

Role of Microbiology in Preventing Surgical Site Infections During Anesthesia

Dr Sapana Tharwani¹, Dr Anisha Nagaria², Dr Santosh Tharwani³

1. Dr Sapana Tharwani, Assistant Professor, Department of Microbiology, Sri Balaji Institute of Medical Sciences, Raipur, CG, drsapanatharwani@gmail.com
2. Dr Anisha Nagaria, Associate Professor, Department of Anaesthesia, Sri Balaji Institute of Medical Sciences, Raipur CG, dranishanagaria@gmail.com
3. Dr Santosh Tharwani, Associate Professor, Department of Anaesthesia, Sri Balaji Institute of Medical Sciences, Raipur, CG , drsantoshtharwani@gmail.com

Corresponding author:

Dr Santosh Tharwani, Associate Professor, Department of Anaesthesia, Sri Balaji Institute of Medical Sciences, Raipur, CG , drsantoshtharwani@gmail.com

ABSTRACT

Background: Surgical site infection (SSI) is one of the most common postoperative complications and contributes significantly to postoperative morbidity and mortality. **Patients and Methods:** A prospective study was conducted on 80 patients undergoing clean and clean-contaminated surgeries in the departments of orthopedics, general surgery, and obstetrics & gynecology. Relevant clinical details were recorded for each patient. Patients were followed from admission until discharge and for 30 days postoperatively, according to Centers for Disease Control and Prevention criteria. Identification of infecting organisms was performed using staining and culture techniques, and antibiotic susceptibility testing was carried out using the disc diffusion method. **Results:** Out of 80 patients, 28 (35%) developed postoperative infections. Staphylococcus aureus was the most commonly isolated organism, and none of the strains were methicillin-resistant. Significant drug resistance was observed, particularly among Enterobacteriaceae, where cefotaxime-

resistant strains of Escherichia coli and Klebsiella pneumoniae were identified as extended-spectrum beta-lactamase (ESBL) producers. The emergence of Acinetobacter species from surgical wounds was also noted as a growing concern. Various patient-related factors and hospital protocols were analyzed in relation to treatment outcomes. **Conclusion:** Surgical site infections remain a significant clinical challenge. The emergence of multidrug-resistant organisms highlights the importance of rational antibiotic use and adherence to evidence-based practices to curb the rise of resistant pathogens.

Keywords: Antibiotic susceptibility, nosocomial infection, surgical site infection

INTRODUCTION

Surgical site infections (SSI) are among the most significant postoperative complications, contributing substantially to patient morbidity, mortality, prolonged hospital stay, and increased healthcare costs. According to the Centers for Disease Control and Prevention, SSI is defined as an infection occurring at or near the

surgical incision within 30 days of a procedure (or up to one year in the presence of implants). These infections are classified into superficial incisional, deep incisional, and organ/space infections based on the depth and anatomical involvement.[1]

The incidence of SSI varies across different healthcare settings and populations. In developed countries such as the United States, SSI accounts for approximately 2–5% of all surgical procedures, making it one of the most common postoperative complications.[2] Despite advances in surgical techniques, sterilization procedures, and antimicrobial therapy, SSI continues to remain a major challenge in modern surgical practice. The pathogenesis of SSI is multifactorial and involves both endogenous and exogenous sources of infection. Endogenous infections arise from the patient's own microbial flora, particularly skin commensals, whereas exogenous infections may result from contaminated surgical instruments, operating room environment, or transmission via healthcare personnel. The development of SSI is influenced by several factors, including the microbial load, virulence of the pathogen, and host immune response.[3] Risk factors associated with SSI can be broadly categorized into intrinsic (patient-related) and extrinsic (procedure-related) factors. Intrinsic factors include age, nutritional status, obesity, comorbid conditions, and duration of preoperative hospitalization. Extrinsic factors include perioperative practices such as patient preparation, hair removal, skin antisepsis, antimicrobial prophylaxis, and duration of surgery. Inadequate adherence to these measures increases the likelihood of infection.[3]

