# ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF ORGANISMS CAUSING SURGICAL SITE INFECTIONS (SSI)

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**ABSTRACT**

**BACKGROUND**

CDC defines surgical site infection as 'Infections related to operative procedure that occurs at or near surgical incision within 30 days of operative procedure or within one year if the implant is left in situ'. Surgical site infection (SSI) is 3rd most frequently reported nosocomial infection (12%-16%) as per National Nosocomial Infection Surveillance (NNIS).

The aim of this study was to investigate the antimicrobial susceptibility pattern of organisms causing SSI.

**MATERIALS AND METHODS**

During a two year study period in a tertiary care hospital, 19,127 patients underwent surgeries in various surgical departments. Of these 517 (2.7%) developed surgical site infection. The surgical wounds were classified by CDC & NNIS criteria into 4 classes. Two wound swabs were taken and processed by standard microbiological techniques. Antimicrobial susceptibility along with testing of ESBLs, MBLs, AmpCβ lactamases was done for all isolates causing SSI.

**RESULTS**

Among 19,127 patients, 517 (2.7%) developed SSI. It was highest in patients of perforation peritonitis (11.99%).Among 517 specimens, 340 (65.76%) showed growth and 177 (34.23%) were culture negative. E.coli (23.33%) was the commonest organism isolated followed by Acinetobacter spp. (16%), Klebsiella spp. (15.66%), Pseudomonas spp. (15.33%), S. aureus (10.33%), S. epidermidis(7.3%), Proteus spp. (6.00%) and Citrobacter spp. (2.66%).Staphylococcus spp. were 100 % sensitive to Vancomycin & Linezolid. (27.5%) S. aureus were MRSA and (17.5%) were Inducible Clindamycin resistant (ICR). Enterobacteriaceae isolates showed maximum sensitivity towards Imipenem, Piperacillin-Tazobactam and Amikacin. Klebsiella spp. (40.62%), E.coli (35.89%), Citrobacter spp. (33.33%), Proteus spp. (26.08%) were ESBL producers. Klebsiella spp. (17.18%), E.coli (10.25%), Proteus spp. (11.11%) and Citrobacter spp. (8.69%) were AmpC producers. Acinetobacter spp. (28.57%) was commonest MBL producer followed by Klebsiella spp. (20.31%), Pseudomonas spp. (18.84%), E.coli (15.38%), Proteus spp. (4.43%) & Citrobacter spp. (11.11%).

**CONCLUSION**

In sight of the high incidence of MRSA, ESBL and MDR reported in this study, there is a need for continuous monitoring to determine the susceptibility pattern of the common isolates causing SSI and to emphasize precise empirical therapy. Policies on prescription patterns should be reviewed, which will ensure reduced patient morbidity & mortality related to SSIs.

**KEYWORDS**

SSI, CDC, NNIS, Antimicrobial Susceptibility Testing.

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## BACKGROUND

Globally, surgical site infection rates have been reported to range from 2.5% to 41.9%.[1,2] According to National Nosocomial Infections Surveillance (NNIS) system, surgical site infections (SSIs) is the third most frequently reported nosocomial infection, accounting for 12% to 16% of all nosocomial infections among hospitalized patients.[3]

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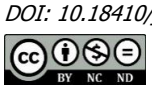

The Centre for Disease Control and Prevention (CDC) has developed criteria for defining SSIs, which have become the national standard and are widely used by surveillance and surgical personnel. These criteria defines SSIs as "infections related to the operative procedure that occur at or near the surgical incision within 30 days of an operative procedure or within 1 year if an implant is left in place".[4]

Antibiotics have potential impact on preventing mortality and morbidity in developing countries.[5] Appropriate surgical antibiotic prophylaxis (SAP) can reduce the postoperative wound infection while inappropriate use increases the selective pressure and favours the development of antimicrobial resistance. Around 30-50% of antibiotic use in hospitals is for Surgical Antibiotic Prophylaxis (SAP) and 30-90% of this prophylaxis is

inappropriate. The antibiotic is either given at the wrong time or continued for a long period.[6]

Henceforth, the study was undertaken to study the problem of postoperative surgical site infections in reference to emergence of antimicrobial-resistant pathogens in tertiary care hospital.

## MATERIALS AND METHODS

The study was carried out for a two year period in the Department of Microbiology in a tertiary care hospital.

