Research Article

DOI: 10.5455/2320-6012.ijrms20140552

A study of superficial surgical site infections in a tertiary care hospital at Bangalore

Saroj Golia, Asha S. Kamath B*, Nirmala AR

Department of Microbiology, Dr B. R. Ambedkar Medical College, Bangalore - 560045, Karnataka, India

Received: 5 March 2014 Accepted: 25 March 2014

*Correspondence:

Dr. Asha S. Kamath B, E-mail: drashakamath@yahoo.co.in

© 2014 Golia S et al. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: All postoperative surgical infections occurring in an operative site are termed surgical site infections (SSI). Superficial incisional surgical site infection occurs within 30 days after the operation and infection involves only skin or subcutaneous tissue of the incision and represents a substantial burden of disease for patients and health services. The study was conducted to know the incidence of surgical site infection in our hospital, risk factors associated with it and the antibiotic susceptibility pattern of the pathogens.

Methods: This prospective study was carried out in the Department of Microbiology at Dr B R AMC for a period of 1 year from Jan 2013to Jan 2014. Samples of SSI received in the Microbiology laboratory were processed and Data collected.

Results: The overall surgical site infection rate in our hospital during the study period is 4.3%. Staphylococcus aureus (S. aureus) was the most common isolate obtained followed by Escherichia coli (E. coli) and Coagulase negative Staphylococcus (CONS). Other organisms isolated were Pseudomonas aeruginosa, Enterococcus, Klebsiella pneumoniae and Proteus mirabilis. Among them, 88.8% of S. aureus and 50% of CONS isolates were methicillin-resistant strains. 80% of E. coli and 100% of Klebsiella species were ESBL producers. 50% of Enterococci were Vancomycin resistant. Risk factors like diabetes mellitus and duration plays a significant role in causing surgical site infection.

Conclusion: Implementation of an effective infection control programme and judicious use of antibiotic prophylaxis reduces the incidence of SSI in the hospital.

Keywords: Surgical site infection, Wound infection, Incision, Postoperative infection

INTRODUCTION

Skin is a natural barrier against infection, so any surgery that causes a break in the skin can lead to a postoperative infection. Any purulent discharge from a closed surgical incision, together with signs of inflammation of the surrounding tissue should be considered as wound infection, irrespective of whether micro-organisms can be cultured. Infection can occur at an incision site within 30 days of an operation, but wounds that are closed and primarily healed are not considered infected.¹ Surgical

site infection (SSI) is an important postoperative complication. Based on extensive epidemiologic surveys, it has been estimated that SSI develops in at least 2% of hospitalized patients undergoing operative procedures, although this is a likely underestimate because of incomplete post-discharge data.²

Criteria for superficial surgical site infection are: Infection within 30 days after the operation and only involves skin and subcutaneous tissue of the incision and at least one of the following:

- 1. Purulent drainage with or without laboratory confirmation, from the superficial incision.
- 2. Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision.
- 3. At least one of the following signs or symptoms of infection: pain or tenderness, localised swelling, redness, or heat and superficial incision are deliberately opened by surgeon, unless incision is culture-negative.
- 4. Diagnosis of superficial incisional SSI made by a surgeon or attending physician.³

Factors underlying nosocomial postoperative infection are multiple and include the type of surgical procedure, the skills of the surgeon, the duration of surgery and the underlying disease of the host.⁴ Both infection and wound healing are adversely influenced by poorly controlled diabetes mellitus. Age is considered an important factor, with neonates and the elderly at particular risk of infection.⁵ Life style can also impinge on immunocompetency especially stress, alcohol and drug abuse, smoking and lack of exercise or sleep.⁵

A multivariate index combining patient susceptibility and wound contamination was developed and tested during the CDC Study on the Efficacy of Nosocomial Infection Control (SENIC).⁶ This index involves the following four risk factors:

- 1. An operation that involves the abdomen;
- 2. An operation lasting longer than 2 hours;
- 3. An operation classified as either contaminated, dirty, or infected; and
- 4. A patient having three or more discharge diagnoses.