Hospital-acquired infections represent a major category of nosocomial infections among surgical patients, with

postoperative wound infections being the most frequently encountered.[4] Although surgical care has advanced significantly over the past decades, wound infections remain a persistent clinical problem. These infections can delay healing, prolong recovery, and may lead to long-term disability or even death in severe cases. Strict aseptic surgical techniques can significantly reduce microbial contamination; however, infection may still occur if microorganisms successfully colonize and proliferate within the wound. In such situations, antimicrobial therapy plays a crucial role in both prevention and treatment. The increasing availability and irrational use of antibiotics have led to the emergence of resistant strains, complicating treatment strategies. Therefore, identification of the causative organism through microbiological culture and antibiotic susceptibility testing is essential for appropriate therapy.

Both local and systemic factors influence the development of postoperative wound infections. Local factors include hematoma, seroma formation, presence of foreign materials such as sutures, poor surgical technique, and degree of wound contamination. Systemic factors such as age, nutritional status, personal hygiene, and associated comorbidities also significantly affect patient outcomes. A wide range of aerobic and anaerobic bacteria may be involved, often leading to pyogenic infections characterized by pus formation. In recent years, the emergence of extended-spectrum beta-lactamase (ESBL)-producing organisms has become a serious concern. ESBLs are enzymes produced by Gram-negative bacteria, particularly members of Enterobacteriaceae and *Pseudomonas aeruginosa*, which confer resistance to penicillins, cephalosporins, and monobactams, thereby limiting therapeutic options.[5]

Wounds are commonly classified as clean (Class I) or clean-contaminated (Class II) based on the degree of microbial contamination, as per Centers for Disease Control and Prevention guidelines.[6]

In view of these considerations, the present study was undertaken with the following objectives:

- To determine the incidence of postoperative surgical site infections in clean and clean-contaminated surgeries in orthopedic, general surgery, and obstetrics & gynecology departments.
- To identify patient-related and procedure-related factors influencing the occurrence of SSI.
- To study the bacteriological profile and antimicrobial susceptibility patterns of the isolates.

MATERIALS AND METHODS

Study Design and Setting

The present prospective study was conducted in the Departments of Microbiology and Anesthesiology at Sri Balaji Institute of Medical Sciences. Institutional Ethics Committee approval was obtained prior to the commencement of the study.

Sample Size and Study Population

A total of 80 patients undergoing clean and clean-contaminated surgeries in the Departments of Orthopedics, General Surgery, and Obstetrics & Gynecology were included. Patient details were recorded using a structured proforma. All patients were followed from the time of admission until discharge and up to 30 days postoperatively, in accordance with Centers for Disease Control and Prevention.[7]

Inclusion Criteria

- Clean surgeries (Class I operative wounds)
- Clean-contaminated surgeries (Class II operative wounds) as per Centers for Disease Control and Prevention.[6]

Exclusion Criteria

- Contaminated (Class III) and dirty (Class IV) operative wounds

- Stitch abscess
 - Episiotomy and circumcision wounds
- ### **Definition of Surgical Site Infection**
- A wound was considered infected if any one of the following criteria was present:[8]
- Serous or non-purulent discharge from the wound
 - Purulent discharge (pus) from the wound
 - Serous discharge with signs of inflammation such as edema, redness, warmth, local rise in temperature, tenderness, or induration
- ### **Sample Collection and Transport**
- Wound swabs were collected aseptically from the depth of the wound, avoiding contamination from surrounding skin.[9]
 - Surgical wounds were inspected at the time of first dressing and subsequently at weekly intervals until discharge, following Centers for Disease Control and Prevention.[7]

Microbiological Processing

Smear Examination

A smear was prepared from the collected sample and stained using the Gram-staining technique for preliminary identification of microorganisms.