During the study period 19,127 patients were operated in different surgical departments. Of these, 517 patients developed surgical site infections. The surgical wound was inspected at the time of first dressing and swabs were collected.

All samples were processed aerobically and identified by using standard microbiological techniques.[7] All isolates were subjected to antimicrobial susceptibility testing by the Kirby-Bauer disc diffusion method using Clinical and Laboratory Standards Institute (CLSI) guidelines.[8,9,10]

The isolates were further tested for MRSA, ICR (Inducible Clindamycin resistance), HLSR, ESBL, AmpC β lactamases and MBL, using standard control strains of Staphylococcus aureus ATCC 25923; E.coli ATCC 25922; Klebsiella pneumonia ATCC 700603 and Pseudomonas aeruginosa ATCC 27853;

### A. Detection of MRSA, MSA and ICR

All the isolates of Staphylococci species were subjected for Methicillin Resistance (MRSA and MRS) by Cefoxitin disc diffusion method using a 30 µg disc. A 0.5 McFarland standard suspension of the isolate was prepared and lawn culture was made on Muller Hinton Agar (MHA) plate. The plates were incubated at 37° C for 18 h and zone diameters were measured. An inhibition zone diameter of ≤19 mm was reported as resistant and ≥20 mm was considered as sensitive.[11]

The Staphylococcus species were also detected for Inducible Clindamycin Resistance (ICR). Detection of phenotypic expression of inducible ERM genes was carried out by double disk diffusion test (D-test) using Erythromycin and Clindamycin discs.[12] When these drugs are placed in close proximity to each other, Erythromycin (inducing agent) diffuses into the media and induces the ERM gene expression. This effect extends up to the sensitivity zone on one side of the Clindamycin disc leading to a D-shaped zone of inhibition.

Isolates showing circular zones of inhibition with diameter of <13 mm for Erythromycin and >21 mm for Clindamycin were interpreted as negative for inducible resistance (D-test negative). While isolates with same inhibitory diameters as above but a D-shaped zone around the Clindamycin, were interpreted as positive for inducible resistance (D-test positive).

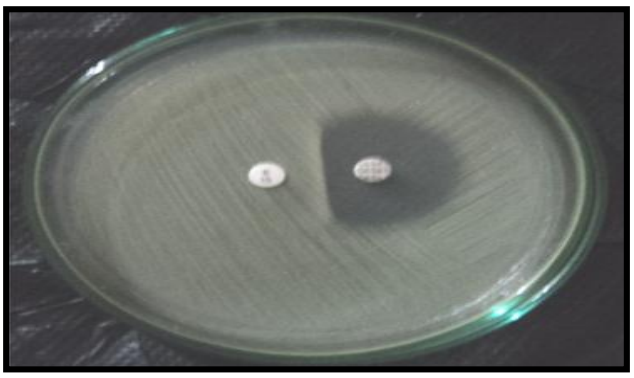

***Figure 1. Inducible Clindamycin Resistance (D-test using Erythromycin and Clindamycin Discs)***

### B. Detection of ESBL Production among Enterobacteriaceae Group of Organisms

Detection of ESBL production among Enterobacteriaceae group of organisms was tested by initial screening test followed by Phenotypic Confirmatory test and Double disc synergy test (DDST)/Disc approximation method.[12,13]

### Initial Screen Test

The isolates which were found resistant to Ceftazidime, Aztreonam or Cefotaxime were subjected for Phenotypic Confirmatory test (double disc diffusion test) for detection of ESBL production.

### Phenotypic Confirmatory Test

In this test a disk of third generation Cephalosporin (Ceftazidime or Cefotaxime) and its combination with clavulanic acid was kept on inoculated Mueller-Hinton Agar (MHA) plate followed by incubation overnight at 37°c. Interpretation of test was read as an equal or more than 5-mm increase in a zone diameter for either antimicrobial agent in combination with clavulanic acid vs its zone when tested alone was labelled as ESBL producer.

### Double Disc Synergy Test (DDST)/Disc Approximation Method.[12,13]

In DDST, synergy was determined between a disc of β-lactamase inhibitor (Amoxyclav) and Ceftazidime or Cefotaxime (30 µg disc). The antibiotic agent is placed at a distance of 30 mm apart on lawn culture of the resistant isolates on Muller-Hinton Agar. The test organism was considered to produce ESBL, if the zone size around the antibiotic disc increased towards the β- lactamase inhibitor disc. This increase occurs because the clavulanic acid inactivates the ESBL produced by the test organism resulting in the formation of extended inhibitory zone.


