

Original Article

Surveillance of healthcare associated infections at a tertiary care teaching hospital from 2014 to 2016: Prevalence, microbiological spectrum and antibiogram

Savitha N^{1*}, Anitha D¹, PM Beena²

1. Assistant Professor, Department of Microbiology, Sri Devaraj Urs Medical College, Sri Devaraj Urs Academy of Higher Education and Research, Kolar.
2. Professor & HOD, Department of Microbiology, Sri Devaraj Urs Medical College, Sri Devaraj Urs Academy of Higher Education and Research, Kolar.

Abstract

Background: Healthcare-associated infections have emerged as a significant cause of mortality in the recent years. One of the major concerns in the prevalence of such infections is the development of multidrug-resistant microorganisms.

Aim: The present surveillance study was conducted to assess the prevalence, microbiological spectrum, and antimicrobial susceptibility aminoof the microorganisms at a tertiary care hospital.

Methods: The study was conducted between May 2014 and December 2016 including 466 patients (255 men and 211 women) diagnosed with healthcare-associated infections. Data related to sex, age, time of admission, type of surgery, and complication were collected. Samples of the pus, urine, and blood were collected and sent for isolation and characterization of microorganisms. Antimicrobial sensitivity was conducted by following the Clinical Laboratory Standards Institute (CLSI) guidelines. Data obtained were recorded in Microsoft Excel and analyzed using R 3.3.1; $P < 0.05$ indicative of significant differences.

Result: Mean age of the patients was 41.01 ± 18.11 years. Prevalence results were highest in surgical site infection (79.18%) and gram-negative organisms (80.92%) were the prevalent causes of infection. Escherichia coli (21.54%), Pseudomonas aeruginosa (15.76%), and Acinetobacter sp. (12.22%) were the predominant organisms with maximum sensitivity towards carbapenems, aminoglycosides, and cotrimoxazole, respectively. E. coli was the prevalent producer of AmpC and extended-spectrum beta-lactamases.

Conclusion: Reduced sensitivity to the majority of antimicrobials has led to the prevalence of gram-negative organisms in the healthcare-associated infections. However, carbapenems and chloramphenicol along with aminoglycosides remain the effective broad-spectrum antimicrobials. Further studies are required strategies to minimize antibiotic resistance, which can lead to improvement in therapeutic methods.

Keywords: Escherichia coli, Carbapenems, Surgical site infection, Lactamases

Introduction

Healthcare-associated infections (HAIs) are one of the most frequent causes of morbidity and mortality, globally.¹ Poor infrastructure and limited re-

sources are the prominent factors responsible for the difference in prevalence of HAIs in developed and developing countries. The impact of HAIs leads to prolonged hospital stay and increased financial load. According to the 2014 HAI report of World Health Organization (WHO), prevalence of HAIs is more in middle and low income countries (5.7–19.1%), as compared to the high income countries (3.5–12%). In addition, the incidence of HAIs are most commonly observed in the intensive care units (ICU), with approximately 51% cases of HAIs in the high income countries and 4.4%–88.8% cases of HAIs in the middle and low income countries. Between 2004 and 2012, the rate of HAIs ranged from 4.36% to 83.09% in the Indian subcontinent.²

*Corresponding Author

Dr. Savitha N

Assistant Professor, Department of Microbiology,
Sri Devaraj Urs Medical College, Sri Devaraj Urs
Academy of Higher Education and Research,
Kolar-563101, Karnataka, India.

Mobile No : 9886998046

E-mail : drsavitha2003@gmail.com

Conflict of Interest: None

Financial Aid: Nil

Inappropriate and inefficient use of invasive and life-support therapies in the healthcare facilities contribute to the development of HAIs.³ Catheter-associated urinary tract infections (CAUTI) are the commonly encountered HAIs in the high income countries whereas surgical site infections (SSI) are prevalent in middle and low income countries.² In the Indian subcontinent, SSI accounts for 1.6%–21% of HAIs reported between 2003 and 2012.⁴ However, the incidence of CAUTI in India was observed to be comparatively higher with approximately 49%–51% cases between 1998 and 2015.⁵ Ventilator associated pneumonia (VAP) is another prominent HAI with an incidence rate of 52.7/1000 days in the developing countries and 40% in India.^{6,7}

Prolonged indiscriminate use of antibiotics plays a major role in the development of antibiotic-resistant strains.² Along with the drug-resistant strains of *Staphylococcus aureus* and *Staphylococcus epidermidis*, pathogens such as *Escherichia coli*, *Pseudomonas aeruginosa*, and *Proteus sp.* are commonly detected in HAIs.⁴ Therefore, antimicrobial resistance and surveillance of drug usage in healthcare facilities may help in preparing a baseline data on the pattern of microbial susceptibility and infection control.⁸ Apart from providing quality healthcare to the admitted patients, an effective surveillance may also help in ensuring better health of the hospital personnel and selection of an appropriate drug for treating the HAI cases.⁹

Studies on prevalence and microbiological spectrum of HAIs in India is limited. The present survey was undertaken to evaluate the prevalence and risk factors of HAI in a tertiary care teaching hospital, and analyze the spectrum of microorganisms associated with HAI and antibiotic sensitivity pattern of the respective isolates.