Culture and Identification

Samples were inoculated onto Blood agar and MacConkey agar using a sterile nichrome loop and incubated at 37°C for 18–24 hours. Identification of isolates was performed based on colony morphology, Gram staining characteristics, and standard biochemical reactions, in accordance with Clinical and Laboratory Standards Institute.[10]

Antibiotic Susceptibility Testing

Antimicrobial susceptibility testing was carried out using the Kirby–Bauer disk diffusion method on Mueller-Hinton agar, following Clinical and Laboratory Standards Institute.[10]

Detection of ESBL Production

Isolates of *Escherichia coli* and *Klebsiella pneumoniae* resistant to cefotaxime were further tested for extended-spectrum beta-lactamase (ESBL) production using the Double Disk Diffusion Test.[5] A clear extension of the inhibition zone of the cephalosporin disk towards the amoxicillin-clavulanic acid disk after incubation for 18 hours on Mueller-Hinton agar was interpreted as positive for ESBL production.[5]

RESULTS

Infection rates after various surgical procedures at surgical sites were observed. Table 1 shows the high infection rate in appendectomy, gastric, small and large bowel surgeries (66.6%). The infection rate in uterus and adnexal structures (33.3%), urinary tract and genitalia (40%) and lower segment caesarean structure (LSCS) (31%) are comparatively lower. The infection rate after breast surgery is 33.3% [Table 1]. Overall postoperative SSI rate was 35% [Table 1].

It was higher (50%) in age group above 60 years. There was no sex predilection. Totally, 28 (35%) wounds were classified as clean wounds. Of these, 5 cases developed wound infection with an infection rate of 17.65%. Rest of the 52 (65%) cases were classified as clean contaminated wounds and out of which 23 cases (37.1%) got infected postoperatively ($\chi^2 = 26.1$; $P = 0.00021$). Rate of infection was higher in emergency cases (46.3%) than elective cases (20%) ($\chi^2 = 75$; $P = 0.024$). There is an increase in infection rate with increase in preoperative hospital stay ($\chi^2 = 14.1$; $P = 0.029$). The highest infection rate is seen among the patients with preoperative stay of more than 21 days (65%) while it is 27.5% in patients with preoperative stay up to 7 days. Infection rate is 18.8% in patients who received preoperative antibiotics compared to 47.5% in patients who did not ($\chi^2 = 11.6$; $P = 0.0016$).

Table 1: Various surgical procedures and surgical site infection rate

<i>Surgical procedure</i>	<i>Number of cases</i>	<i>Number of cases infected</i>	<i>Percentage</i>
LSCS	19	6	31
Orthopedic procedures	12	2	16.7
Gastric and small bowel	11	7	63.6
Appendectomy	7	3	42.8
Excision of cysts, lipomas*	6	0	00
Uterus and adnexa	6	2	33.3
Urinary tract and genitalia	5	2	40
Breast	3	1	33.3
Large bowel	3	2	66.6
Hernia	3	1	33.3
Amputation@	1	0	000
Others"	5	2	40
Total	80	28	35

*

Dermoid cysts 2, lipoma 4; @Amputation for malignancy, #Thyroidectomy 2, cholelithiasis (hepatobiliary) 2, pancreas 2. LSCS: Lower segment caesarean structure

The infection rate in patients operated under general anesthesia is 45% and under subarachnoid block anesthesia is 25% while one of the patients operated under local anesthesia is infected (12.5%) ($\chi^2 = 18.2$; $P = 0.014$). Infection rate varied with duration of operation with rate of 10.75% in surgeries those lasted for >1 h, which is higher than the rate in surgeries which lasted between 30 min to 1 h (1.3%) ($\chi^2 = 8.6$; $P = 0.036$). None of the wound is infected in surgeries that lasted for <30 min. Use of drain is associated with more infection rate (13.8%). The nondrained wounds have less rate of infection (6.6%) ($\chi^2 = 12.5$; $P = 0.016$).

Surgical site infection was more in patients with preexisting illness such as diabetes (62.5%), hypertension (65%), sickle cell disease (35%) and other past medical/surgical history (tuberculosis, anemia, malaria, jaundice, other high risk surgery in the past 1-year. Polymicrobial infection was experienced by 14 (50%) patients, 14 (23.07%) had *Staphylococcus aureus*, 8 (15.4%) had *E. coli* and 8 (15.4%), had *P. aeruginosa* 6 (11.5%), had *K. pneumoniae* 5 (9.6%) had *Acinetobacter* and 2 (3.8%) cases had *Staphylococcus epidermidis*. Gram-negative organisms were mostly isolated from surgeries on bowel, urinary tract and appendix.