***Figure 2. Detection of ESBL Production (Phenotypic Confirmatory Test)***

**C. Detection of AmpC β-Lactamase Production (Screening test) in Gram Negative Isolates.**[12]

For testing AmpC β-lactamases production, a lawn culture of test strain was exposed to a disc of cefotaxime (30 µg) and cefoxitin (30 µg) placed at a distance of 15 mm from edge to edge. After overnight incubation, there was flattening of radius of zone of inhibition produced by cefotaxime on the side nearest the cefoxitin disc in AmpC β-lactamases producing strains.

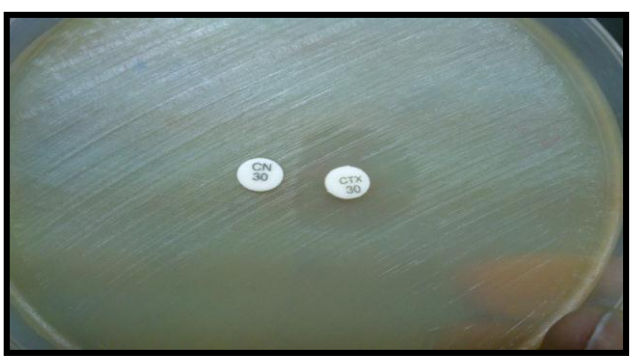


***Figure 3. AmpC Production (Screening Test using Cefotaxime and Cefoxitin Disc)***

**D. Detection of MBL (Metallo-β-lactamase) Producing Isolates** [14,15]

By Double Disc Synergy Test- The strains which were found resistant to Imipenem were further confirmed for production of Metallo-β-lactamase by Double disc synergy test. For the EDTA-disc synergy test an overnight broth culture of the test strain, (turbidity adjusted to 0.5 McFarland turbidity standards) was used to inoculate a plate of Mueller-Hinton agar. The EDTA-Imipenem disc and the Imipenem disc were placed on the test culture, 30 mm distance apart. After overnight incubation, the presence of an enlarged zone of inhibition around the EDTA-Imipenem of ≥5 mm than Imipenem disc, it was interpreted as positive test.

In case of Pseudomonas aeruginosa and Acinetobacter baumannii, if the zone of inhibition around EDTA-Imipenem was ≥7 mm than the zone of inhibition around Imipenem disc, it was labelled as MBL producer.

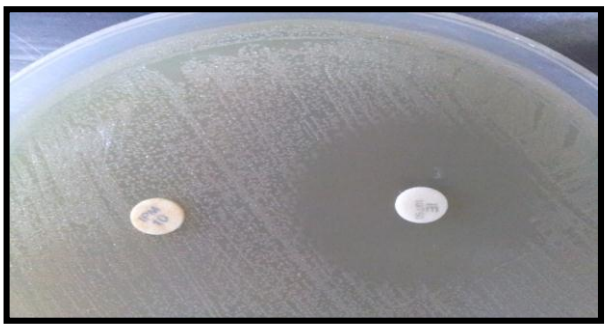

***Figure 4. MBL Production (Double Disc Synergy Test using Imipenem-Imipenem-EDTA Disc)***

**RESULTS**

In our study, total 19,127 cases underwent surgery and 517 (2.7%) patients developed Surgical Site Infections (SSIs). Out of 517 specimens processed 340 were culture positive and 177 were culture negative. 40 specimens had mixed growth. E.coli (23.33%) was the most frequently isolated organism among the pure growth specimens causing SSI.

| | **Isolated Organisms** | **Total Number (%)** |
|---|---|---|
| 1 | Escherichia coli | 70 (23.33) |
| 2 | Acinetobacter baumannii | 48 (16.00) |
| 3 | Klebsiella pneumonia | 47 (15.66) |
| 4 | Pseudomonas aeruginosa | 46 (15.33) |
| 5 | Staphylococcus aureus | 31 (10.33) |
| 6 | Staphylococcus epidermidis | 22 (7.33) |
| 7 | Proteus mirabilis | 18 (6.00) |
| 8 | Citrobacter freundii | 8 (2.66) |
| 9 | Enterococcus faecalis | 6 (2.00) |
| 10 | Enterobacter aerogenes | 2 (0.66) |
| 11 | Morganella morganii | 1 (0.33) |
| 12 | Serratia marcescens | 1 (0.33) |
| | **Total (Pure Growth)** | **300** |

***Table 1. Distribution of Organisms causing SSIs***

Staphylococcus spp. showed 100% sensitivity for Vancomycin & Linezolid. S. aureus (27.5%) was MRSA & (17.5%) was ICR. S. epidermidis (13.63%) was methicillin resistant & (13.63%) were resistant to Clindamycin.