Methodology

Study design

This retrospective study was conducted at the Department of Microbiology in collaboration with other departments of a tertiary care teaching hospital, Kolar. Approval was obtained from the Institutional Ethics Committee.

Patient eligibility and assessment

A total of 466 patients diagnosed with HAIs including 255 men and 211 women of different age-groups were enrolled for the study. The study was conducted between May 2014 and December 2016. The patients were screened from the departments of Otolaryngology, medicine, neurosurgery, obstetrics and gynecology, orthopedics, pediatrics, plastic surgery, surgery and urology and classified with SSIs, CAUTIs

and VAP infection based on the USA Centers for Disease Control and Prevention (CDC, Table 1). Data on age, sex, date of admission, preoperative and postoperative stay, and the reason for admission and type of surgery—emergency or elective were recorded in the case record form.

Samples of pus, urine, and endotracheal aspirate for HAIs were collected from the patients and transported to the laboratory for isolation and identification of the microorganisms.

Microbiological analysis

Prevalence of the isolates was recorded on the basis of the type of HAI and Gram stain. In vitro antimicrobial sensitivity of the isolated microorganisms was performed according to the Clinical Laboratory Standards Institute (CLSI) guidelines.⁸ The antibiotics used in the survey include beta lactam antibiotics (penicillin, ampicillin, imipenem, meropenem, ertapenem and piperacillin), beta lactam drugs in combination with betalactamase inhibitors (amoxicillin-clavulanic acid, piperacillin-tazobactam, and ampicillin-sulbactam), inhibitors of protein synthesis (erythromycin, clindamycin, amikacin, gentamicin, tobramycin, chloramphenicol, tetracycline, vancomycin, linezolid, and doxycycline), fluoroquinolones (ciprofloxacin, levofloxacin, norfloxacin and ofloxacin), cephalosporins (ceftazidime, ceftriaxone, cefoxitin and cefotaxime), cotrimoxazole and nitrofurantoin. Production of lactamases (AmpC beta lactamases and extended-spectrum beta-lactamases or ESBL) by all the isolates was also analyzed using CLSI guidelines.¹⁰

Statistical analysis

Recorded data were coded using Microsoft Excel. R 3.3.1 software was used to perform the statistical analysis. Continuous data were expressed as mean \pm SD and compared using one-way analysis of variance (ANOVA). Categorical data were analyzed using chi-square test. $P \leq 0.05$ was considered as statistically significant.

Results

Patient characteristics

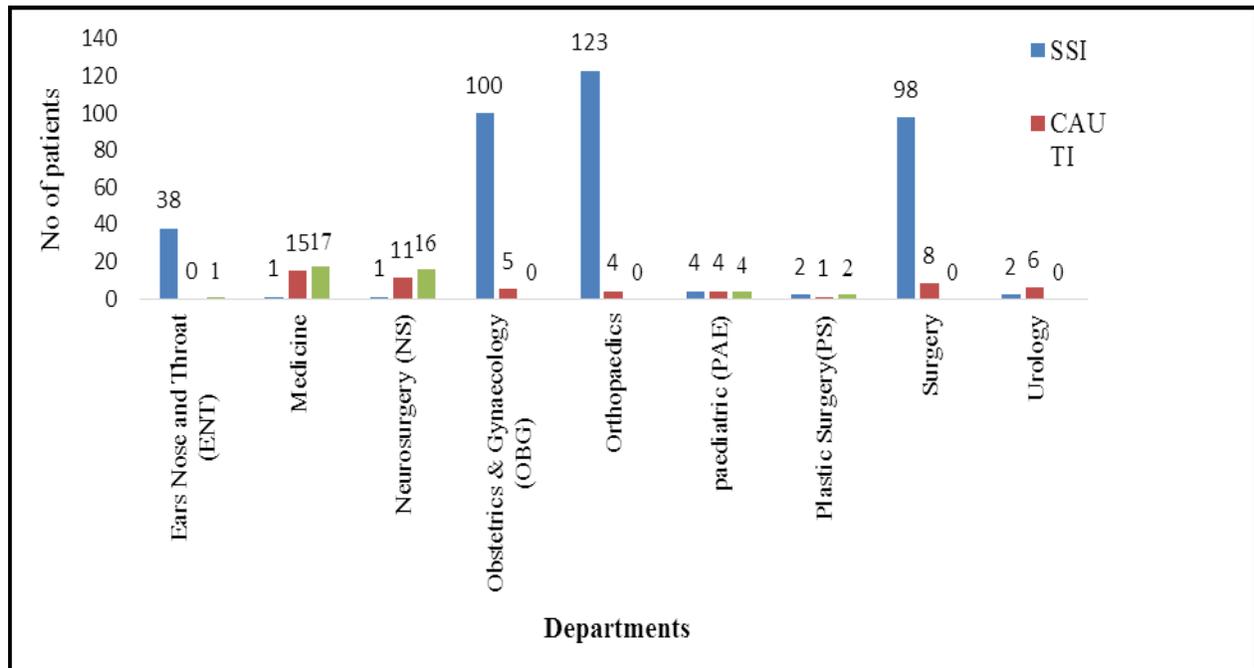
The mean age of the patients was 41.01 ± 18.11 years. Sex-wise distribution of the patients revealed a male predominance (54.5%) in the study. Age-wise distribution of the patients was significant ($P = 0.01$), in which the maximum number of patients were in the age-group of 20-39 years. Among the SSI patients,

mean duration of hospital stay was 25 days with the majority of the patients (58.60%) undergoing elective type of surgery. The demographic and clinical characteristics of the patients are shown in **Table 1**.

