pseudomonas were in 1 patients Staphylococcus were in 1 patients , Enterobacter were in 1 patients.

Figure 14: CA-UTI organisms isolated



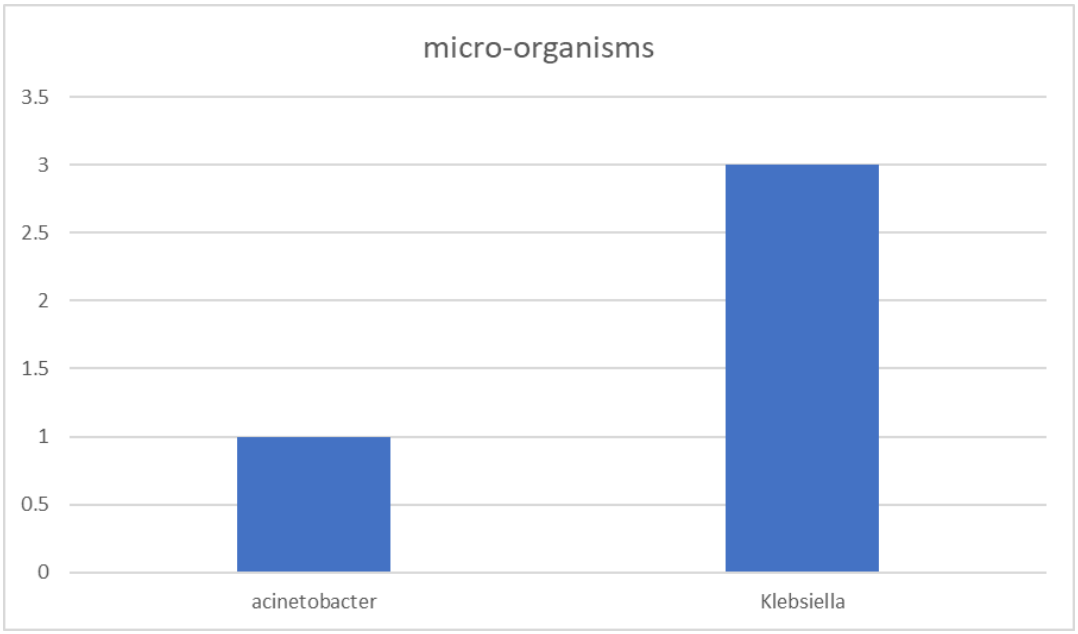
Among VAP patients culture showed positive for following organism , Acinetobacter were in 19 patients, Klebsiella were in 15 patients, Pseudomonas were in 4 patients, Staphylococcus were in 4 patients , Enterobacter were in 5 patients, Ecoli were in 2 patients , Candida were in 3 patients.

Figure 15: VAP organisms isolated



Among CLABSI patients culture showed positive for following organism , Acinetobacter were in 1 patients, Klebsiella were in 2patients.

Figure 16: Venous infection organism isolated



Among Blood stream infection patients culture showed positive for following organism , Acinetobacter were in 2 patients, Klebsiella were in 3 patients, Pseudomonas were in 1 patients, Staphylococcus were in 2 patients , Enterobacter were in 1 patients.

Figure 17: BSI-organisms isolated

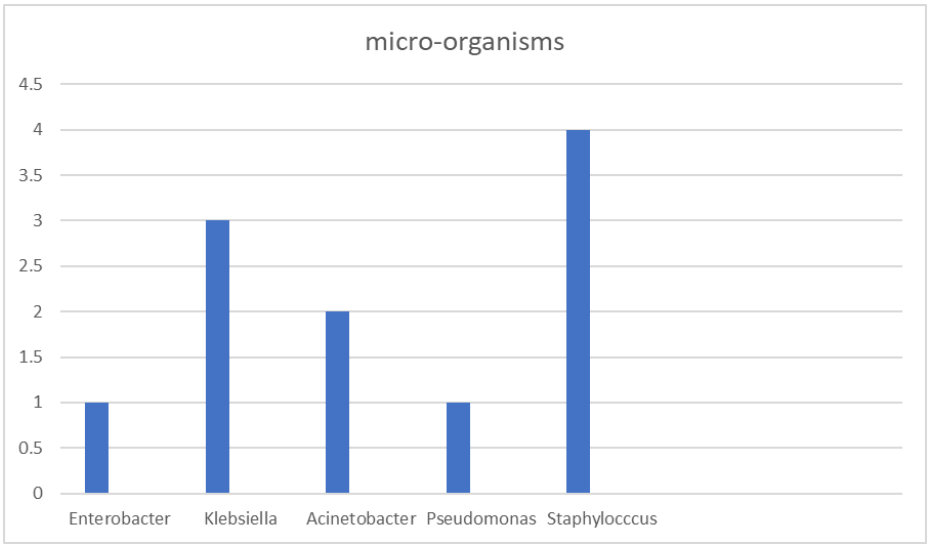


Table 10:- Drug sensitivity for various organism

	Acinetobacter	Klebsiella	Pseudomonas	Staphylococcus	E.coli	Enterobacter	Candida
Ceftazidime	**S**	**R**	**S**	**R**	**R**	**S**	
Cefipime	**S**	**S**	**S**	**R**	**R**	**S**	
Ceftazidime-clavulanic acid	**R**	**R**	**S**	**R**	**S**	**S**	
Ceftriaxone	**R**	**R**	**R**	**R**	**R**	**S**	
Imipenem	**S**	**R**	**S**	**S**	**S**	**S**	
Meropenem	**S**	**S**	**S**	**S**	**S**	**S**	
Piperacillin Tazobactum	**R**	**S**	**S**	**S**	**S**	**S**	
Amikacin	**R**	**R**	**S**	**R**	**S**	**S**	
Levofloxacin	**R**	**R**	**R**	**S**	**R**	**S**	
Cotrimoxizole	**R**	**R**	-	**S**	**R**	-	
Chloramphenicol	**R**	**R**	-	-	**S**	-	
Tetracycline	**R**	**R**	**R**	-	**R**	**S**	
Doxycycline	**R**	**R**	-	**S**	**R**	-	
Nitrofuratoin(URINE)	**R**	**R**	**R**	-	**S**	**S**	

Acinetobacter was sensitive for Ceftazidime,Cefipime,Imipenem,Meropenem.

Klebsiella was sensitive for Cefipime,Meropenem,Piperacillin-tazobactum.