S. aureus (45%) was MRSA and 7 (17.5%) were inducible Clindamycin resistant (ICR). Out of 22 isolates of Staphylococcus epidermidis, 5 (22.72%) were methicillin resistant and 3 (13.63%) were resistant to Clindamycin.

| **Group** | **Antimicrobial Agents** | **S.aureus (n=40)** | **S. epidermidis (n=22)** |
|---|---|---|---|
| A | Erythromycin | 18 (45) | 12 (54.54) |
| | Clindamycin | 30 (75) | 19 (86.36) |
| | Cefoxitin | 22 (55) | 17 (77.27) |
| | Penicillin G | 3 (7.5) | 7 (31.81) |
| B | Linezolid | 40 (100) | 22 (100) |
| | Tetracycline | 16 (40) | 13 (59.09) |
| | Vancomycin | 40 (100) | 22 (100) |
| | Rifampicin | 31 (77.5) | 18 (81.81) |
| C | Chloramphenicol | 27 (67.5) | 16 (72.72) |
| | Ciprofloxacin | 16 (40) | 12 (54.54) |
| | Gentamicin | 19 (47.5) | 14 (63.63) |

***Table 2. Antimicrobial Susceptibility of Staphylococcus Species***

| Group | Antimicrobial Agents | Sensitive (%) |
|---|---|---|
| A | Ampicillin | 3 (37.5) |
| | Penicillin G | 0 |
| B | Linezolid | 8 (100) |
| | Vancomycin | 8 (100) |
| HLAR | Gentamicin | 5 (62.5) |
| | Streptomycin | 5 (62.5) |

***Table 3. Antimicrobial Susceptibility of Enterococcus Species***

All Enterococcus spp. were sensitive to Vancomycin and Linezolid and (62.5 %) were resistant to Gentamicin and Streptomycin (HLAR).

| Group | Antimicrobial Agents | E. coli (n=78) | Klebsiella pneumoniae (n=64) | Proteus mirabilis (n= 23) | Citrobacter freundii (n=9) | Enterobacter aerogenes (n=2) | Morganella morganii (n=1) | Serratia marcescens (n=1) |
|---|---|---|---|---|---|---|---|---|
| A | Ampicillin | 18 (23.07) | 15 (23.43) | 3 (13.04) | 3 (33.33) | 2 (100) | 0 | 0 |
| | Gentamicin | 49 (62.82) | 36 (56.25) | 18 (78.26) | 7 (77.77) | 2 (100) | 1 (100) | 1 (100) |
| | Tobramycin | 50 (64.10) | 34 (53.12) | 18 (78.26) | 7 (77.77) | 2 (100) | 1 (100) | 1 (100) |
| B | Amikacin | 61 (78.20) | 46 (71.87) | 15 (65.21) | 6 (66.66) | 2 (100) | 1 (100) | 1 (100) |
| | Amoxyclav | 53 (67.94) | 40 (62.5) | 18 (78.26) | 4 (44.44) | 2 (100) | 1 (100) | 1 (100) |
| | Piperacillin-Tazobactam | 62 (79.48) | 47 (73.43) | 20 (86.95) | 8 (88.88) | 2 (100) | 1 (100) | 1 (100) |
| | Cefuroxime | 28 (35.89) | 14 (21.87) | 13 (56.52) | 1 (11.11) | 1 (50) | 0 | 0 |
| | Cefepime | 42 (53.84) | 37 (57.81) | 19 (82.60) | 4 (44.44) | 1 (50) | 0 | 1 (100) |
| | Cefoxitin | 30 (38.46) | 32 (50.00) | 13 (56.52) | 2 (22.22) | 1 (50) | 1 (100) | 1 (100) |
| | Cefotaxime | 40 (51.28) | 33 (51.56) | 14 (6086) | 3 (33.33) | 1 (50) | 0 | 1 (100) |
| | Ciprofloxacin | 38 (48.71) | 28 (43.75) | 18 (78.26) | 4 (44.44) | 1 (50) | 1 (100) | 1 (100) |
| | Imipenem | 66 (84.61) | 51 (79.68) | 20 (86.95) | 8 (88.88) | 2 (100) | 1 (100) | 1 (100) |
| | Piperacillin | 21 (26.92) | 12 (18.75) | 8 (34.78) | 5 (55.55) | 1 (50) | 0 | 0 |
| C | Ceftazidime | 41 (52.56) | 33 (51.56) | 14 (6086) | 4 (44.44) | 1 (50) | 1 (100) | 1 (100) |
| | Chloramphenicol | 61 (78.20) | 46 (71.87) | 15 (65.21) | 6 (66.66) | 2 (100) | 1 (100) | 1 (100) |
| | Tetracycline | 38 (48.71) | 28 (43.75) | 18 (78.26) | 4 (44.44) | 1 (50) | 1 (100) | 1 (100) |