Table 1: Patient characteristics

Variable	n (%)	SSI	CAUTI	VAP	Mean ± SD	p-value
Sex						
Male	255 (54.5)	196 (52.7%)	32 (59.3%)	27 (67.5%)	-	0.16
Female	211 (45.3)	176 (47.3%)	22 (40.7%)	13 (32.5%)	-	
Age (Mean ± SD in years)						
Male	-	41.91± 17.27	47.97±22.14	40.58±17.32	41.01±18.11	0.06
Female	-	40±17.11	40.92±23.66	34.23±22.08		
Age group (years)						
<20	28 (6.14)	16 (4.40%)	6 (11.32%)	6 (15.38%)	-	0.01*
20-39	208 (45.61)	175 (48.10%)	17 (32.10%)	16 (41.02%)	-	
40-59	114 (25)	94 (25.82%)	11 (20.75%)	9 (23.08%)	-	
60-79	97 (21.27)	74 (20.33%)	16 (30.19%)	7 (17.95%)	-	
≥80	9 (1.97)	5 (1.37%)	3 (5.66%)	1 (2.56%)	-	

Figure 1. Department-wise characterization of HAIs

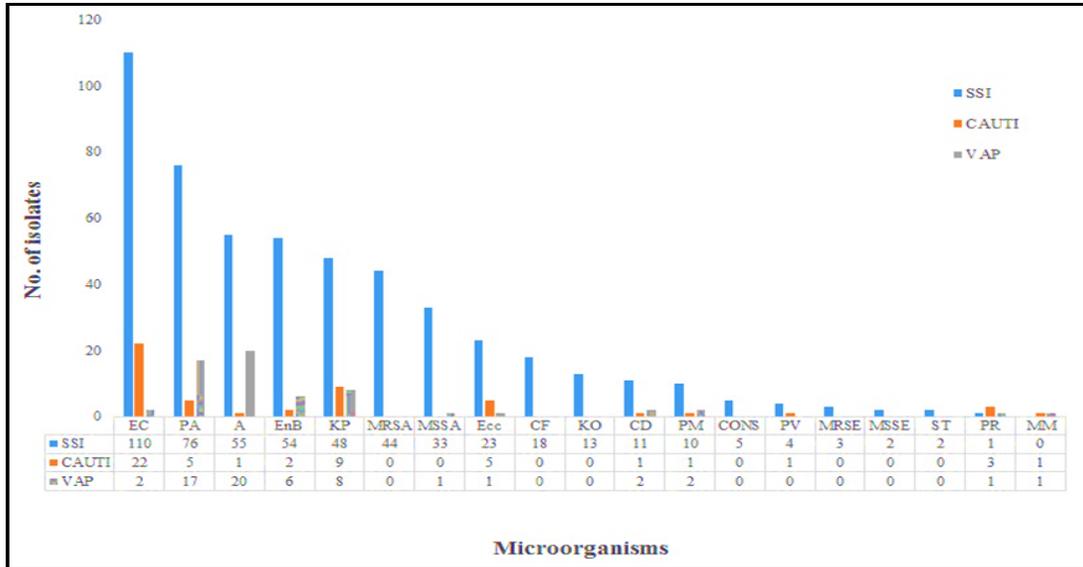


Identification and prevalence of isolates

Prevalence of SSIs (79.18%) was highest among HAIs. The prevalence of SSIs was maximum in the departments of ENT, obstetrics & gynecology, orthopedics and surgery (**Figure 1**). Out of 466 clinical samples, 624 isolates were identified upon their growth in the culture media and Gram staining. Out of these isolates, 19.07% were gram-positive cocci (GPC), 53.04% were gram-negative bacteria (GNB), and 27.88% were non-

fermenting gram-negative bacteria (NFGNB). Culture reports of the isolates revealed the predominance of GNB in all the cases of HAIs. The most common microorganisms causing HAIs were *Escherichia coli* (21.54%), *Pseudomonas aeruginosa* (15.76%), and *Acinetobacter* sp. (12.22%). In addition, *E. coli* was predominant in SSI (21.48%) and CAUTI (47.83%), whereas *Acinetobacter* sp. was predominant in VAP (33.33%) infection. The distribution of the isolates with respect to infection is shown in **Figure 2**.

