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# DETERMINATION OF DRUG SUSCEPTIBILITY PATTERN AGAINST NOSOCOMIAL INFECTION

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## ABSTRACT

Nosocomial infections even in this modern era antibiotics, continue to remain an important and formidable consequence of hospitalization. Our aim of the study was to investigate the antimicrobial susceptibility against hospital-associated infecting microorganisms. 30 Clinical samples were taken from OPD of GMC Hospital, Bhopal (MP), India. Among 30 clinical isolates we identified bacterial pathogens as *Staphylococcus aureus* (10), *Escherichia coli* (10) and *Pseudomonas aeruginosa* (8). Antimicrobial susceptibility assay was performed by the reference criteria of clinical and laboratory standard institute guidelines. In the present study antibiotic susceptibility results showed that all 10 (100%) *S. aureus* isolates were resistant to penicillin, 8 (80%) *S. aureus* isolates were resistant to cefuroxime, 7 (70%) *S .aureus* isolates were resistant to ciprofloxacin, 5 (50%) *S. aureus* isolates were resistant to amikacin, 2 (20%) *S. aureus* isolates were resistant to nitrofurantoin whereas, 0 (0%) *S. aureus* isolates were found to be resistant to cefazolin. For *E. coli* isolates all 10 (100%) were resistant to cefuroxime, 7 (70%) *E. coli* isolates were resistant to amikacin, 5 (50%) *E. coli* isolates were resistant to penicillin, 2 (20%) *E. coli* isolates were resistant to cefazolin as well as nitrofurantoin whereas, 0 (0%) *E. coli* isolates found to be resistant to ciprofloxacin. For *P. aeruginosa* isolates all 8 (100%) were resistant to nitrofurantoin, 6 (75%) *P. aeruginosa* isolates were resistant to cefuroxime, 5 (62.5%) *P. aeruginosa* isolates were resistant to amikacin, 4 (50%) *P. aeruginosa* isolates were resistant to penicillin, 3 (37.5%) *P. aeruginosa* isolates were resistant to ciprofloxacin whereas, 2 (25%) *P. aeruginosa* isolates were resistant to cefazolin. Hence we concluded that the efficacy of cefazolin and ciprofloxacin was higher than other antibiotics tested against Nosocomial infection causing pathogens.

**INTRODUCTION:** Nosocomial infections in this modern era continue to remain an important and formidable consequence of hospitalization. It may be defined as any infection that is being acquired in a hospital, particularly when the source or the risk factor for it is one peculiar to the hospital [1]. Nosocomial infections are also divided into two classes, endemic or epidemic [2, 3].

It has been estimated that about 4-5% of patients leave the hospital after having acquired infections, depending on the case, hospital size and multiple other factors [4, 5].

Nosocomial infections are caused by a relatively few opportunistic organisms [6]. Infections by *Staphylococcus aureus, Streptococci* (GBS) Enterobacteriaceae and

*Pseudomonas aeruginosa* could either be acquired from other persons or by self infections whereas most infections by *Streptococci* (GAS) are from other persons. The common pathogenic bacteria such as *Escherichia coli, Klebsilla pneumoniae* [7], *Haemophilus influenza, S. pneumoniae, S. aureus, P. aeruginosa* and *Proteus vulgaris* have been reported worldwide. Unfortunately, multidrug-resistant organisms, including *P. aeruginosa, Acinetobacter baumannii* and extended-spectrum β-lactamase (ESBL) producing Enterobacteriaceae, are increasingly being reported worldwide.

The urinary tract, gastrointestinal tract, skin and bloodstream were the most common sites of infection and *S. aureus, Salmonella* species and hepatitis B virus were the most common pathogens [8]. *S. aureus* remains the dominant species in surgical wound infection, followed by the enterobacteria. Bacteroides species along with other gut bacteria, very often in mixed growth is found typically in wounds after a colonized viscous has been entered. Although *S. aureus* may occur in all types of wound, it is the typical cause of the less frequent wound infection in clean surgery. *P. aeruginosa* is the epitome of an opportunistic pathogen of humans [9].

The bacterium almost never infects uncompromised tissues, yet there is hardly any tissue that it cannot infect if the tissue defenses are compromised in some manner. It causes urinary tract infections, respiratory system infections, dermatitis, soft tissue infections, bacteremia, bone and joint infections, gastrointestinal infections and a variety of systemic infections, particularly in patients with severe burns and in cancer and AIDS patients who are immunosuppressed [10].