***Table 4. Antimicrobial Susceptibility among Enterobacteriaceae Group Causing SSI***

Enterobacteriaceae group had maximum sensitivity towards Imipenem, Piperacillin-Tazobactam &Amikacin, while most were found resistant to Cefotaxime, followed Ciprofloxacin, Ceftriaxone.

| Antimicrobial Agents | Pseudomonas spp. (Sensitive %) | Acinetobacter spp. (Sensitive %) |
|---|---|---|
| Ceftazidime | 19 (27.53) | 15 (23.80) |
| Gentamicin | 35 (50.72) | 30 (47.61) |
| Tobramycin | 33 (47.82) | 28 (44.44) |
| Piperacillin | 32 (46.37) | 15(23.80) |
| Amikacin | 43(62.31) | 39 (61.90) |
| Aztreonam | 19(27.53) | - |
| Cefotaxime | - | 22 (32.92) |
| Cefepime | 16 (23.18) | 22 (32.92) |
| Ciprofloxacin | 19 (27.53) | 21 (33.33) |
| Imipenem | 56 (81.15) | 45 (71.42) |
| Piperacillin-Tazobactam | 52 (75.36) | 40 (63.49) |
| Tetracycline | - | 11 (17.46) |

***Table 5. Antimicrobial Susceptibility of Pseudomonas Species and Acinetobacter Species Causing SSI***

Pseudomonas species were (81.5%) sensitive to Imipenem, (75.36%) to Piperacillin-Tazobactam and (50.72%) to Gentamicin.

(71.42%) of Acinetobacter species were sensitive to Imipenem followed by (63.49%) to Piperacillin-Tazobactam and (61.90%) to Amikacin.

Among the ESBL & AmpC producing isolates, K. pneumoniae was predominant. Acinetobacter spp (28.57%) was the commonest MBL producing organism.

| Isolates | ESBL Producers | AmpC Producers | MBL Producers |
|---|---|---|---|
| E.coli (n=78) | 28 (35.89%) | 8 (10.25%) | 12 (15.38) |
| Klebsiella pneumoniae (n=64) | 26 (40.62%) | 11 (17.18%) | 13 (20.31) |
| Citrobacter freundii (n=9) | 3 (33.33%) | 1 (11.11%) | 1 (11.11) |
| Proteus mirabilis (n=23) | 6 (26.08%) | 2 (8.69) | 1 (4.43) |
| Pseudomonas aeruginosa (n=69) | - | - | 13 (18.84) |
| Acinetobacter baumannii (n=63) | - | - | 18 (28.57) |
| **Total (n=310)** | **63** | **22** | **58 (18.70)** |

***Table 6. Extended Antimicrobial Susceptibility Pattern Among Isolates***

## DISCUSSION

During the study period 19,127 patients underwent various surgical procedures in different surgical departments. Out of these, 517 (2.7%) patients developed surgical site infections.

This rate of SSI was comparable to the study carried out by Shantanu et al (2011).[16] who reported an incidence of 5 %, while Heidi Misteli et al (2011).[17] reported 4.7%, Franal et al (2010).[18] reported 4.10 % and Rathi et al (2008) reported 7.3%.[19]

In the present study, AMP (antimicrobial prophylaxis) was received by (71.56%) cases and it was observed that preoperative antibiotic administration significantly reduces the rate of postoperative surgical site infection. Studies carried out by Amit k et al (2012).[20] Wassef et al (2012).[21] Shantanu et al (2011).[16] Mahesh et al (2010).[22] and Rathi et al (2008).[19] have also reported around similar findings.