Pseudomonas was sensitive for Cefipime,ceftazidime,Meropenem,Piperacillin-tazobactum.

Staphylococcus was sensitive for Cefipime,Piperacillin-tazobactume,Cotrimoxizole,Doxycycline

E.coli was sensitive for Piperacillin tazobactum,Chloramphenicol

Enterobacter was sensitive for CefaperzoneCeftriaxone,Meropenem,Piperacillin,Penicillin, Linezolid,Tetracycline,Nitrofuratoin.

Candida was sensitive for Fluconazole

Four patients who have developed VAP with Acinetobacter isolated in 3 patients and Klebsiella isolated in 1 patient showed pan resistant to all antibiotics. These patients were started on Colistin. Outcome of these patients was that all patients succumbed due to complications of underlying diseases.

DISCUSSION

DISCUSSION

Hospital-acquired infections (HAI) are a major public health problem all over the world, but particularly in developing nations. However, estimates of the world burden are hampered by a scarcity of data adequately describing endemic infections at national and regional levels, particularly in developing world with resource-limited settings.[2]

Our study included 69 patients admitted in the medical wards and ICU under the Department

of Medicine at RL JALAPPA Hospital, Tamaka, Kolar who met the inclusion criteria and who provided informed written consent for being part of our study. Our study included 42 patients (60.9%) from ICU and 27 (39.1%) of the subjects were ward patients. 42(60.9%) of the patients were male and 27(39.1%) of the patients were female. Majority of the subjects 29% (20 patients)were >60yrs age group followed by 26.1%(18) of the subjects were in 51-60yrs age group , 18% of the subjects were in 31-40yrs age group, 13% (9)of the subjects were in 21-30yrs age group and 41-50yrs each. Minimum age was 23yrs and maximum was 86yrs with mean age 52.26yr. In our study, advanced age and gender were not significantly associated with development of infections in ICU; similar outcome has been shown by Meric et al. and Agarwal et al.[34,35] Few studies have shown that advanced age is a predisposing factor.[36]

In our study there were 27 ward patients and 42 ICU patients. In medical ward, most common HCAI was Venous infection (phlebitis) (81.5%) followed by BSI (7.4%), UTI (3.7%) and HAP (3.8%). Our study showed different results to the study done by B.R. Devrajani where common causes of healthcare associated infection in the medical ward patients were urinary tract infection (UTI) in 34 (68%) patients,

bloodstream infection (BSI) in 19 (38%), lower respiratory tract infection (pneumonia) in 09 (18%) patients, upper respiratory tract infection sinusitis in 4 (8%) patients and otitis in 3 (6%) patients, bronchitis in 07 (14%) patients and tracheitis in 3 (6%) patients.7 CA-UTI was the most common HAI in medical ward in other studies like Iranian study by Mehrdad Askarian et al 37 where 43 cases of UTI, 29 cases of SSI, 13 cases of BSI, and 13 cases of Pneumonia were detected overall. In a study done by Safdar N, the common HCAI in hospitalized patients in medical wards is the venous infection.[36]

In our study common infections in ICU patients were VAP followed by BSI and UTI in that order. Similar results were found in the study by Shabina Habibi et al at a tertiary care hospital in northern India involving 182 patients where pneumonia accounted for (77%); urinary tract infection (24%) and blood stream infection (24%).9 Similarly in a Turkish study by H. Leblebicioglu[38] involving ICU patients, most common HAI was VAP (47.6%), followed by CVC BSI (30.2%) and CAUTI (22.2%). According to the International Nosocomial Infection Control Consortium(INICC) overall data, VAP represented 41.0% of all HCAIs, followed by CVC-BSI (30.0%) and CAUTI (29.0%).34 However according to the Indian study by A. Mehta et al, HCAI distribution was: CVC-BSI 61.3%, VAP 29.6% and CAUTI 9.0% in ICU. 19 According to Columbian study involving ICU patients by Carlos A´

lvarez Moreno et al, CVC-associated BSIs represented 47.4% of all device-associated

infections, VAP represented 32.3%, and CAUTI represented 20.3%.18 In the Iranian study by Mehrdad Askarian et al[37], CA-UTI was the most common HAI in ICU followed by VAP and

BSI. This was possibly because more stringent precautions were taken in our ICUs during the procedures like Central line insertion and Foley s insertion.

Majority of the patients had either an acute severe disease such as CVA, poisoning or chronic disease like CLD, COPD and CKD . This is in agreement with various studies which have shown severity of underlying illness and immunosuppressed state to be a significant predisposing factor for development of HAI.[39,40] In our study, in patients of medical ward who developed HAI, device days >4 days, was the most common predisposing factor followed by prior antibiotic use, prolonged hospital stay (>5 days) immunosuppressed state and poor GCS (<9) . In ICU patients who had HAI, mostcommon predisposing factors were device days >4, followed by prolonged hospital stay , respiratory conditions, prior antibiotic use and poor GCS (<9) . In medical ward patients with Poor GCS most common infection was BSI, followed by UTI and HAP. In ICU patients with VAP, 20 out of 38 had received prior antibiotics, 30 patients had underlying respiratory disorders, and 14 patients had Poor GCS. Similar risk factors i.e. longer duration of hospital stay, exposure to intravascular or urinary catheter, mechanical ventilation and use of antimicrobials, were found in the studies.[41,42] In our study most of the patients (92.8%) developed HAI in first 10 days in medical ward and almost fifty percent developed HCAI in 5 days. In ICU, 38 out of 42 patients developed HAI between 6-10 days and only four within first five days. This may be due to lack of proper aseptic precautions during the procedures in medical ward. The other reason may be venous infections which mostly developed early accounting for >85% of all the infections in medical ward. In the study by B.R. Devrajani[7], the mean time from hospitalization to the onset of fever was approximately 9 days whereas, the it was7 days in the study of Arbo et al 43. and the 13 days in the study of Filice et al.[44] CA-UTI, accounted for the 10.1% of HAI. In our