*P. aeruginosa* is frequently resistant to many commonly used antibiotics. Although many strains are susceptible to gentamicin, tobramycin, colistin and fluoroquinolins, resistant forms have developed. The combination of gentamicin and carbenicillin is frequently used to treat severe *Pseudomonas* infections [11]. Moreover, *Pseudomonas* maintains antibiotic resistance plasmids, both R-factors and RTFs and it is able to transfer these genes by means of the bacterial mechanisms of horizontal gene transfer (HGT), mainly transduction and conjugation.

*E. coli* is one of the most frequent causes of many common bacterial infections, including cholecystitis, bacteremia, cholangitis, urinary tract infection [12-14] and traveler's diarrhea and other clinical infections such as neonatal meningitis and pneumonia.

To overcome the arising effects of nosocomial infection researches have been performing worldwide. The problem of microbial resistance is growing and the outlook for the use of antimicrobial drugs in the future is still uncertain. Therefore, actions must be taken to reduce this problem, for example, to control the use of antibiotic, develop research to better understand the genetic mechanisms of resistance and to continue studies to develop new drugs, either synthetic or natural. The ultimate goal is to offer appropriate and efficient antimicrobial drugs to the patient.

The aim of the study was to investigate the antimicrobial susceptibility against hospital-associated infection causing microorganisms.

## MATERIALS AND METHODS:

**Bacterial Isolates and Sampling Procedure:** Thirty Clinical samples were taken from OPD of GMC Hospital, Bhopal (MP), India. Samples were taken from patient's bed, stretcher and labor room using swab with wooden stick and taken to the laboratory in cold condition within 4 hr for microbiological analysis.

**Bacterial Isolation and Identification:** The samples were inoculated on nutrient agar and MacConkey agar media plate and incubated at 37°C for 24 hr. The colonies of isolated organism have been sub culture on nutrient agar plate and pure culture were obtained and identified by biochemical tests. Out of 30 clinical samples, 28 bacterial strains were isolated out of which 10 strains were of *S. aureus*, 10 strains were of *E. coli* and 8 strains were of *P. aeruginosa*.

**Antibiotic Susceptibility Assay:** Antibiotic sensitivity testing of the isolates was carried out by Kirby Bauer's assay[15]. Briefly, the culture medium used for the antibiotic susceptibility was Mueller Hinton Agar (MHA). Inoculation in the nutrient broth for 24 hr for antibiotic sensitivity assay was performed by the reference criteria of clinical and laboratory standard institute guidelines [16].

Broth culture of test organisms was homogeneously was spread on the surface with the help of glass spreader on Mueller-Hinton agar (MHA) plate and then the antibiotic discs were prepared using different antibiotics (Amikacin, Cefazolin, Cefuroxime, Ciprofloxacin, Nitrofurantoin and Penicillin) and placed immediately with a sterile forceps. Sensitivity test plate was then incubated by keeping them inverted in incubator at 37°C for 24 hrs results were interpreted as per the standard literature provided with the commercial disc.

**RESULTS:** Among 30 clinical samples, 28 bacterial strains were isolated out of which 10 strains were of *S. aureus*, 10 strains were of *E. coli* and 8 strains were of *P. aeruginosa*. The isolated organisms were characterized on the basis of morphological, cultural and biochemical characteristics.

Antibiotic susceptibility results in **figure 1** showed that all 10 (100%) *S .aureus* isolates were resistant to penicillin, 8 (80%) *S. aureus* isolates were resistant to cefuroxime, 7 (70%) *S. aureus* isolates were resistant to ciprofloxacin, 5 (50%) *S. aureus* isolates were resistant to amikacin, 2 (20%) *S. aureus* isolates were resistant to nitrofurantoin whereas, 0 (0%) *S. aureus* isolates were found to be resistant to cefazolin that are shown in following **Table 1**.