In this study, 517 specimens were collected and 300 (58.02%) had pure growth while 40 specimens (7.73%) had mixed growth. 177 (34.23%) specimens showed no growth.

Out of 300 pure growth specimens, the predominant organism was E.coli (23.33%). This is comparable to a study by Brian M et al (2011).[23] who reported 25% of E.coli in their study.

Among 40 isolates of S. aureus, 100 % sensitivity was shown towards Vancomycin and Linezolid. This is comparable to study carried out by Gayathree et al (2011) who reported 100 % sensitivity to Vancomycin.[24]

In our study, all the 8 (100%) isolates of Enterococcus faecalis were sensitive to Vancomycin and Linezolid. This is comparable to study carried out by Mahesh et al (2010).[22]

In a study carried out by Sanjay et al (2010).[25] majority of the E. coli isolates were found sensitive to Imipenem, Amikacin, Chloramphenicol while most were found resistant to Cefotaxime, followed by Ciprofloxacin, Ceftriaxone.

In the present study, Klebsiella species showed highest sensitivity towards Imipenem and Piperacillin-Tazobactam while maximum resistance was shown towards Ceftazidime, Ciprofloxacin, Amoxicillin and Gentamicin.

In our study, Enterobacter aerogenes and Proteus mirabilis were sensitive to most of the commonly used antibiotics.

Among 69 isolated species of Pseudomonas sp, (81.15%) were sensitive to Imipenem and (75.36%) were sensitive to Piperacillin-Tazobactam. This was comparable to Waqar et al (2012).[26] who found that most of the gram negative organisms are resistant to commonly used antibiotic agents such as Gentamicin, third generation Cephalosporins and Quinolone, Ciprofloxacin.

Out of 63 isolates of Acinetobacter, (71.42%) were found sensitive to Imipenem and (63.49%) sensitive to Piperacillin-Tazobactam. Amit k et al and Waseff et al (2012).[20,21] noted (87.5%) of Acinetobacter baumannii as multi drug resistant (MDR).

The percentage of MRSA in our study was (45%) while Weigelt et al (2010).[27] reported (20.6%) of MRSA in his study. A study carried out by Azap O K et al (2005).[28] detected (5.7%) of MRSA.

In our study, (17.5%) of S. aureus were detected as Inducible Clindamycin resistant (ICR).

Among S. epidermidis, (22.72%) were detected as Methicillin resistant. while (13.63 %) of S. epidermidis had constitutive resistance to Clindamycin.

In our study, Klebsiella pneumoniae was predominant ESBL as well as AmpC producer. Rizvi et al (2009) and Hemlata et al (2007).[29] also reported similar findings.

Acinetobacter species was predominant MBL producer (28.57%), followed by Klebsiella pneumoniae (20.31%). This is comparable to study carried out by Sanjay et al (2010) and Rizvi (2009).[25,30]

## CONCLUSION

Although, a large number of antimicrobial agents have been developed more recently, yet development of resistance to large number of antimicrobials is quite alarming. Further, imprudent use of antimicrobial agents promotes growth of resistant micro-organisms and can cause serious toxicity. Initiation of optimal empirical antibiotic therapy requires knowledge of the most likely infecting micro-organisms and their susceptibilities to antimicrobial drugs. Thus for surgeons to decide the appropriate prophylactic and therapeutic antibiotics, there should be data on the spectrum of common pathogens encountered in the surgical unit and their antimicrobial susceptibility data at each hospital setting and thus, the finding in our study could have relevant clinical use in the antibiotic policy guidelines for hospitals setup.

In sight of the high incidence of MRSA, ESBL and MDR reported in this study, there is a need for continuous monitoring to determine the susceptibility pattern of the common isolates which are found in hospitals & to emphasize precise empirical therapy. Policies on prescription patterns should be reviewed, which will ensure reduced patient morbidity & mortality related to SSIs. Advances in infection control practices include improved operating room ventilation, sterilization methods, barriers, surgical technique, and availability of antimicrobial prophylaxis. Despite these activities, SSIs remain a substantial cause of morbidity and mortality among hospitalized patients. This may be partially explained by the emergence of antimicrobial-resistant pathogens and the increased numbers of surgical patients who are elderly and/or have a wide variety of chronic, debilitating, or immuno-compromising underlying diseases. Moreover, reinforcement of infection control measures is strongly recommended in order to prevent healthcare-associated infections.

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