Staphylococcus aureus is the predominant organism infecting LSCS surgeries. No other organism is particularly associated with specific surgery [Tables 2 and 3].

Antibiotic susceptibility testing showed that all the strains of *S. aureus* resistant to penicillin, moderately sensitive to erythromycin (42.8%), tetracycline (28.6%), gentamicin (35.7%) and ciprofloxacin (42.9%). All the strains were sensitive to ceftazidime, means none of the strains were methicillin resistant. *Escherichia coli* was moderately sensitive to ampicillin (32.1%), gentamicin (50%), ciprofloxacin (50%), amikacin (64.3%) and cefotaxime (32.1%) and extensive resistance to tetracycline (89.3%). In case of *K. pneumoniae*, poor sensitivity to ampicillin and gentamicin (14.3%), moderately sensitive to ceftazidime (25%), amikacin and ciprofloxacin (42.9%) was observed. None of the strains were sensitive to tetracycline. *Pseudomonas aeruginosa* was moderately sensitive to ciprofloxacin (46.4%), gentamicin (78.6%) and all the strains sensitive to ceftazidime, amikacin and imipenem. The 2 strains of *Acinetobacter* isolated were sensitive to Amikacin, Imipenem and resistant to Ciprofloxacin, Ceftazidime, Tetracycline and Gentamicin. The 2 strains of *S. epidermidis* isolated were sensitive to all the antibiotics-gentamicin, tetracycline, ciprofloxacin, ceftazidime, cefoxitin except penicillin and erythromycin.

Table 2: Organisms isolated from postoperative infected cases

<i>Organisms isolated</i>	<i>Number of infected cases</i>
S. aureus	7
S. aureus+P. aeruginosa	3
S. aureus+K. pneumoniae	2

E. coli	3
E. coli+P. aeruginosa	2
Acinetobacter+E. coli	3
P. aeruginosa+K. pneumoniae	2
K. pneumoniae	2
P. aeruginosa+Acinetobacter	2
CoNS	2
Total	28

P. aeruginosa: *Pseudomonas aeruginosa*, *S. aureus*: *Staphylococcus aureus*, *K. pneumoniae*: *Klebsiella pneumoniae*, *E. coli*: *Escherichia coli*, CoNS: Coagulase nega Θ ve *staphylococcus*, Cefotaxime resistant

strains of *E. coli* (7 strains) and *K. pneumoniae* (5 strains) were tested for ESBL production by Double Disk, Diffusion Test. All the 12 strains tested were ESBL producing.

Table 3: Organisms isolated from various surgeries

Isolates	<i>S. aureus</i>	<i>E. coli</i>	<i>P. aeruginosa</i>	<i>K. pneumoniae</i>	<i>Acinetobacter</i>	CoNS	Total
Surgery/Surgical Site							
LSCS	5		2		1		8
Bone	1		1				2
Gastric and small bowel		2	1	3	3		9
Appendectomy	1	1	1	2			5
Excision of cysts, lipomas	1		2				3
Uterus and adnexa	2	1	1	1			5
Urinary tract and genitalia		1			1		2
Breast	1						1
Large bowel		2					2
Hernia	1						1
Others		1				2	3
Total (%)	12(29.2)	8(19.5)	8(19.5)	6(14.6)	5(12.2)	2(4.9)	41

LSCS: Lower segment caesarean structure, *P. aeruginosa*: *Pseudomonas aeruginosa*, *S. aureus*: *Staphylococcus*

aureus, *E. coli*: *Escherichia coli*, CoNS: Coagulase nega Θ ve *staphylococcus*, *K. pneumoniae*: *Klebsiella pneumoniae*

Organisms isolated from postoperative wound

Staphylococcus aureus was the predominant organism isolated (29%) followed by Gram-negative organisms. Other studies have also implicated *S. aureus* as the predominant cause of postoperative wound infections.[6,10-12] Among the Gram-negative organisms *E. coli* was the predominant organism (17.9%), followed by *P. aeruginosa* (14.3%), *K. pneumoniae* (14.3%), *Acinetobacter* (17.9%) and *S. epidermidis* (3.6%) [Tables 2 and 3].