Figure 2. Characterization of isolates with respect to HAIs



Antimicrobial sensitivity test

Antimicrobial sensitivity pattern of the isolates is shown in **Table 2a** and **b**. *E. coli* was the most predominant isolate with high rates of sensitivity to imipenem (88.81%), amikacin (85.07%), meropenem (85.07%), chloramphenicol (70.15%), gentamicin (65.67%), ertapenem (65.67%), tobramycin (63.43%), piperacillin-tazobactam (51.49%), amoxicillin-clavulanic acid (40.30%), tetracycline (36.57%), cotrimoxazole (33.58%), and levofloxacin (30.60%). Least sensitivity was noted for ciprofloxacin (17.16%), nitrofurantoin (13.43%), ceftazidime (5.22%), piperacillin (3.73%), ampicillin (2.99%), doxycycline (2.99%), cefotaxime (2.24%) and ceftriaxone (2.24%). *P. aeruginosa* demonstrated high rate of sensitivity to meropenem (89.80%), imipenem (85.71%), piperacillin-tazobactam (75.51%), ceftazidime (69.39%), amikacin (67.35%), gentamicin (63.27%), piperacillin (62.24%), levofloxacin (60.20%), tobramycin (57.14%) and ciprofloxacin (57.14%). Reduced sensitivity of *P. aeruginosa* was observed to tetracycline (23.47%), cotrimoxazole (23.47%), doxycycline (22.45%), and ampicillin-sulbactam (21.43%). *Acinetobacter* sp., another predominant organism in HAIs in the present study,

demonstrated sensitivity to levofloxacin (63.16%), meropenem (53.95%), imipenem (52.63%), and tetracycline (34.21%). On the other hand, reduced sensitivity was observed to ampicillin-sulbactam (25%), amikacin (23.68%), gentamicin (18.42%), tobramycin (18.42%), cotrimoxazole (14.47%), piperacillin-tazobactam (10.53%), doxycycline (9.21%), ciprofloxacin (5.26%), ceftazidime (1.32%), and piperacillin (1.32%).

In general, the GNB were highly sensitive to amikacin (75.53%), imipenem (87.01%), ertapenem (68.28%), and meropenem (85.80%) and least sensitive to ampicillin (3.02%), doxycycline (3.02%), piperacillin (4.23%), ceftazidime (4.23%), nitrofurantoin (6.65%), cefotaxime (1.81%), and ceftriaxone (1.51%). GPC were highly sensitive to Vancomycin (69.75%), Linezolid (77.31%), chloramphenicol (66.39%), tetracycline (67.23%), and doxycycline (68.07%) and least sensitive to nitrofurantoin was observed to be high in the case of carbapenems—imipenem (71.26%), meropenem (74.14%), and levofloxacin (61.49%); however, In the case of NFGNB, sensitivity was observed to be high in the case of carbapenems—imipenem (71.26%), meropenem (74.14%), and levofloxacin (61.49%).