SSIs are a consequence of a summation of several factors: the inoculum of bacteria introduced into the wound during the procedure, the virulence of the contaminants, the microenvironment of each wound, and the integrity of the patient's host defence mechanisms. Factors intrinsic to the patient, as well as those related to the type and circumstances of surgery, affect the incidence of infection.⁷

SSI arises secondary to exogenous or endogenous bacterial contamination at the time of the operative procedure. Bacterial proliferation results in tissue reaction and outpouring of inflammatory cells, leading to tissue destruction and pus formation. The presence of local factors such as necrosis, haematoma, and dead space provide bacteria with a milieu for growth, and the presence of other foreign bodies inhibits local tissue resistance.⁸

Microorganisms may contain or produce toxins and other substances that increase their ability to invade a host, produce damage within the host, or survive on or in host tissue. Many gram-negative bacteria produce endotoxin, which stimulates cytokine production. In turn, cytokines can trigger the systemic inflammatory response syndrome that sometimes leads to multiple system organ failure.⁹

METHODS

This prospective study was carried out in the department of microbiology at Dr B. R. AMC for a period of 1 year from Jan 2013to Jan 2014.

A total number of 28 samples of suspected surgical site infection were obtained from 650 surgical cases during the period of 1 year from Jan 2013 to Jan 2014.

Samples were received in the laboratory either as a wound swab or as an aspirate from the infected wound. The samples were immediately cultured on blood agar, Mac Conkey's agar and nutrient agar, followed by the identification of the isolates based on their colony characteristics and biochemical reactions.

The Kirby Bauer disc diffusion method was used for the antimicrobial susceptibility patterns following Clinical Laboratory Standards Institute (CLSI) guidelines. The included antibiotics are:

AMP - ampicillin, CPZ - cefoperazone, AK - amikacin, GEN - gentamicin, CAZ - ceftazidime, CAC ceftazidime-clavulanic acid, CTX - cefotaxime, PIT pipercillin-tazobactam, IMP - imipenem, P - penicillin, AMC - amoxyclav, CX - cefoxitin, E - erythromycin, CD - clindamycin, LZ- linezolid, VA - vancomycin, TEIteicoplanin.

The results were recorded and interpreted as per the recommendations of the clinical laboratory standards institute. A cefoxitin (30 μ g) was used for determination of methicillin resistance in S. aureus and a vancomycin disc (30 μ g) was used to determine vancomycin resistance in Enterococcus spp.

The screening for ESBL production was done by the phenotypic confirmatory test by using ceftazidime disk in the presence of clavulinic acid. A greater than or equal to 5mm increase in the zone diameter with clavulanic acid and ceftazidime versus ceftazidime alone confirmed an ESBL production.

RESULTS

The overall surgical site infection rate in our hospital during the study period is 4.3%.

Out of the 28 isolates obtained, 2 were culture negative (7.14%). A single etiologic agent was identified and isolated in all the cases.

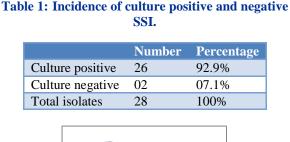




Figure 1: Incidence of culture positive and negative SSI.

Table 2: Various positive and negative isolates from
the sample.

Isolate	Number	%
S. aureus	09	32.2%
E. coli	05	17.9%
CONS	04	14.3%
Pseudomonas aeruginosa	03	10.7%
Enterococcus species	02	7.1%
Klebsiella pneumoniae	02	7.1%
Proteus mirabilis	01	3.6%
Culture negative	02	7.1%
Total	28	100%

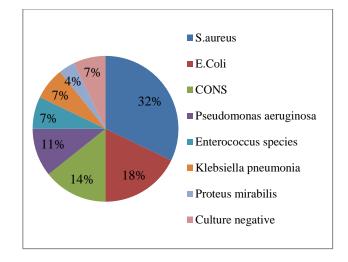


Figure 2: Various positive and negative isolates from the sample.

The commonest etiologic agents were S. aureus and E. coli. In total, 32.2% (n = 9) of the organisms isolated were S. aureus of which 88.8% (n = 8) were methicillinresistant strains (MRSA). 14.3% (n = 4) were CONS among which 50% (n=2) were methicillin resistant and 7.1% (n = 2) were Enterococcus species out of which 50% (n=1) were vancomycin resistant.

Among gram negative organisms, 17.9% (n = 5) were E. coli, 10.75 (n=3) were Pseudomonas aeruginosa, 7.1% (n=2) were Klebsiella spp. and 3.6% (n = 1) was Proteus mirabilis. 80% (n = 4) of E. coli and 100% (n = 2) of Klebsiella species were ESBL producers.