study, total 49 patients, 7 from ward and 42 from ICU had Foley's catheter. Out of 7 CA-UTI, 1 was from ward and 6 from ICU. Amongst CAUTI patients in ICU Males and females were equal. Other studies have shown that CAUTI is more common in females. In ward only 1 patient had CA-UTI. This less percentage of CA-UTI compared to other studies is possibly due to stringent aseptic precautions taken in ICU. All patients who developed CA-UTI had Foley s for >4 days. In our study all 7 patients had fever, 3 had burning micturition, and 2 had suprapubic tenderness. This fits with CDC definition. Thus only clinical features were sufficient for diagnosing CA-UTI. Culture was positive in all patients in ICU and wards. Most common organism cultured was E.coli followed by candidia, Acinetobacter, pseudomonas, staphylococcus and Enterobacter. Recent U.S. data indicate that E. coli is the most common etiologic gram-negative organism, in CAUTI followed in descending order of frequency by P. aeruginosa, klebsiella species, enterobacter species, and A. baumannii.[31] In a study by B.R. Devrajani, from Pakistan out of 34 urinary tract infection, 32 had their urine culture positive. In Gram negative organisms E. coli was cultured in 02 (28.5%) patients, Acinetobacter in 01 (12%), Enterobacter in 01 (12%) patients and pseudomonas in 01 (12%). In gram positive organisms coagulase negative staphylococci including staphylococcus epidermis was cultured in 01 (12%) patients. Candida albicans was grown in 02 (28.5%) patients.Like in B.R. Devrjani's study, our culture positivity was 100% as compared to 96% . However the profile of bacteriological agents cultured were similar. We did have a 2 cases of candida infection in CAUTI. Further fungal infections are common when broad spectrum and higher antibiotics remove the bacteria and normal commensal fungi become pathogenic. In our patients we used only third generation cephalosporins presumptively. Broad spectrum antibiotics increase the risk of fungal infections.[45]

VAP accounted for 55.07% of total infections. In our study, 38 patients from ICU and none from medical ward had ET tube. In patients with VAP in ICU, prolonged hospital stay, prior antibiotics and days>4 days were prominent risk factors . This is similar to the studies by Kalidas R et al[13], Noyal J [46]et al and Lynch JP et al[47]. All patients with VAP in our study, developed VAP after 4 days. Average day of development of VAP was 5.4 days in ICU. There were only 2 (5%) patients with early VAP in our study which may be due to the fact that the total no. of VAP in our study were only 38. Similar findings were seen in a study by Kalidas Rit et al[13] involving 140 adult patients, where most of the patients had late onset VAP (60.7%) with average number of days for onset around 8 days. In our study, all patients had fever, increased tracheal secretions and 30 out 0f 38 patients had new onset chest infiltrates. 28 of the patients had TLC>12,000 and 2 of them had TLC <4000. In patients with VAP most

common organism isolated was Acinetobacter similar to other studies[13,38]. In our study, culture positivity was 100% similar to 100% in the Kalidas et al 13study. Culture showed growth of 19 Acinetobacter organisms, which except for 3 patients were sensitive to Carbapenems and these 3(15.7%) were resistant to even carbapenems and classified as multi drug resistant and were started on colistin. 15 patients had Klebsiella positive growth which was sensitive to fourth generation cephalosporin , piperacillin- tazobactam, carbapenems and one (6%) patient with multi drug resistance. This was unlike the study by Kalidas R et al , involving 140 patients, where thirty (69.7%) of them were multidrug resistant (MDR), among which ESBL contributed 23.25%, MBL 30.23%, AmpC beta-lactamases 9.30%, and to methicillin resistant S. aureus (MRSA) contributed 6.97%. This was probably because in our hospital, 3rd generation Cephalosporins along with Tazobactum-Pipericillin

were the most commonly used empirical antibiotics. Linezolid, Vancomycin, and Carbapenems are reserve drugs which are used mostly after culture reports are available. In our study, mortality rate of VAP was 18% (6 patients) in ICU. All 4 patients with Multi drug resistance VAP died of complications with GCS 3/15 from the time of admissions.

Out of 6 patients who died in ICU, 2 are cases of poisoning, 1 was viral haemorrhagic fever, 1 COPD type 2 respiratory failure and 2 had subdural haematoma with GCS of 3/15. According to the previous studies, the mortality rate for VAP ranges from 24% to 50%, and can reach as high as 76% in specific settings or when lung infection is caused by high-risk pathogens.48 In our study, out of 6 patients who died of VAP, 3 had Acinetobacter and 1 had Klebsiella resistant to all antimicrobials and were started on Colistin. Mortality was related to severity of

underlying illness and not to high risk pathogens.

HAP accounted for <1.5% of HAIs. In our study we had 1 patient with HAP in medical ward with GCS 15 admitted for CKD and patient had Fever and new onset cough with expectoration and had new chest infiltrates on chest x ray. Patient has Acinetobacter positive culture sensitive to Crabepenems. In Devrjani's study in patients with pneumonia 14% were unconscious, 4% were semiconscious and 20% were conscious. Most common organism cultured in sputum was E. coli 06 (12%) patients, pseudomonas in 3 (6%) patients, enterobacter in 01 (2%) patients, klebsiella in 02 (4%) patients, staphylococcus aureus in 03 (6%) patients and anaerobes in 04 (8%) patients.

Venous infection was the second most common HAI in our study accounting for 39.17%. All

69 patients in our study had IV cannula. 22 venous infections were from medical ward. 5 venous infection was found in ICU out of 42 patients. Most of the venous infections developed within 10 days of hospitalization. 5 out of 27 venous infections were found in patients with CVA and other CNS conditions. All 27 patients had device days >4 days and 7 of them had received prior antibiotics. All patients had fever and pain and redness was present in 84.61% patients. Purulent discharge from the site was not there in any of our patients. No patient has positive growth on IV CANNULA tip culture. All others who didn't fulfil CDC criteria, were diagnosed clinically. The diagnosis was thrombophlebitis. Studies of short peripheral venous catheters show that the incidence of thrombophlebitis and bacterial colonization of catheters increases when catheters are left in place more than 72 hours. However, rates of phlebitis are not significantly different in peripheral catheters left in place 72 hours compared to 96 hours.20 PVC-associated thrombophlebitis rates range from 2 to 80%. There are some intrinsic risk factors directly associated with thrombophlebitis, such as a high haemoglobin level, a thrombophilic predisposition and poor vein quality. It has been shown unequivocally that insertion and maintenance of PVCs by untrained or inexperienced healthcare workers increases the risk of thrombophlebitis[49].

In our study, 8 patients developed BSI, 5 from ICU and 3 patients from medical ward developed BSI. Among 8 patients with BSI 4 patients has Central catheter placed for various reasons. In patients with BSI, in medical ward all patients received antibiotics before development of BSI. Most patients who developed BSI are immune compromised with long standing co-morbidities like CKD. One patient was a burns patient.