Table 2.a. Antimicrobial sensitivity pattern of isolates

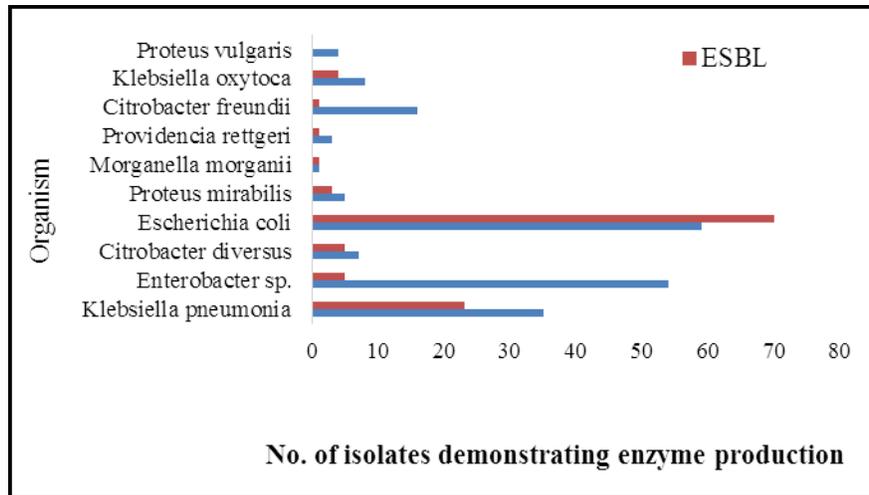
Nature of isolates	Organism	P	AMP	AMC	E	CD	VA	LZ	AK	GEN	TOB	IMP	MRP	ERP	C
GNB	<i>Escherichia coli</i>	-	4 (2.99)	54 (40.29)	-	-	-	-	114 (85.07)	88 (65.67)	85 (63.43)	119 (88.81)	114 (85.07)	88 (65.67)	94 (70.15)
	<i>Klebsiella pneumoniae</i>	-	1 (1.54)	19 (29.23)	-	-	-	-	43 (66.15)	33 (50.77)	33 (50.77)	55 (84.62)	55 (84.62)	46 (70.77)	37 (56.92)
	Enterobacter sp.	-	1 (1.61)	5 (8.06)	-	-	-	-	42 (67.74)	28 (45.16)	28 (45.16)	54 (87.1)	54 (87.1)	43 (69.35)	41 (66.13)
	<i>Citrobacter freundii</i>	-	0 (0)	1 (5.56)	-	-	-	-	13 (72.22)	11 (61.11)	11 (61.11)	11 (61.11)	14 (77.78)	5 (27.78)	11 (61.11)
	<i>Klebsiella oxytoca</i>	-	0 (0)	4 (30.77)	-	-	-	-	9 (69.23)	9 (69.23)	8 (61.54)	13 (100)	11 (84.62)	13 (100)	8 (61.54)
	<i>Citrobacter diversus</i>	-	0 (0)	3 (21.43)	-	-	-	-	12 (85.71)	9 (64.29)	8 (57.14)	11 (78.57)	11 (78.57)	10 (71.43)	11 (78.57)
	<i>Proteus mirabilis</i>	-	3 (23.08)	7 (53.85)	-	-	-	-	12 (92.31)	11 (84.62)	11 (84.62)	13 (100)	13 (100)	10 (76.92)	6 (46.15)
	<i>Proteus vulgaris</i>	-	0 (0)	1 (20)	-	-	-	-	4 (80)	3 (60)	2 (40)	5 (100)	5 (100)	5 (100)	1 (20)
	<i>Providencia rettgeri</i>	-	0 (0)	2 (40)	-	-	-	-	0 (0)	0 (0)	0 (0)	5 (100)	5 (100)	5 (100)	1 (20)
	<i>Morganella morganii</i>	-	1 (50)	1 (50)	-	-	-	-	1 (50)	1 (50)	1 (50)	2 (100)	2 (100)	1 (50)	0 (0)
GPC	Methicillin-resistant <i>Staphylococcus aureus</i>	0 (0)	0 (0)	0 (0)	13 (29.55)	25 (56.82)	31 (70.45)	38 (86.36)	0 (0)	20 (45.45)	-	-	-	-	39 (88.64)
	Methicillin-sensitive <i>Staphylococcus aureus</i>	5 (14.71)	1 (2.94)	31 (91.18)	20 (58.82)	23 (67.65)	20 (58.82)	23 (67.65)	0 (0)	30 (88.24)	-	-	-	-	29 (85.29)
	<i>Enterococci</i> sp.	12 (41.38)	16 (55.17)	0 (0)	0 (0)	0 (0)	26 (89.66)	25 (86.21)	0 (0)	6 (20.69)	-	-	-	-	2 (6.9)
	Coagulase negative <i>Staphylococci</i>	3 (60)	4 (80)	4 (80)	3 (60)	3 (60)	3 (60)	3 (60)	0 (0)	5 (100)	-	-	-	-	4 (80)
	Methicillin-resistant <i>Staphylococcus epidermidis</i>	0 (0)	0 (0)	0 (0)	2 (66.67)	2 (66.67)	1 (33.33)	1 (33.33)	0 (0)	2 (66.67)	-	-	-	-	2 (66.67)
	Methicillin-sensitive <i>Staphylococcus epidermidis</i>	0 (0)	0 (0)	2 (100)	2 (100)	2 (100)	1 (50)	1 (50)	0 (0)	2 (100)	-	-	-	-	2 (100)
<i>Streptococci</i> sp.	1 (50)	0 (0)	0 (0)	0 (0)	0 (0)	1 (50)	1 (50)	0 (0)	2 (100)	-	-	-	-	1 (50)	
NFG NB	<i>Pseudomonas aeruginosa</i>	-	-	-	-	-	-	-	66 (67.35)	62 (63.27)	56 (57.14)	84 (85.71)	88 (89.8)	-	-
	<i>Acinetobacter</i> sp.	-	-	-	-	-	-	-	18 (23.68)	14 (18.42)	14 (18.42)	40 (52.63)	41 (53.95)	-	-