Antibiotic susceptibility of isolates

Staphylococcal isolates were 100% resistant to penicillin. Totally, 6 strains (44.4%) are sensitive to erythromycin, 4 strains (33.3%) are sensitive to tetracycline, 5 strains (33.3%) are sensitive to gentamicin, 6 strains (44.4%) are sensitive to ciprofloxacin and all are sensitive to cefotaxime. All the strains are sensitive to ceftazidime, means none of the strains are methicillin resistant. In this study, 3 strains (33.33%) of *E. coli* were sensitive to ampicillin, 5 strains (50%) were sensitive to gentamicin and ciprofloxacin, 6 strains (66.66%) were sensitive to amikacin, 3 strains (33.33%) were sensitive to cefotaxime and only 1 strain (10%) was sensitive to tetracycline. In case of *K. pneumoniae* 1 strain (14.28%), each shows susceptibility to ampicillin and gentamicin, 2 strains (28.57%) were sensitive to cefotaxime and 3 strains (44.4%) to amikacin and ciprofloxacin. None of the strains were sensitive to tetracycline. Cefotaxime resistant strains of *E. coli* (7 strains) and *K. pneumoniae* (5 strains) were tested for ESBL production by Double Disk Diffusion Test. All the 12 strains tested were ESBL producing. The 2 strains of *Acinetobacter* isolated were sensitive to amikacin, imipenem and resistant to

ciprofloxacin, ceftazidime, tetracycline and gentamicin. Three strains (75%) of *P. aeruginosa* were sensitive to gentamicin, and 2 strains (50%) were sensitive to ciprofloxacin. All the strains were sensitive to ceftazidime, amikacin, imipenem and piperacillin. The 2 strains of *S. epidermidis* isolated were sensitive to all the antibiotics-gentamicin, tetracycline, ciprofloxacin, cefotaxime, ceftazidime except penicillin and erythromycin.

DISCUSSION

Postoperative surgical site infections (SSIs) remain a significant concern in both developed and developing countries, despite the implementation of meticulous antiseptic practices in modern surgical care. These infections may arise from endogenous or exogenous sources. In the present study, 28 patients developed postoperative infections, resulting in an SSI rate of 35%, which is comparable to rates reported by other authors [8,11]. The relatively lower infection rates observed in developed countries may be attributed to better infrastructure, stricter infection control protocols, and improved working conditions [12,13]. Conversely, higher rates reported in some studies may be due to the inclusion of contaminated and dirty wounds, as well as emergency surgical procedures [14,15].

Although contaminated and dirty wounds were excluded in this study, emergency surgeries were included, which likely contributed to the higher SSI rate observed. Several studies have demonstrated that appropriate preoperative antibiotic prophylaxis significantly reduces the incidence of postoperative SSIs [16]. This finding is consistent with the present study, where preoperative antibiotic administration was associated with a statistically significant reduction in SSI rates ($\chi^2 = 11.6$; $P = 0.0016$).

A higher incidence of infection was noted among elderly patients, in agreement with previous studies [17]. Advancing age is often associated with comorbid conditions, malnutrition, and decreased immune function, all of which predispose patients to infection [18]. The increased infection rate observed in this study may also be explained by the higher number of young adults undergoing emergency exploratory laparotomies for conditions such as antral and appendicular perforations. Additionally, a higher proportion of female patients undergoing caesarean sections and gynecological surgeries in the third and fifth decades contributed to increased infection rates in these age groups.

A statistically significant association was found between wound classification and infection rates ($\chi^2 = 26.1$; $P = 0.00021$). Class II (clean-contaminated) wounds involve entry into body cavities, thereby increasing the risk of microbial contamination. The infection rates observed for clean and clean-contaminated wounds in this study are consistent with those reported by Anvikar et al. and Sangrasi et al. [15,19].