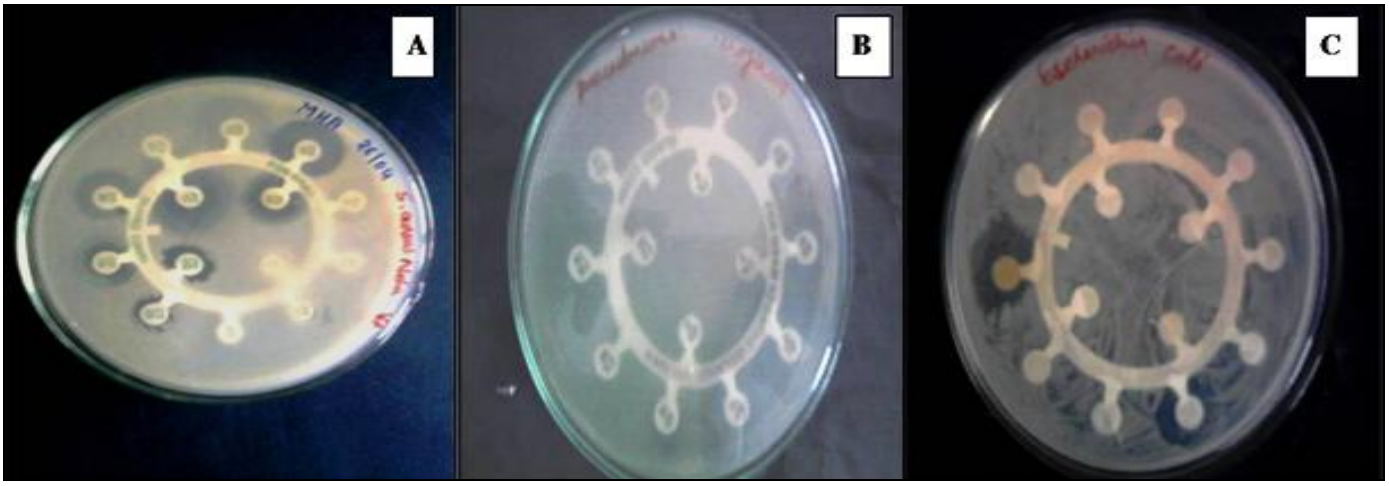


**FIG. 1: SHOWING ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF GIVEN BACTERIAL PATHOGENS FROM VARIOUS SITE OF HOSPITAL ASSOCIATED INFECTIONS (A) *S. AUREUS*; (B) *P. AERUGINOSA*; (C) *E. COLI***

**TABLE 1: ANTIBIOTIC SUSCEPTIBILITY PATTERN AGAINST *S. AUREUS***

| Antibiotics (Concentration in mcg) | No. (%) of resistant strain (n= 10) |
|---|---|
| **Amikacin (30)** | 5 (50%) |
| **Cefazolin (30)** | 0 (0%) |
| **Cefuroxime (30)** | 8 (80%) |
| **Ciprofloxacin (5)** | 7 (70%) |
| **Nitrofurantoin** | 2 (20%) |
| **Penicillin (10)** | 10 (100%) |

For *E. coli* isolates all 10 (100%) were resistant to cefuroxime, 7 (70%) *E. coli* isolates were resistant to amikacin, 5 (50%) *E. coli* isolates were resistant to penicillin, 2 (20%) *E. coli* isolates were resistant to cefazolin as well as nitrofurantoin whereas, 0 (0%) *E. coli* isolates found to be resistant to ciprofloxacin that are shown in following **Table 2**.

**TABLE 2: ANTIBIOTIC SUSCEPTIBILITY PATTERN AGAINST *E. COLI***

| Antibiotics (Concentration in mcg) | No. (%) of resistant strain (n= 10) |
|---|---|
| **Amikacin (30)** | 7 (70%) |
| **Cefazolin (30)** | 2 (20%) |
| **Cefuroxime (30)** | 10 (100%) |
| **Ciprofloxacin (5)** | 0 (0%) |
| **Nitrofurantoin** | 2 (20%) |
| **Penicillin (10)** | 5 (50%) |

For *P. aeruginosa* isolates all 8 (100%) were resistant to nitrofurantoin, 6 (75%) *P. aeruginosa* isolates were resistant to cefuroxime, 5 (62.5%) *P. aeruginosa* isolates were resistant to amikacin, 4 (50%) *P. aeruginosa* isolates were resistant to penicillin, 3 (37.5%) *P. aeruginosa* isolates were resistant to ciprofloxacin whereas, 2 (25%) *P. aeruginosa* isolates were resistant to cefazolin that are shown in following **Table 3**.