According to the study by Andrew F. Shorr et al[50], prior administration of cephalosporins, increased length of ICU stay and packed RBC transfusion was associated with increased risk of BSI. In our study 5 out of 8 patients with BSI had prolonged hospital stay. Half of the study population received pRBC transfusions at some point during their ICU stay. In our study, among Blood stream infection patients culture showed positive for following organism, Acinetobacter were in 2 patients, Klebsiella were in 3 patients, Pseudomonas were in 1 patients, Staphylococcus were in 2 patients, Enterobacter were in 1 patients. There was no candida growth in our study. Candida spp. caused 8% of hospital-acquired BSIs reported to NNIS during 1986-1989 and 1992-1999.[51,52,53] Pawar et al.[54]showed that 11.4% of CRBSI was caused by Candida species in cardiothoracic surgical ICUs and Subba Rao et al [55] demonstrated that 20% of catheter-related infections occurred in pediatric ICUs. Evidence suggests that the actual burden of nosocomial candidemia in Indian hospitals is underrecognized[56]. Around 9600-20,000 patients per year die in ICUs in the United States due to catheter-associated BSI. The attributable mortality for these BSIs has ranged from 12-25% in prospective studies. In our study no patient died due to hospital acquired BSI. The density of skin flora at the catheter insertion site is a major risk factor for CR-BSI. Many authorities recommend that central catheters be placed in a subclavian site in preference to a jugular or femoral site to reduce the risk of infection.

In our study All the patients had IV cannula, 27 out of 69 patient had Phlebitis, 2 out of 69 patients had venous infection, 8 out of 69 patients had Blood stream infection. 46 patient had

Foleys 7 out of 46 patients had UTI, 42patient had ET tube 38 had VAP, 1 out of 11 patients who had central line had blood stream infections. So, device associated infection rate was highest with ET tube followed by Foleys.

In our study, out of 69 patients 53 were culture positive which is same as compared to other studies.[7,13,19] Gram negative organisms were the most common organisms. Acinetobacter was the most common organism followed by Klebsiella. In fungi, Candida was the most common organism. Acinetobacter accounted for 36.23% of all HAIs, Klebsiella 30.43%, Enterococcus 14.49% Pseudomonas 10.14%, Staphylococcus aureus 11.54%, Ecoli 5.7% , and candida accounted for 7.24%. According to study by A. Mehta et al[19], overall 27.3% of all HAI was caused by Pseudomonas spp., 6.2% was caused by Acinetobacter spp, 3.1% was caused by S.aureus infection, 46.4% were caused by Enterobacteriaceae, 8.2% were caused by Candida spp; 2.6% were caused by Enterococcus spp, 3.1% were caused by Stenotrophomonas spp, 2.6% were caused by coagulase negative-staphylococci; and 0.5% were caused by

Streptococcus spp. In our study Acinetobacter was sensitive for Cefaperzone, Cefipime, Imipenem, Meropenem with about 17% (3) of the patients showing resistant to even carbapenems. Klebsiella was sensitive for Cefipime, Meropenem, Piperacillin. 1 patient (6%) showing pan resistance. Pseudomonas was sensitive for Cefipime, Cefebatum, Meropenem, Piperacillin. Staphylococcus was sensitive for Cefipime,Piperacillin,Cotrimoxizole,Doxycycline. E.coli was sensitive for Piperacillin,Chloramphenicol, levofloxacin.Enterobacter was sensitive for CefaperzoneCeftriaxone,Meropenem,Piperacillin,Penicillin,Linezolid,Tetracycline, Nitrofuratoin. Candida was sensitive for Fluconazole where as in study by A. Mehta

et al[19], in Acinetobacter spp 28.6% were resistant to ciprofloxacin, 64.9% to ceftazidime, 42.0% to imipenem and 42.6% to piperacillin-tazobactam; In Klebsiella 87.5% were resistant to methicillin, 71.4% were resistant to ceftriaxone, 74.1% to ceftazidime and 42.6% to piperacilline tazobactam.

In a study by Kalidas Rit et al[13], involving 140 patients, thirty (69.7%) of them were multidrug resistant (MDR), there was a high rate of resistance to all major antibiotics commonly used in ICUs. In the study by Teresa Cardoso et al[36] age >60 years, hospitalization in the previous year (in the last 4 to12 months), previous antibiotic therapy (last month) were found to be significantly and independently associated with infection by MDR pathogens. Patients with infection by MDR microorganisms had a significantly higher rate of inadequate initial antibiotic therapy, but did not have a longer hospital length of stay or higher hospital mortality rate.[36] In our study overall MDR microorganisms are positive in 4 patients, this less number maybe due to less use of higher antibiotics as empirical coverage in our hospital.

We had only 5 cases of HAI caused by fungal infections. They were all UTI and VAP caused by Candida. Other studies have found more number of fungal HAIs.[7,13] This is probably because of minimal use of higher antibiotics(only Ceftriaxone was used as presumptive treatment) in our hospital. It is well documented that prior surgery, acute renal failure, previous yeast colonization, neutropenia, antibacterial agents, parenteral nutrition and central venous catheters were associated with an increased risk of invasive candidiasis, whilst prior antifungals were protective according to Eggimann, P et al study[45].

In our study 53 out of 69 cases of HAI were culture positive. Majority of our diagnosis of

HAI s was made on clinical grounds. This is similar to the Iranian study[42] and has been Mdiscussed as a cause for underreporting of HAIs. The need for developing more sensitive clinical criteria is required for diagnosing subclinical infections.

CONCLUSION

CONCLUSION ANDRECOMMENDATION

- From this study, we concluded that patients admitted to medical ward and ICU are at a risk of acquiring healthcare associated infection.
- In medical ward, most common HAI was Venous infection (PHLEBITIS)followed by BSI, CA-UTI, HAP.
- Most common HCAIs in ICU patients were VAP followed by BSI and UTI.
- In our study most of the patients (66.7%) developed HCAI in first 5 days in medical ward and almost fifty percent developed HCAI in 5 days. In ICU, 5 out of 12 patients developed HAI between 6-10 days and only three within first five days
- In our study, in patients of medical ward who developed HAI, device days >4 days was the most common predisposing factor followed by prior antibiotic use, prolonged hospital stay, immunosuppressed states.
- In ICU patients who had HAI , most common predisposing factors were device days >4 days , followed by respiratory disorders, prolonged hospital stay, prior antibiotic use.
- In our study gram negative organisms were the most common organisms. Acinetobacter was the most common organism followed by Klebsiella. In fungi, Candida was the most common organism.
- More stringent aseptic measures should be taken in medical wards and ICU to reduce incidence of HAIs.
- Guidelines for empirical antibiotics should be formed in hospital according to the sensitivity and resistance patterns. Indiscriminate use of antibiotics should be avoided.
- For diagnosis of HAI, clinical guidelines should be formed especially in resource limited settings.