Prolonged preoperative hospital stay was associated with a higher risk of infection. This may be due to reduced host resistance and increased exposure to hospital-acquired, often antibiotic-resistant, microorganisms [20]. Furthermore, a significantly higher infection rate was observed in patients undergoing surgery under general anesthesia compared to spinal or local anesthesia ($\chi^2 = 18.2$; $P = 0.014$) [21]. The duration of surgery also plays a critical role in SSI development. Prolonged procedures increase the risk of bacterial contamination and tissue damage due to prolonged exposure, surgical manipulation, blood loss, and potential hemodynamic instability, all

of which compromise host defense mechanisms [22].

The use of surgical drains may further increase infection risk, as they can act as conduits for microorganisms between the external environment and the surgical site [23]. Higher infection rates following gastrointestinal, hepatobiliary, pancreatic, and appendectomy procedures are consistent with the known risks associated with abdominal surgeries involving entry into the gastrointestinal tract. These findings are comparable to those reported by Olson et al. [13]. Infections associated with surgeries involving the uterus and adnexa may be linked to comorbid conditions such as diabetes mellitus and sickle cell disease in the study population. A significant proportion of lower segment caesarean section (LSCS) procedures were performed on an emergency basis, which may have contributed to higher SSI rates. Pre-existing illnesses not only increase susceptibility to infection but also prolong preoperative hospital stay, further increasing the risk of bacterial colonization and infection [24].

Microbiologically, Enterobacteriaceae showed high susceptibility to cefotaxime and amikacin, supporting their effectiveness in treating such infections [25]. Similarly, *Pseudomonas aeruginosa* demonstrated sensitivity to amikacin and ceftazidime, indicating their potential utility in managing pseudomonal infections. Previous studies, such as that by Majumder et al. [26], reported oxacillin resistance in 15% of *Staphylococcus epidermidis* and 52.9% of *Staphylococcus aureus* isolates.

CONCLUSION

Postoperative surgical site infections are influenced by multiple risk factors, including emergency surgeries, prolonged hospital stay, comorbidities, and duration of surgery. *Staphylococcus aureus* was the most

common pathogen, though Gram-negative organisms were also frequently isolated. Appropriate preoperative antibiotic use, reduction in hospital stay, and proper management of underlying conditions can significantly reduce SSI rates. The emergence of drug-resistant organisms highlights the need for rational antibiotic use and continuous surveillance to prevent further resistance.