Table 3: Resistance pattern in gram positive organisms.

	Р	AK	AMP	AMC	E	CD	GEN	LE	СХ	VA	TEI	LZ
S. aureus	8	1	7	2	2	3	3	3	8	0	0	0
CONS	3	0	0	0	2	0	0	0	2	0	0	0
Enterococci	1	0	0	0	0	0	0	0	0	1	0	0

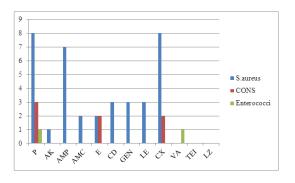


Figure 3: Resistance pattern in gram positive organisms.

SSI were also associated with certain risk factors like underlying disease, duration of surgery and length of hospital stay. Among the isolates, 80.8% of them were from surgeries which lasted for more than 2 hours and 19.2% from surgeries which lasted for less than 2 hours.46.2% of patients who had a hospital stay for more than 20 days developed SSI.

Among 650 patients who underwent surgery during the study period, 102 were diabetic, out of which 20.58% (n = 21) developed SSI.

	AK	AMP	CTX	CTR	CPZ	CAZ	CAC	PIT	CPM	GEN	IMP	COT	TGC	OF
E. coli	2	4	4	4	4	4	4	1	1	3	0	0	0	0
K. pneumoniae	2	2	2	2	2	2	2	1	2	1	0	0	0	0
P. mirabilis	0	0	1	0	0	0	0	0	1	1	0	0	0	0
P. aeruginosa	1	3	3	1	1	3	3	3	1	2	3	0	0	0



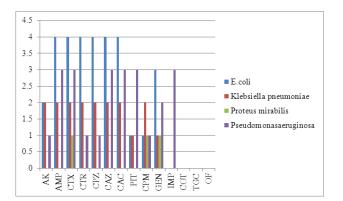


Figure 4: Resistance pattern in gram negative organisms.

Table 5: Comparison of incidence of superficialincisional surgical site infection with duration ofsurgery.

Duration	No. of cases	Percentage
<2 hours	5	19.2%
>2 hours	21	80.8%

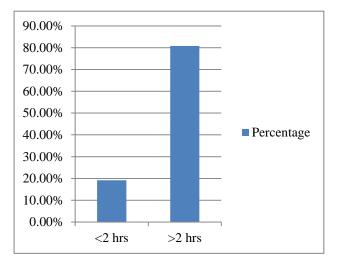


Figure 5: Comparison of incidence of superficial incisional surgical site infection with duration of surgery.

Table 6: Superficial incisional SSI in relation to length of postoperative hospital stay.

Duration of stay (days)	No. of cases	Percentage
1-5	1	3.9%
6-10	3	11.5%
11-15	4	15.3%
15-20	6	23.1%
>20	12	46.2%

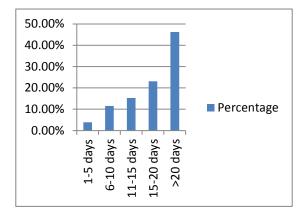


Figure 6: Superficial incisional SSI in relation to length of postoperative hospital stay.



Figure 7: Gram negative organism showing ESBL production.

Difference between zone of inhibition of ceftazidime (CAZ) and ceftazidime - clavulanic acid (CAC) is >5 mm indicating ESBL production

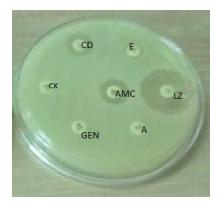


Figure 8: S. aureus showing resistance to cefoxitin.

No zone of inhibition around cefoxitin disc (CX) indicates the MRSA strains

DISCUSSION

A recent prevalence study found that SSIs were the most common health care-associated infection, accounting for 31% of all HAIs among hospitalized patients.¹⁰ Anvikar et al. demonstrated that preoperative hospital stay predisposed an individual to 1.76% risk of acquiring an infection.¹¹ Prolonged postoperative hospitalization, which is a major concern of most of the hospitals, has been evident in patients developing surgical site infection.¹²

In our study, out of 650 surgeries that were conducted during the study period, 28 cases developed surgical site infection which amounts to the overall infection rate of 4.3%. This similar to a study conducted by S.K. Sahu.et al where the incidence of infection was 5%.¹³ The infection rate in a study conducted by Lilani et al. is 8. 95%.¹⁴ In a study conducted by Suchitra Joyce B and Lakshmidevi N. reported that 12% of patients undergoing surgeries developed SSI.¹⁵