SUMMARY

SUMMARY

Our study included 69 patients admitted in the medical wards and ICU under the Department of Medicine at RL JALAPPA Hospital, Tamaka, Kolar who met the inclusion criteria and who provided informed written consent for being part of our study. Our study included 42 patients (60.9%) from ICU and 27 (39.1%) of the subjects were ward patients. 42(60.9%) of the patients were male and 27(39.1%) of the patients were female.

- Among ICU patients most common HAI was VAP, it was present in 90.5% of ICU patients followed by 14.3% of ICU patients had UTI, 14.3% of ICU patients had Blood stream infection, 11.9% of ICU patients had Phlebitis.
- Among ward patients most common HAI was Phlebitis, it was present in 81.5% of ward patients followed by CLABSI was present in 7.4% of ward patients, 7.4% of ward patients had Blood stream infection and 3.7% of ward patients had UTI.
- In patients of medical ward who developed HAI, device days >4 days ,was the most common predisposing factor followed by prior antibiotic use, prolonged hospital stay(>5 days) ,immunosuppressed states. Immunosuppressed states included patients with chronic liver disease , chronic kidney disease, connective tissue disorders and Tuberculosis.
- In ICU patients who had HAI, most common predisposing factors were device days >4 days, followed by respiratory disorders, prolonged hospital stay, prior antibiotic use. Patients with poor GCS (<9) accounted for 33.33%.
- Four patients who have developed VAP with Acinetobacter isolated in 3 patients and Klebsiella isolated in 1 patient showed pan resistant to all antibiotics. These patients were started on Colistin. Outcome of these patients was that all patients

succumbed due to complications of underlying diseases.

- From this study, we concluded that patients admitted to medical ward and ICU are at a risk of acquiring healthcare associated infection. In medical ward, most common HAI was PHLEBITIS followed by BSI, CA-UTI. Most common HCAIs in ICU patients were VAP followed by BSI and UTI. Acinetobacter was the most common organism followed by Klebsiella. In fungi, Candida was the most common organism.
- More stringent aseptic measures should be taken in medical wards and ICU to reduce incidence of HAIs. Guidelines for empirical antibiotics should be formed in hospital according to the sensitivity and resistance patterns. Indiscriminate use of antibiotics should be avoided.

BIBLIOGRAPHY

REFERENCES

1. Collins AS. Preventing Health Care-Associated Infections. In: Hughes RG, editor. Patient Safety and Quality: An Evidence-Based Handbook for Nurses. Rockville (MD): Agency for Healthcare Research and Quality (US); 2008. Apr, [Accessed April 1, 2018]. Chapter 41.

2. Revelas A. Healthcare - associated infections: A public health problem. Niger Med J. 2012;53(2):59–64.

3. Available from: https://www.who.int/gpsc/country_work/gpsc_ccisc_fact_sheet_en.pdf. Accessed on Oct 20, 2019.

4. Available from: https://apps.who.int/iris/bitstream/handle/10665/80135/9789241501507_eng.pdf;jsessionid=B54891BAAB559AFE98CE14663235A7D0?sequence=1. Accessed on Oct 20, 2019.

5. Horan TC, Andrus M and Dudeck MA. 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.

6. Haque M, Sartelli M, McKimm, et al. Health care-associated infections – an overview. Infection and Drug Resistance. 2018;11:2321–2333.

7. Devrajani BR, Shah SZ, DevrajaniT, et al. Nosocomial Infections inMedical Ward.Four Months Descriptive Study in a Tertiary Care Hospital. World Journal of Medical Sciences. 2009:4(1):13-17.

8. Donowitz LG, Wenzel RP and Hoyt JW.High risk of hospital-acquired infection in the ICU patient.Critical Care Medicine.1982;10(6):355-357.

9. Habibi S, Wig N, Agarwal S, et al. Epidemiology ofnosocomial infections in medicine intensive care unit at a tertiary care hospital in northernIndia. Trop Doct. 2008;38(4):233-5.

10. Kollef M. SMART approaches for reducing nosocomial infections in the ICU.Chest. 2008;134(2):447-456.

11. Hooton TM, Bradley SF, Cardenas DD, et al. Diagnosis, Prevention, and Treatment of Catheter-Associated Urinary Tract Infection in Adults:2009 International Clinical Practice Guidelinesfrom the Infectious Diseases Society of America.Clinical Infectious Diseases. 2010;50:625–663.

12. Rit K, Chakraborty B, SahaR, et al. Ventilator associated pneumonia in a tertiary care hospital in India: Incidence, etiology, risk factors, role of multidrug resistant pathogens: International Journal of Medicine & Public Health. 2014;4(1):p51-56.

13. Rello J, Allegri C, Rodriguez A, et al. Risk Factors for Ventilator-associated Pneumonia by Pseudomonas aeruginosa in Presence of Recent AntibioticExposure. Anesthesiology.2006; 105:709–1

14. Akkoyunlu Y, Öztoprak N, Aydemir H, et al. Risk factors for nosocomialpneumonia in intensive care units of a University Hospital :Journal of Microbiology and Infectious Diseases. 2013;3(1):3-7.

15. Wisplinghoff H, Bischoff T, Tallent SM, et al. Nosocomial bloodstream infections in UShospitals: analysis of 24,179 cases from a prospective nationwide surveillance study. ClinInfect Dis. 2004;39(3):309–17.

16. Gahlot R, Nigam C, Kumar V, et al. Catheter-related bloodstream infections. Int J CritIllnInj Sci. 2014;4(2):162–167.

17. Mermel LA, Farr BM, Sherertz RJ, et al. Guidelines for the management of intravascular catheter–relatedinfections. Clin Infect Dis.2001; 32:1249–72.

18. Moreno CA, Rosenthal VD, Olarte N, et al. Device-associated infection rate and mortalityin intensive care units of 9 Colombian hospitals: findings of the International NosocomialInfection Control Consortium. Infect ContrHospEpidemiol. 2006;27(4):349–356.