Table 2.b. Antimicrobial sensitivity pattern of isolates

Nature of isolates	Organism	COT	TE	DO	CIP	LEV	PIP	PI	CAZ	AS	NIT	CX	CTX	CRO	NO R	OF L	
GNB	<i>Escherichia coli</i>	45 (33.58)	49 (36.57)	4 (2.99)	23 (17.16)	41 (30.6)	5 (3.73)	69 (51.49)	7 (5.22)	-	18 (13.43)	-	3 (2.24)	2 (1.49)	-	-	
	<i>Klebsiella pneumoniae</i>	11 (16.92)	29 (44.62)	1 (1.54)	30 (46.15)	38 (58.46)	1 (1.54)	27 (41.54)	1 (1.54)	-	2 (3.08)	-	0 (0)	0 (0)	-	-	
	Enterobacter sp.	15 (24.19)	41 (66.13)	1 (1.61)	30 (48.39)	35 (56.45)	2 (3.23)	7 (11.29)	1 (1.61)	-	0 (0)	-	0 (0)	0 (0)	-	-	
	<i>Citrobacter freundii</i>	1 (5.56)	13 (72.22)	1 (5.56)	12 (66.67)	8 (44.44)	0 (0)	1 (5.56)	0 (0)	-	0 (0)	-	0 (0)	0 (0)	-	-	
	<i>Klebsiella oxytoca</i>	5 (38.46)	4 (30.77)	0 (0)	3 (23.08)	5 (38.46)	0 (0)	3 (23.08)	0 (0)	-	0 (0)	-	0 (0)	0 (0)	-	-	
	<i>Citrobacter diversus</i>	2 (14.29)	5 (35.71)	0 (0)	3 (21.43)	6 (42.86)	0 (0)	7 (50)	1 (7.14)	-	1 (7.14)	-	0 (0)	0 (0)	-	-	
	<i>Proteus mirabilis</i>	4 (30.77)	4 (30.77)	3 (23.08)	10 (76.92)	13 (100)	5 (38.46)	9 (69.23)	4 (30.77)	-	0 (0)	-	3 (23.08)	3 (23.07)	-	-	
	<i>Proteus vulgaris</i>	0 (0)	0 (0)	0 (0)	2 (40)	3 (60)	0 (0)	2 (40)	0 (0)	-	1 (20)	-	0 (0)	0 (0)	-	-	
	<i>Providencia rettgeri</i>	0 (0)	2 (40)	0 (0)	1 (20)	3 (60)	1 (20)	1 (20)	0 (0)	-	0 (0)	-	0 (0)	0 (0)	-	-	
	<i>Morganella morganii</i>	0 (0)	1 (50)	0 (0)	0 (0)	1 (50)	0 (0)	0 (0)	0 (0)	-	0 (0)	-	0 (0)	0 (0)	-	-	
GPC	Methicillin-resistant <i>Staphylococcus aureus</i>	22 (50)	39 (88.64)	41 (93.18)	6 (13.64)	-	-	-	-	-	0 (0)	0 (0)	0 (0)	-	-	-	
	Methicillin-sensitive <i>Staphylococcus aureus</i>	19 (55.88)	30 (88.24)	30 (88.24)	17 (50)	-	-	-	-	-	0 (0)	33 (97.06)	0 (0)	-	-	-	
	<i>Enterococci</i> sp.	1 (3.45)	1 (3.45)	1 (3.45)	1 (3.45)	-	-	-	-	-	-	2 (6.9)	1 (3.45)	0 (0)	-	-	-
	Coagulase negative <i>Staphylococci</i>	4 (80)	5 (100)	5 (100)	3 (60)	-	-	-	-	-	-	0 (0)	3 (60)	0 (0)	-	-	-
	Methicillin-resistant <i>Staphylococcus epidermidis</i>	0 (0)	2 (66.67)	2 (66.67)	1 (33.33)	-	-	-	-	-	-	0 (0)	3 (100)	0 (0)	-	-	-
	Methicillin-sensitive <i>Staphylococcus epidermidis</i>	0 (0)	2 (100)	2 (100)	1 (50)	-	-	-	-	-	-	0 (0)	0 (0)	0 (0)	-	-	-
	<i>Streptococci</i> sp.	0 (0)	1 (50)	0 (0)	0 (0)	-	-	-	-	-	-	0 (0)	0 (0)	0 (0)	-	-	-
NFGNB	<i>Pseudomonas aeruginosa</i>	23 (23.47)	23 (23.47)	22 (22.45)	56 (57.14)	59 (60.2)	61 (62.24)	74 (75.51)	68 (69.39)	21 (21.43)	-	-	-	-	-	-	
	<i>Acinetobacter</i> sp.	11 (14.47)	26 (34.21)	7 (9.21)	4 (5.26)	48 (63.16)	1 (1.32)	8 (10.53)	1 (1.32)	19 (25)	-	-	-	-	-	-	

Production of AmpC lactamases and ESBL were also detected in the isolates with *E. coli* being the highest producer of AmpC and ESBL lactamases (Figure 3).

Figure 3: Production of ESBL and AmpC lactamases by isolates



Discussion

The retrospective study was performed at a tertiary care hospital to analyze the prevalence and risk factors of HAIs. The antimicrobial sensitivity and lactamase production assay was also performed to prepare a baseline data of microbial susceptibility.

Bacterial infections have been commonly encountered in the hospitals due to the ability of the causative agent to survive extreme conditions such as the dry surfaces of hospitals. The spread of these organisms through patients (infected or carrier) and hospital personnel play a major role in the increased incidence of HAIs.¹¹ The majority of the isolates, identified in the present study, were GNB (53.04%), with *E. coli* being the most frequently encountered strain. The predominance of *E. coli* in HAIs had also been reported in earlier studies.^{3, 12} However, in the recent years, NFGNB have also emerged as the prominent pathogens in HAIs. These microorganisms are usually encountered on the skin surfaces of hospital staffs, as well as in ventilators and hospital linens.⁸ In support of this statement, the present study reported the predominance of NFGNB (27.88%), especially *P. aeruginosa* and *Acinetobacter sp.* in SSI, CAUTI, and VAP. In contrast to the incidence of GNB and NFGNB, the prevalence of GPC was minimal (19.07%) and drug-resistant *S. aureus* (12.5%) was the predominant microorganism. Previous studies have observed the predominance of GNB in HAIs, as compared to the GPC.^{13, 14}

In the present study, the GNB belonged to Enterobacteriaceae family and were highly sensitive to carbapenems (ertapenem, meropenem, and

imipenem). Carbapenems, being broad spectrum beta lactam antibiotics, have been reported to be highly active against the Enterobacteriaceae family of microorganisms, including those possessing the ability to produce AmpC beta lactamases¹⁵ and hence were highly active against the GNB isolates in the present study. NFGNB were also sensitive to carbapenems in the present study. Carbapenems have been used against infections caused by *Acinetobacter sp.* and *P. aeruginosa*; however, recent studies have demonstrated the development of carbapenems-resistant strains of NFGNB due to its extensive use in the hospitals.^{16, 17}