REFERENCES

1. Kolasinski W. Surgical site infections - review of current knowledge, methods of prevention. *Pol Przegl Chir.* 2019;91(4):41-7.
2. Prabhakar H, Arora S. A bacteriological study of wound infections. *J Indian Med Assoc.* 1979;73:145-8.
3. Garner BH, Anderson DJ. Surgical Site Infections. *Infect Dis Clin N Am.* 2016;30(4):909-29. doi:10.1016/j.idc.2016.07.010
4. Emori TG, Gaynes RP. An overview of nosocomial infections, including the role of the microbiology laboratory. *Clin Microbiol Rev* 1993; 6:428-42.
5. Bradford PA. Extended-spectrum beta-lactamases in the 21st century: Characterization, epidemiology, and detection of this important resistance threat. *Clin Microbiol Rev* 2001;14:933-51.
6. Garner JS. CDC guideline for prevention of surgical wound infections, 1985. Supersedes guideline for prevention of surgical wound infections published in 1982. (Originally published in November 1985). Revised. *Infect Control* 1986;7:193-200.
7. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. *Infect Control Hosp Epidemiol* 1999;20:250-78.
8. Siguan SS, Ang BS, Pala IM, Baclig RM. Aerobic surgical infection: A surveillance on microbiological etiology and antimicrobial sensitivity pattern of commonly used antibiotics. *Philipp J Microbiol Infect Dis* 1990;19:27-33.
9. Collee JG, Duguid JP, Fraser AG, Marmion BP, Simmons A. Laboratory strategy in the diagnosis of infective syndromes. In: Collee JG, Marmion BP, Fraser AG, Simmons A, editors. Mackie and McCartney Practical Medical Microbiology. 14th ed. London: Churchill Livingstone; 2006. p. 53-94.
10. Clinical and Laboratory Standards Institute. "Performance Standards for Antimicrobial Susceptibility Testing," Fifteenth Informational Supplement. Approved Standard MS100-S16. Wayne, PA: CLSI; 2006.
11. Anvikar AR, Deshmukh AB, Karyakarte RP, Damle AS, Patwardhan NS, Malik AK, et al. A one year prospective study of 3280 surgical wounds. *Indian J Med Microbiol* 1999;17:129-32.
12. Cruse P. Wound infection surveillance. *Rev Infect Dis* 1981;3:734-7.
13. Olson M, O'Connor M, Schwartz ML. Surgical wound infections. A 5-year prospective study of 20,193 wounds at the Minneapolis VA Medical Center. *Ann Surg* 1984;199:253-9.
14. Agarwal PK, Agarwal M, Bal A, Gahlaut YV. Incidence of post-operative wound infection at Aligarh. *Indian J Surg* 1984;46:326-33.
15. Sangrasi AK, Leghari AA, Memon A, Talpur AK, Qureshi GA, Memon JM. Surgical site infection rate and associated risk factors in elective general surgery at a public sector medical university in Pakistan. *Int Wound J* 2008;5:74-8.
16. Gupta R, Sinnett D, Carpenter R, Preece PE, Royle GT. Antibiotic prophylaxis for post-operative wound infection in clean elective breast surgery. *Eur J Surg Oncol* 2000;26:363-6.

17. Moro ML, Morsillo F, Tangenti M, Mongardi M, Pirazzini MC, Ragni P, *et al.* Rates of surgical-site infection: An international comparison. *Infect Control Hosp Epidemiol* 2005;26:442-8.
18. McNicol L, Story DA, Leslie K, Myles PS, Fink M, Shelton AC, *et al.* Postoperative complications and mortality in older patients having non-cardiac surgery at three Melbourne teaching hospitals. *Med J Aust* 2007;186:447-52.
19. Moorhouse E, Fenelon L, Hone R, Smyth E, McGahon J, Dillon M. *Staphylococcus aureus* sensitivity to various antibiotics — a national survey in Ireland 1993. *Ir J Med Sci* 1996;165:40-3.
20. Nagachinta T, Stephens M, Reitz B, Polk BF. Risk factors for surgicalwound infection following cardiac surgery. *J Infect Dis* 1987;156:967-73.
21. Rodgers A, Walker N, Schug S, McKee A, Kehlet H, van Zundert A, *et al.* Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: Results from overview of randomised trials. *BMJ* 2000;321:1493.
22. Haley RW, Culver DH, Morgan WM, White JW, Emori TG, Hooton TM. Identifying patients at high risk of surgical wound infection. A simple multivariate index of patient susceptibility and wound contamination. *Am J Epidemiol* 1985;121:206-15.
23. Claesson BE, Holmlund DE. Predictors of intraoperative bacterial contamination and postoperative infection in elective colorectal surgery. *J Hosp Infect* 1988;11:127-35.
24. Margenthaler JA, Longo WE, Virgo KS, Johnson FE, Oprian CA, Henderson WG, *et al.* Risk factors for adverse outcomes after the surgical treatment of appendicitis in adults. *Ann Surg* 2003;238:59-66.
25. Duttaroy B, Mehta S. Extended spectrum b lactamases (ESBL) in clinical isolates of *Klebsiella pneumoniae* and *Escherichia coli*. *Indian J Pathol Microbiol* 2005;48:45-8.
26. Majumder D, Bordoloi JS, Phukan AC, Mahanta J. Antimicrobial susceptibility pattern among methicillin resistant *Staphylococcus* isolates in Assam. *Indian J Med Microbiol* 2001;19:138-40.