None of the patients in our study developed signs of infection at the surgical site within 48 hours of surgery. 88.8 % of S. aureus and 50% of CONS isolates were methicillin-resistant strains (MRSA). 80% of E. coli and 100% of Klebsiella species were ESBL producers. 50% of enterococci were vancomycin resistant. This indicates that the drug resistant strains are the major causes of SSI. Patients who had a prolonged hospital stay (>20 days) had 4 times more incidence of SSI, indicating that it contributes to the development of SSIs. 88.8% Patients for whom the duration of surgery was more than 2 hours developed SSI. 20.58% of diabetics developed SSI.

To conclude, our study reveals a superficial incisional surgical site infection incidence rate of 4.3%. Length of stay, duration of surgery and Diabetes mellitus were found to be major risk factors responsible for causing surgical site infection. Minimizing the incidence of postoperative wound infection relies on adequate asepsis

and antisepsis, and preservation of the local host defences.

Funding: No funding sources Conflict of interest: None declared Ethical approval: Not required

REFERENCES

- PL Nandi, S Soundara Rajan, KC Mak, SC Chan, YP S. Surgical wound infection. Hong Kong Med J. 1999 Mar;5(1):82-6.
- 2. Klevens RM, Edwards JR, Richards CL Jr et al. Estimating health care associated infections and deaths in U.S. hospitals, 2002. Public Health Rep. 2007;122:160-6.
- Horan TC, Gaynes RP, Martone WJ, Jarvis WR, Emori TG. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. Infect Control Hosp Epidemiol. 1992;13:606-8.
- 4. Mustafa A, Burkhari A, Kakru KD. Incidence of nosocomial wound infection in postoperative patients at a teaching hospital in Kashmir. JK Practitioner. 2004;11:38-40.
- Cooper RA. Understanding wound infection, European Wound Management Association (EWMA). In: Cooper RA, eds. Identifying Criteria for Wound Infection. 1st ed. London: MEP Ltd; 2005: 6-9.
- 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(2):206-15.
- Aldo Cunha Medeiros, Tertuliano Aires-Neto, George Dantas Azevedo, Maria José Pereira Vilar, Laíza Araújo Mohana Pinheiro, José Brandão-Neto. Surgical site infection in a university hospital in north-east Brazil. Braz J Infect Dis. 2005 Aug;9(4):310-4.
- 8. Lawal OO, Adejuyigbe O, Oluwole SF. The predictive value of bacterial contamination at operation in post-operative wound sepsis. Afr J Med Sci. 1990;19(3):173-9.
- Demling R, LaLonde C, Saldinger P, Knox J. Multiple-organ dysfunction in the surgical patient: pathophysiology, prevention, and treatment. Curr Probl Surg. 1993;30(4):345-414.
- 10. CDC. CDC/NHSN protocol clarifications, 2013. Available at: http://www.cdc.gov/nhsn/pdf/pscmanual/protocolclarification.pdf.
- 11. Anvikar AR, Deshmukh AB, Karyakarte RP, Damle AS, Patwardhan NS, Malik AK, Bichile LK, Bajaj JK, Baradkar VP, Kulkarni JD, Sachdeo SM. A one year prospective study of 3280 surgical wounds. Indian J Med Microbiol. 1999;17:129-32.

- 12. Nichols RL. Prevention of infection in high risk gastrointestinal surgery. Am J Med. 1984;76:111-9.
- S. K. Sahu, J. S. Shergill., P. K. Sachan, P. Gupta. Superficial incisional surgical site infection in elective abdominal surgeries: a prospective study. Int J Surg. 2011;26(1):1.
- SP Lilani, N Janghaley, A Chowdhary, GB Daver. Surgical site infection in clean and clean contaminated cases. Indian J Med Microbiol. 2005;23(4):249-54.
- 15. Suchitra Joyce B. and Lakshmidevi N. Surgical site infections: assessing risk factors, outcomes and antimicrobial sensitivity patterns. African J Microbiol Res. 2009 Apr;3(4):175-9.

DOI: 10.5455/2320-6012.ijrms20140552 **Cite this article as:** Golia S, Kamath A, Nirmala AR. A study of superficial surgical site infections in a tertiary care hospital at Bangalore. Int J Res Med Sci 2014;2:647-52.