According to the observations of the present study, aminoglycosides, such as amikacin are highly effective against several GNB, as well as GPC infections, especially the multidrug resistant forms. These antimicrobials inhibit protein synthesis by binding to 16S rRNA leading to disruption of the integrity of bacterial cell membrane.¹⁸⁻²⁰ Similar rates of sensitivity against GNB, as well as the majority of GPC has been exhibited by chloramphenicol in the present study. Chloramphenicol acts by reversible binding to the 50S ribosome leading to inhibition of protein synthesis. It is especially effective against multidrug-resistant pathogens,^[21] which can be a possible cause of its high rate of sensitivity in the present study. Tetracycline, doxycycline, and gentamicin are the inhibitors of protein synthesis, which have shown similar activities in the present study. These antimicrobials have demonstrated maximum activity towards *Staphylococcal sp.* in the present surveillance study. Tetracycline acts by inhibiting the binding of tRNA to mRNA²², doxycycline binds to 30S subunit of the bacterial ribosome, and

aminoglycoside and gentamicin bind to 30S subunit of the ribosome and 16S rRNA for limiting the synthesis of proteins.^[23] However, extensive use of these antimicrobials has led to the reduction of sensitivity and increase in the development of multidrug-resistant strains.²²

Fluoroquinolones, especially ciprofloxacin and levofloxacin, have demonstrated effective activity against GNB and *P. aeruginosa* in the present surveillance study. Fluoroquinolones interact with DNA gyrase (especially in GNB) and topoisomerase IV (especially in GPC) leading to inhibition of DNA synthesis.²⁴ Positive interaction of ciprofloxacin and levofloxacin with DNA gyrase may possibly be the reason of the effectiveness demonstrated in the present study. The majority of the isolates in the current surveillance study demonstrated sensitivity toward cotrimoxazole, which is a combination of trimethoprim and sulfamethoxazole. The effectiveness of the antimicrobial drug can be attributed to its ability of selectively inhibiting microbial reductases and synthesis of tetrahydrofolate leading to restricted cellular growth and survival.²⁵

The isolates in the present study have demonstrated low levels of sensitivity toward beta lactam antibiotics. This may be due to the ability of AmpC and ESBL lactamases production, as observed in the GNB isolates of the present study. The reduced sensitivity can also be due to the utilization of cell wall transpeptidases in the synthesis of the cell wall, which are beta lactam resistant. Apart from the production of lactamases, GNB can also resist the adverse actions of beta lactam antibiotics by actively expelling the antimicrobials from within the cells with the help of efflux pumps.²⁶ The sensitivity to cephalosporins also reduced due to the similar reasons, which could be the possible reason for its reduced activity towards the isolates of the present study.²⁷

These findings support the increasing and undesirable trend in the development of multidrug-resistant strains, indicating decreased efficacy of beta lactam antibiotics, cephalosporins, fluoroquinolones, and inhibitors of protein synthesis. Production of lactamases has become the major cause of reduced sensitivity along with repetitive exposure of the microorganisms to the same antimicrobials over the years. Such findings have led to extensive research over the factors contributing to the development of resistance, the alternative modes of treatment, and performance of similar surveillance study in the same hospital, with a gap of few years.

Conclusion

The current research finding indicated that the isolated microorganisms were susceptible to carbapenems, aminoglycosides, and chloramphenicol.

The majority of the microorganisms demonstrated minimal sensitivity to cephalosporins and fluoroquinolone. In addition, the GNB were found to produce AmpC and ESBL, which might have played a role in their resistance to several antimicrobials along with other inherent factors of the microorganisms. Thus, this surveillance study could facilitate the preparation of baseline data for providing an effective and improved healthcare facility to patients. In addition, studies involving factors responsible for virulence and resistance of the microorganism to drugs could be performed to monitor the drug administration and improve the therapeutic methods for treatment of HAIs.

References

1. Sodhi J, Satpathy S, Sharma D, Lodha R, Kapil A, Wadhwa N, et al. Healthcare associated infections in Paediatric Intensive Care Unit of a tertiary care hospital in India: Hospital stay & extra costs. *The Indian journal of medical research.* 2016;143(4):502.
2. Organization WH. Health care-associated infections fact sheet. Im Internet: www.who.int/gpsc/country_work/gpsc_ccisc_fact_sheet_en.pdf. 2014.
3. Khan MS, Kundra P, Cherian A, Joseph NM, Sistla S. Epidemiology of nosocomial infections in an intensive care unit at a tertiary care hospital in India: A retrospective study. *International Journal of Infection Control.* 2015;11(2).
4. Ramasubramanian V, Iyer V, Sewlikar S, Desai A. Epidemiology of healthcare acquired infection—An Indian perspective on surgical site infection and catheter related blood stream infection. *Indian Journal of Basic and Applied Medical Research.* 2014;3:46-63.
5. Vyawahare CR, Gandham NR, Misra RN, Jadhav SV, Gupta NS, Angadi KM. Occurrence of catheter-associated urinary tract infection in critical care units. *Medical Journal of Dr DY Patil University.* 2015;8(5):585.
6. Gadani H, Vyas A, Kar AK. A study of ventilator-associated pneumonia: Incidence, outcome, risk factors and measures to be taken for prevention. *Indian journal of anaesthesia.* 2010;54(6):535.
7. Ranjan N, Chaudhary U, Chaudhry D, Ranjan K. Ventilator-associated pneumonia in a tertiary care intensive care unit: Analysis of incidence, risk factors and mortality. *Indian Journal of Critical Care Medicine.* 2014;18(4):200.
8. Moolchandani K, Sastry AS, Deepashree R, Sistla S, Harish B, Mandal J. Antimicrobial Resistance Surveillance among Intensive Care Units of a Tertiary Care Hospital in Southern India. *Journal of Clinical and Diagnostic Research: JCDR.* 2017;11(2):DC01.