**TABLE 3: ANTIBIOTIC SUSCEPTIBILITY PATTERN AGAINST *P. AERUGINOSA***

| Antibiotics (Concentration in mcg) | No. (%) of resistant strain (n= 8) |
|---|---|
| **Amikacin (30)** | 5 (62.5%) |
| **Cefazolin (30)** | 2 (25%) |
| **Cefuroxime (30)** | 6 (75%) |
| **Ciprofloxacin (5)** | 3 (37.5%) |
| **Nitrofurantoin** | 8 (100%) |
| **Penicillin (10)** | 4 (50%) |

**DISCUSSION:** The present study is intended to evaluate the influence of severity of illness and the drug activity on the development of nosocomial infections. It was found that gram positive and gram negative bacterial pathogens are a common cause of infection and the prevalence and rates of resistance to existing antimicrobial agents are increasing.

Our study showed a total number of thirty isolates as gram positive and gram negative. All the strains of bacteria were subjected to biochemical tests. Among these ten were identified as *S. aureus*, ten as *E. coli* and eight as *P. aeruginosa*, were aimed to study their drug susceptibility pattern.

The cephalosporin group of antibiotic cefazolin showed maximum sensitivity against *S. aureus*. This group of antibiotic interfere with the bacterial cell wall synthesis by blocking the cross linking of the peptide glycan units. Whereas, *S. aureus* were found highly resistant to penicillin and as well as cefuroxime. *S. aureus* resistance to penicillin is mediated by penicillase production, an enzyme that cleaves the β- lactam ring of the penicillin molecule, rendering the antibiotic ineffective.

The antibiotic ciprofloxacin showed maximum sensitivity against *E. coli*[17,18]. This binds to the A subunit of DNA gyrase (topoisomerase) and prevent supercoiling of DNA, thereby inhibiting DNA synthesis. Nitrofurantoin also showed higher sensitivity against *E. coli*. The antibiotic nitrofurantoin works by damaging bacterial DNA, since its reduced form is highly reactive which is made possible by the rapid reduction of nitrofurantoin inside the bacterial cell by flavoproteins (nitrofuranreductase) to multiply reactive intermediates that attack ribosomal proteins, DNA, respiration, pyruvate metabolism and other macromolecules within the cell. Whereas, *E. coli* were found highly resistant to cefuroxime and as well as amikacin.

The antibiotic cefazolin and ciprofloxacin showed maximum sensitivity against *P. aeruginosa.* The antibiotic ciprofloxacin bind to the A subunit of DNA gyrase (topoisomerase) and prevent supercoiling of DNA, thereby inhibiting DNA synthesis. Whereas, *P. aeruginosa* were found highly resistant to nirtofurantoin and as well as cefuroxime. Therefore use of these antibiotics should be restricted to severe nosocomial infections, in order to avoid rapid emergence of resistant strains.

In order to reduce the development of drug resistance, combination therapies should be used that is achieved by giving separate drugs or by giving combination drugs which are dosage forms that contain more than one active ingredient.

**CONCLUSION:** We can conclude that knowledge of emerging pathogens and resistance profile is essential for treatment against nosocomial infections. Shorter duration of treatment and correct dosage of antibiotic therapy is recommended to reduce the selection pressure for resistant isolates.

Effective infection control programs are essential to controlling and preventing nosocomial infections. These programs include a core of the infection control committee, infection control practitioner and individual employee actions.

In the future, issues of concern about the emergence of nosocomial infections, increasing antimicrobial resistance, and the increase in morbidity, mortality and costs associated with these infections will drive the need for refinement of molecular approaches to aid in the diagnosis and epidemiologic analysis of nosocomial infections.

The evaluation of hospital-associate infections will continue to rely on clinical infection surveillance as the first step to understanding disease epidemiology and management of infections. The most accurate assessment of epidemiologic relationships in a nosocomial setting is always accomplished by careful assessment of all available information. Molecular testing will continue to be an essential tool, for the testing has proven to be cost-effective and medically needed. Molecular typing is a powerful tool in the armamentarium for combating the spread of problem microorganisms in the hospital environment.

Thus, it is important that we seek to continually improve existing infection control policies and programs.

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