19. Mehta A, Rosenthal VD, Mehta Y, et al. Device-associated nosocomial infection rates in intensive care units of seven Indian cities. J HospInfect. 2007;67(2):168-74.

20. O'Grady NP, Alexander M, Burns LA, et al. Guidelinesfor the prevention of intravascular catheter-related infections. Clin Infect Dis. 2011;52:e162–93.

21. Ibrahim EH, Sherman G, Ward S, et al. The Influence of Inadequate Antimicrobial Treatment of Bloodstream Infections on Patient Outcomes in the ICU Setting: CHEST. 2000;118:146–155.

22. Datta P, Rani H, Chauhan R, et al. Health-care-associated infections: Risk factorsand epidemiology from an intensive care unit in Northern India. Indian journal ofanaesthesia. 2014;58(1):30-35.

23. Parameswaran R, Sherchan JB, VarmaM, et al. Intravascularcatheter-related infections in an Indian tertiary care hospital J Infect DevCtries 2011;5(6):452-458.

24. Weber DJ, Peppercorn A, Miller MB, et al. Preventing healthcare-associated Aspergillus infections:review of recent CDC/HICPAC recommendations. Medical Mycology. 2009;47(1):S199–S209.

25. Marr KA. Antifungal prophylaxis in hematopoietic stem cell transplant recipients.CurrOpin Infect Dis. 2001;14:423–6.

26. Eggimann P, Garbino J and Pittet D. Epidemiology of Candida species infections in criticallyill non-immunosuppressed patients. Lancet Infect Dis. 2003;3:685–702.

27. Yang SP, Chen YY, Hsu HS. A risk factor analysis of healthcare-associated fungal infections in an intensive care unit: a retrospective cohort study: BMCInfectious Diseases. 2013;13:10. doi:10.1186/1471-2334-13-10.

28. LundstromT and Sobel J. Nosocomial Candiduria: A Review: ClinicalInfectious Diseases 2001; 32:1602–7.

29. Nawrot U, Zubkiewicz LU,Pajączkowska M, et al. FungalContamination of Air in the Department of Haematology: Polish J. of Environ. Stud. 2010;19(5):967-971.

30. Pegues DA, Lasker BA, McNeil MM, et al. Cluster of Cases of Invasive Aspergillosis in a Transplant Intensive Care Unit: Evidence of Person-to- Person Airborne Transmission: ClinicalInfectious Diseases. 2002;34:412–6.

31. Fridkin SK and William R. JARVIS2 Epidemiology of Nosocomial Fungal Infections.American Society for Microbiology CLINICAL MICROBIOLOGY REVIEWS. 1996;9(4):499–511.

32. Peleg AY and Hooper DC. Hospital-Acquired Infections Due to Gram-Negative Bacteria. The new England journal of medicine 1804 n englj med 362;19 nejm.org may 13, 2010.

33. Hidron AI, Edwards JR, Patel J ,et al. NHSN annual update: antimicrobial-resistant pathogens associated with healthcare-associatedinfections: annual summary of data reported to the National Healthcare Safety Network at the Centers for Disease Control and Prevention, 2006–2007. Infect Control Hosp Epidemiol. 2008;29(11):996–1011.

34. Gopalakrishnan R and Sureshkumar D. Changing Trends in AntimicrobialSusceptibility and Hospital Acquired Infections Over an 8 Year Period in a Tertiary CareHospital in Relation to Introduction of an Infection Control Programme. SUPPLEMENT TOJAPI. 2010:58.

35. Agarwal R, Gupta D, Ray P, Aggarwal AN, Jindal SK. Epidemiology, risk factors and

outcome of nosocomial infections in a respiratory intensive care unit in north India. J Infect 2006;53:98‐105.

36. Meric M, Willke A, Caglayan C, Toker K. Intensive Care Unit‐.Acquired infections: Incidence, risk factors and associated mortality in a Turkish University Hospital. Jpn J Infect Dis 2005;58:297‐302.

37. Teresa Cardoso, Orquídea Ribeiro, Irene César Aragão et al :Additional risk factors for infection by multidrug-resistant pathogens in healthcare-associated infection: a large cohort study: BMC Infectious Diseases 2012, 12:375

38. Mehrdad Askarian, Hilda Mahmoudi, and Ojan Assadian et al. Incidence of nosocomial infections in a big university affiliated hospital in Shiraz, Iran: A 6 month experience. Int J Prev Med. Mar 2013; 4(3): 366–372.

39. Leblebicioglu H, Rosenthal VD, Arikan OA, Ozgultekin A, Yalcin AN, Koksal I, Usluer G, Sardan YC, Ulusoy S. Turkish branch of INICC: device-associated hospital-acquired infection rates in Turkish intensive care units: findings of the International Nosocomial Infection Control Consortium (INICC). J Hosp Infect 2007;65:251–257

40.National Institute of Child Health and Human Development, NIH, DHHS. Primary immunodeficiency (99-4149) Washington, DC: U.S. Government Printing Office; 1999.

41.Andrews LB, Stocking C, Krizek T, et al. An alternative strategy for studying adverse events in medical care. Lancet. 1997 Feb 1;349(9048):309–13.

42. Jroundi I, Khoudri I, Azzouzi A,et al:. Prevalence of hospital-acquired infection in a Moroccan university hospital. Am J Infect Control. 2007;35:412–416.

43. Askarian M, Yadollahi M, Assadian O. Point prevalence and risk factors of hospital acquired infections in a cluster of university-affiliated hospitals in Shiraz, Iran. J Infect Public Health.2012;5:169–176

44.Arbo, M.J., M.J. Fine, B.H. Hanusa, T. Sefcik and W.N. Kapoor, 1993. Fever of nosocomial origin: etiology, risk factors and outcomes. Am. J. Med.,95: 505-512

45. Filice, G.A., M.D. Weiler, R.A. Hughes and D.N. Gerding, 1989. Nosocomial febrile illnesses in patients on an internal medicine service. Arch. Intern. Med., 149: 319-324.

46.Eggimann, P., Garbino, J. & Pittet, D. (2003). Epidemiology of Candida species infections in critically ill non-immunosuppressed patients. Lancet Infect Dis 3, 685–702.

47. Joseph NM, Sistla S, Dutta TK, Badhe AS, Rasitha D, Parija SC. Ventilator-associated pneumonia in a tertiary care hospital in India: Role of multi-drug resistant pathogens. J Infect Dev Ctries. 2010;4:218–25.