9. Hughes R. Patient safety and quality: An evidence-based handbook for nurses Cites eer; 2008.
10. Peter-Getzlaff S, Polsfuss S, Poledica M, Hombach M, Giger J, Böttger E, et al. Detection of AmpC beta-lactamase in *Escherichia coli*: comparison of three phenotypic confirmation assays and genetic analysis. *J Clin Microbiol* 2011 49(8):2924-32. DOI: 10.1128/JCM.00091-11
11. Saka K, Akanbi I, AA OT, Raheem R, Oshodi A. Pathogenic Aerobic Bacterial Contaminants on Non-Critical Hospital Surfaces within Paediatric Ward of a Nigerian Hospital. *J Med Microb Diagn*. 2016;5(241):2161-0703.1000241.
12. Ghanshani R, Gupta R, Gupta BS, Kalra S, Khedar RS, Sood S. Epidemiological study of prevalence, determinants, and outcomes of infections in medical ICU at a tertiary care hospital in India. *Lung India: official organ of Indian Chest Society*. 2015;32(5):441.
13. Manyahi J, Matee MI, Majigo M, Moyo S, Mshana SE, Lyamuya EF. Predominance of multi-drug resistant bacterial pathogens causing surgical site infections in Muhimbili National Hospital, Tanzania. *BMC research notes*. 2014;7(1):500.
14. Shah S, Singhal T, Naik R. A 4-year prospective study to determine the incidence and microbial etiology of surgical site infections at a private tertiary care hospital in Mumbai, India. *American journal of infection control*. 2015;43(1):59-62.
15. Rao MB, Murthy L, Jesmi D, Prasad M. Resistance of *Escherichia coli* and *Salmonella* isolated from marine and freshwater fishes towards Carbapenems. *Fishery Technology* 2014; 51 : 207 - 212 .
16. Guzek A, Korzeniewski K, Nitsch-Osuch A, Rybicki Z, Prokop E. In vitro sensitivity of *Acinetobacter baumannii* and *Pseudomonas aeruginosa* to carbapenems among intensive care unit patients. *Neurobiology of Respiration: Springer*; 2013. p. 109-16.
17. Liu Q, Li X, Li W, Du X, He J-Q, Tao C, et al. Influence of carbapenem resistance on mortality of patients with *Pseudomonas aeruginosa* infection: a meta-analysis. *Scientific Reports* 2015 25;5:11715. doi: 10.1038/srep11715.
18. Gad GF, Mohamed HA, Ashour HM. Aminoglycoside resistance rates, phenotypes, and mechanisms of Gram-negative bacteria from infected patients in upper Egypt. *PLoS One*. 2011;6(2):e17224.
19. Shyamala R, Rama Rao M, Janardhan Rao M. The Sensitivity pattern of *Escherichia coli* to Amikacin in a tertiary care hospital. *Der Pharmacia Lettre*. 2012;4(3):1010-2.
20. Verma P. Antibiotic sensitivity treatment for gram positive bacteria isolated from pus sample. *Bull Environ Pharmacol Life Sci* 2012;1(10):03-6.
21. Sood S. Chloramphenicol—A Potent Armament Against Multi-Drug Resistant (MDR) Gram Negative Bacilli? *Journal of clinical and diagnostic research* 2016;10(2):DC01.
22. Chopra I, Roberts M. Tetracycline antibiotics: mode of action, applications, molecular biology, and epidemiology of bacterial resistance. *Microbiology and molecular biology reviews* 2001;65(2):232-60.
23. Kotra LP, Haddad J, Mobashery S. Aminoglycosides: perspectives on mechanisms of action and resistance and strategies to counter resistance. *Antimicrobial agents and chemotherapy* 2000;44(12):3249-56.
24. Hooper DC. Mode of action of fluoroquinolones. *Drugs* 1999;58(2):6-10.
25. Hitchings GH. Mechanism of action of trimethoprim-sulfamethoxazole—I. *Journal of Infectious Diseases*. 1973;128(Supplement 3):S433-S6.
26. Wilke MS, Lovering AL, Strynadka NC. β -Lactam antibiotic resistance: a current structural perspective. *Current opinion in microbiology* 2005;8(5):525-33.
27. Livermore DM. Mechanisms of resistance to cephalosporin antibiotics. *Drugs* 1987;34(2):64-88.