48.Lynch JP III. Hospital-acquired pneumonia: Risk factors, microbiology, and treatment. Chest 2001;119:373S-84S.

49.Chastre J, Fagon JY. Ventilator-associated pneumonia. Am J Respir Crit Care Med 2002;165:867-903.

50. Peripheral venous catheters: an under-evaluated problem

Walter Zingg , Didier Pittet: International Journal of Antimicrobial Agents 34S (2009)

51. Transfusion Practice and Blood Stream Infections in Critically Ill Patients Andrew F. Shorr, MD, MPH; William L. Jackson, MD; Kathleen M. Kelly, MD; Min Fu, MS, MBA; and Marin H. Kollef, MD (CHEST 2005; 127:1722–1728).

52. Centers for Disease Control and Prevention. National Nosocomial Infections Surveillance (NNIS) System report, data summary from January 1990-May 1999, issued June 1999. Am J Infect Control 1999;27:520-32.

53.Schaberg DR, Culver DH, Gaynes RP.Major trends in the microbial etiology of nosocomial infection. Am JMed 1991;91:72S-75S.

54. Banerjee SN, Emori TG, Culver DH, et al. Secular trends in nosocomial primary bloodstream infections in the United States, 1980-1989. National Nosocomial Infections Surveillance System. Am J Med 1991;91:86-9.

55.Mandakini Pawar, Yatin Mehta, Pawan Kapoor, Sharma J, Gupta A, Trehan N Central venous catheter-related blood stream infections: incidence, risk factors, outcome, and associated pathogens. J Cardiothorac Vasc Anesth 2004: 18: 304-08.

56.Subba Rao, Joseph MP, Lavi R, Macaden R Infections related to vascular catheters in a pediatric intensive care unit. Indian Pediatrics 2005:42: 667-72.

57.Sahni V, Agarwal SK, Singh NP, Anuradha S, Sikdar S, Wadhwa A, Kaur R Candidemia--an under-recognized nosocomial infection in Indian hospitals. J Assoc Physicians India. 2005; 53:607-11.

58. V. Krčméry Jr.; F. Matejička: Documented Fungal Infections after Prophylaxis or Therapy with Wide Spectrum Antibiotics: Relationship Between Certain Fungal Pathogens and Particular Antimicrobials? : Journal of Chemotherapy. 1999;385-90.

ANNEXURES

ANNEXURES

PROFORMA

1. IP No: 2. Date:

3.Serial No:

4. Name: 5. Age: 6.Gender:

7. Occupation:

8. Date of admission:

9. Address 10. phone no:

11. Chief complaints:

12. Past history:

13. Drug/ Treatment therapy:

14. Personal history:

15. General physical examination (at admission):

PR:	BP:	Temp:
Resp rate:	SpO_2:	
Pallor:	Icterus:	Cyanosis:
Clubbing:	Lymphadenopathy:	Oedema:

16. Systemic examination:

CVS:

RS:

PA:

CNS:

17. Diagnosis:
18. Duration of hospital stay:
19. Type of devices used :
20. Procedures done:
21. INVESTIGATIONS:

- COMPLETE BLOOD COUNT
- RENAL FUNCTION TEST
- URINE ROUTINE AND CULTURE
- BLOOD CULTURE AND SENSITIVITY
- TRACHEAL ASPIRATE CULTURE AND SENSITIVITY
- SPUTUM CULTURE AND SENSITIVITY
- CSF ANALYSIS

SIGNATURE

PATIENT INFORMATION SHEET

Study title: **Risk factors, Clinical and Microbiological profile of health care associated infections in patients admitted in Medical wards.**

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

- **Details-**

Health care associated infections are deemed the most frequent adverse event threatening patients safety worldwide. Both developed and developing countries are faced with burden of health care associated infections. Urinary tract infections, surgical site infections, blood stream infections, hospital acquired and ventilator associated pneumonia are 4 major site specific infections. There are only few studies focusing on profiles of healthcare associated infections in developing countries like India. Our study proposes to be descriptive study of risk factors, clinical and microbiological profiles of HAIs in patients admitted in medical ward and ICU.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee.

There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact

Dr. JITHENDRA CHAITANYA (Post graduate)
Department of General Medicine
SDUMC , KOLAR
Contact NO : 9490645990

INFORMED CONSENT FORM

Name of the investigator: DR. JITHENDRA CHAITANYA GUBBALA

Name of the organisation: R L JALAPPA HOSPITAL AND RESEARCH CENTRE ATTACHED TO SRI

DEVARAJ URS MEDICAL COLLEGE

Name of the participant: SI no:

I Mr./Mrs. , have been explained in my own understandable language, that I will be included in a study which is "CLINICAL AND MICROBIOLOGICAL PROFILE OF HEALTH CARE ASSOCIATED INFECTIONS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL WARDS AND ICU" being conducted in RL JALAPPA HOSPITAL.

If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. We will collect the treatment and relevant details from your hospital record. This information collected will be used for only dissertation and publication. This study has been reviewed by the institutional ethical committee. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

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

I have read or have been read to me and understood the purpose of the study, the procedure that will be used, the risk and benefits associated with my involvement in the study and the nature of information that will be collected and disclosed during the study.

I have had the opportunity to ask my questions regarding various aspects of the study and my questions are answered to my satisfaction.

I, the undersigned agree to participate in this study and authorize the collection and disclosure of my personal information for dissertation.

Subject name

(Parents / Guardians name)

DATE: SIGNATURE /THUMB IMPRESSION

ತಾಳ್ಮೆಯ ಸಂಬಂಧಗಳಿಂದ ತಿಳಿವಳಿಕೆ ಸಮ್ಮತಿ ನಮೂನೆ

ಸಂಶೋಧಕರ ಹೆಸರು: ಡಿಆರ್. ಜಿಥೆಂದ್ರ ಚೆತನ್ಯಾ ಜಿ

ಸಂಸ್ಥೆಯ ಹೆಸರು: ಶ್ರೀ ದವರಾಜ್ ಯುಆರ್ಎಸ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ಗೆ ಸೇರ್ಪಡೆಗೊಂಡ ಆರ್ ಎಲ್ ಜಲಪಪಾ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ

ಪಾಲ್ಗೊಳ್ಳುವವರ ಹೆಸರು: SI ಸಂಖ್ಯೆ:

ನಾನು ಶ್ರೀ / ಶ್ರೀ. , ಪೋಷಕ / ಗಾರ್ಡಿಯನ್ / ಹೆಸರಿನ ರೋಗಿಗೆ ಸಂಬಂಧಿಸಿರುವವರು ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವಂತಹ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಿದ್ದಾರೆ, ನನ್ನ ರೋಗಿಯನ್ನು ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುತ್ತದೆ, ಇದು "ವೈದ್ಯಕೀಯ ಆರೋಗ್ಯ ಮತ್ತು ಆರೋಗ್ಯದ ಆರೋಗ್ಯದ ಆರೋಗ್ಯದ ಆರೋಗ್ಯದ ಆರೋಗ್ಯದ ಒಳನೋಟಗಳನ್ನು ಒಳಗೊಳ್ಳುವ ವೈದ್ಯಕೀಯ ವೈದ್ಯರು ಮತ್ತು ICU" ಆರ್ಎಲ್ ಜಲಪಾ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ನಡೆಸಲಾಗುತ್ತಿದೆ.

ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ರೋಗಿಗೆ ಒಪ್ಪಿಗೆ ನೀಡಲು ನನ್ನನ್ನು ಆಮಂತ್ರಿಸಲಾಗಿದೆ. ಈ ಡಾಕ್ಯುಮೆಂಟ್ನಲ್ಲಿನ ಮಾಹಿತಿಯು ಪಾಲ್ಗೊಳ್ಳಲು ಇಲ್ಲವೇ ಎಂಬುದನ್ನು ನಿರ್ಧರಿಸಲು ನನಗೆ ಸಹಾಯ ಮಾಡುವ ಉದ್ದೇಶವಾಗಿದೆ. ಪ್ರಧಾನ ಸಂಶೋಧಕನೊಂದಿಗೆ ನಾನು ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನನ್ನ ಅನುಮಾನಗಳನ್ನು ಸ್ಪಷ್ಟಪಡಿಸಿದೆ.

ನನ್ನ ರೋಗಿಯ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಂತೆ ಕೇಳಲಾಗಿದೆ ಏಕೆಂದರೆ ನನ್ನ ರೋಗಿಯ ಅರ್ಹತಾ ಮಾನದಂಡವನ್ನು ಪೂರೈಸುತ್ತದೆ.

ನನ್ನ ರೋಗಿಯ ರಕ್ತ ಮಾದರಿಯ ಗೊತ್ತುಪಡಿಸಿದ ಪರೀಕ್ಷೆಗಳನ್ನು ನಿರ್ವಹಿಸಲು ನಾನು ಡಾ. ಜಿತೇಂದ್ರ ಚೆತನ್ಯಾ ಜಿಗೆ ಮನವಿ ಮಾಡಿ ಮತ್ತು ಅಧಿಕಾರ ನೀಡುತ್ತೇನೆ. ಕೆಳಗಿನ ನನ್ನ ಸಹಿ ನನ್ನ ಅರ್ಹತೆಯನ್ನು ಹೊಂದಿದ್ದು, ಈ ಪರೀಕ್ಷೆಯ ಪ್ರಯೋಜನಗಳು, ಅಪಾಯಗಳು ಮತ್ತು ಮಿತಿಗಳನ್ನು ಅರ್ಹ ಆರೋಗ್ಯ ವೃತ್ತಿಪರರಿಂದ ನನ್ನ ತೃಪ್ತಿಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಭಾಗವಹಿಸುವಿಕೆ ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿರುತ್ತದೆ ಮತ್ತು ಮಾದರಿ ಸಂಗ್ರಹಣೆಗೆ ಯಾವುದೇ ಪಾವತಿಯಿಲ್ಲ. ಎಲ್ಲಾ ಪರೀಕ್ಷಾ ಫಲಿತಾಂಶಗಳನ್ನು ವೈದ್ಯಕೀಯ ಗೌಪ್ಯತೆಯೊಂದಿಗೆ ಪರಿಗಣಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಕಾನೂನಿನ ಅಗತ್ಯವಿದ್ದರೆ ಹೊರತುಪಡಿಸಿ ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ.

ನನ್ನ ರೋಗಿಗಳ ಗೌಪ್ಯತೆಯನ್ನು ಕಾಪಾಡಿಕೊಳ್ಳುವವರೆಗೂ ನನ್ನ ರೋಗಿಗಳಿಗೆ ವೈದ್ಯಕೀಯ ಸಂಶೋಧನೆ, ಪರೀಕ್ಷೆ ಉರ್ಜಿತಗೊಳಿಸುವಿಕೆ ಅಥವಾ ಶಿಕ್ಷಣಕ್ಕಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ ಎಂದು ನನ್ನ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ನನ್ನ ರೋಗಿಯನ್ನು ಈ ಅಧ್ಯಯನದಿಂದ ಯಾವ ಸಮಯದಲ್ಲಾದರೂ ಹಿಂಪಡೆಯಲು ಮುಕ್ತವಾಗಿರುತ್ತೇನೆ ಮತ್ತು ಅದು ನನ್ನ ರೋಗಿಗಳ ಭವಿಷ್ಯದ ಕಾಳಜಿಯನ್ನು ಬದಲಿಸುವುದಿಲ್ಲ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ನಾನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಸ್ವೀಕರಿಸಿದ್ದೇನೆ. ಈ ಡಾಕ್ಯುಮೆಂಟಿನಲ್ಲಿ ಒದಗಿಸಿದ ಮಾಹಿತಿಯನ್ನು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ ಮತ್ತು ಪರೀಕ್ಷೆ, ಕಾರ್ಯವಿಧಾನ, ಸಂಬಂಧಿಸಿದ ಅಪಾಯ ಮತ್ತು ಪರ್ಯಾಯಗಳ ಬಗ್ಗೆ ನಾನು ಹೊಂದಿರುವ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ.

ವಿಷಯದ ಹೆಸರು:

ಪೋಷಕರ / ಪೋಷಕರ ಹೆಸರು:

ರೋಗಿಯ ಸಂಬಂಧಿತ ಮತ್ತು ದಿನಾಂಕದ ಸಹಿ / ಹೆಬ್ಬೆರಳು ಗುರುತು:

ಒಪ್ಪಿಗೆ ತೆಗೆದುಕೊಳ್ಳುವ ವ್ಯಕ್ತಿಯ ಸಹಿ ದಿನಾಂಕ: