

Surveillance Method for Surgical Site Infection

Rochak Goyal^{1*}, Harinder Pal Singh Sandhu², Ashwani Kumar², Sourabh Kosey¹ and Neelesh Mehra¹

¹Department of Pharmacy Practice, I.S.F College of Pharmacy, Moga, India.

²Department of Surgery-II, GGSMCH, Faridkot, Punjab, India.

ABSTRACT

Surgical Site Infection (SSI) is the third most commonly reported Nosocomial infection and accounts for 14–16% of all Nosocomial infections among hospital inpatients. The morbidity, mortality and the cost to health services of surgical site infections is huge. In addition, many workers have shown that feedback of appropriate data to surgeons has been an indispensable component of strategies to reduce SSI rates. The elements essential for a successful programmed of prevention of SSIs include intensive surveillance, infection control activities and regular feedback of SSI rates to surgeons. Surveillance with information feedback to surgeons and other medical staff has been shown to be an important element in the overall strategy to reduce the numbers of SSIs. Despite the apparent effectiveness in lowering SSI rates when surgeons receive feedback, however, there has been no consensus on which surveillance methods are best for collecting data on SSIs. A successful SSI surveillance program should include standardized definitions of infection, effective surveillance methods and stratification of the SSI rates according to risk factors associated with the development of SSI. For many years wound contamination class was the only factor that was well described for predicting the risk for SSI. During the Study on the Efficacy of Nosocomial Infection Control (SENIC) Project, an index was developed that provided a better assessment of the risk of SSIs than had the traditional wound classification system. In 1991, a modification of the SENIC risk index by *Culver et al.* led to the National Nosocomial Infections Surveillance (NNIS) System risk index. This review examines the best surveillance method for surgical site infection.

Key words: National Nosocomial infection surveillance, Nosocomial infection, Study of the Efficacy of Nosocomial Infection Control (SENIC) Score, Surgical site infection, Wound classification system.

INTRODUCTION

Quality of healthcare in hospitals is of major public health importance. Surgical site infections (SSI) are the most common Hospital acquired infection. Despite the advances made in asepsis, antimicrobial drugs, sterilization and operative techniques, surgical site infections (SSI) continue to be a major problem in all branches of surgery in the hospitals.¹ The morbidity, mortality and the cost to health services of surgical site infections is huge. In the United States alone there are an estimated 27 million surgical procedures performed each year with almost one-third of patients over the age of sixty-five years.² Surgical site infections are the third most frequently reported Nosocomial infection, accounting for 14–16% of all Nosocomial infections among hospital inpatients.³ During 1986–1996, hospi-

tals conducting SSI surveillance employing the Center for Disease Control's (CDC's) National Nosocomial Infections Surveillance (NNIS) System reported 15523 SSIs following 593344 surgical procedures.⁴ Among surgical patients, SSIs accounted for 38% of Nosocomial infections and were the commonest Nosocomial infection encountered.⁵ Two-thirds of SSIs were confined to the surgical incision and one third involved organs or spaces accessed during the surgical procedure. Seventy-seven percent of patients who died with an SSI were reported as having the infection causally related to death.

The Study of the Efficacy of Nosocomial Infection Control (SENIC) investigators showed that an adequately designed and funded SSI surveillance programme could

Submitted date : 05/07/2015

Accepted date : 06/18/2015

DOI: 10.5530/ijopp.8.2.2

Address for

correspondence:

Mr. Rochak Goyal,

Department of Pharmacy

Practice, I.S.F College of Pharmacy, Moga, India.

E-mail : goyal.rochak@yahoo.com



www.ijopp.org

be expected to decrease the overall rates by 32%.⁶ However, only a small percentage (8%) occurred in hospitals recruited.⁶ In addition, many workers have shown that feedback of appropriate data to surgeons has been an indispensable component of strategies to reduce SSI rates.⁶ However, not all workers have shown a reduction in SSI rates after continuous surveillance.⁷ The elements essential for a successful programme of prevention of SSIs include intensive surveillance and infection control activities and regular feedback of SSI rates to surgeons.⁸ Despite the apparent effectiveness in lowering SSI rates when surgeons receive feedback, there has been no consensus on which surveillance methods are best for collecting data on SSIs.⁹ In addition, there is no evidence to support an argument that high frequency reporting (e.g., monthly) yields better infection control than lower frequency reporting (e.g., quarterly or semi-annually).¹⁰ In devising an SSI surveillance programme, consideration needs to be given to the definitions of infection, data collection, handling and analysis, presentation of results and which patients are to be included.¹¹

Surveillance components

• Objectives and priorities

There should be clear and unambiguous objectives and priorities regarding an SSI surveillance programme.¹¹ Obviously the major aim is to reduce the rate of SSI, thereby reducing patient morbidity and mortality.¹² This should also result in financial savings for the hospital.¹³ Depending on infection control personnel available, and the willingness of clinical staff to become involved, surveillance will have to be tailored to the art of the possible rather than risk the integrity of the total surveillance strategy by being over ambitious. Surveillance initiatives require total staff involvement lest they become something that the infection control team undertakes but is of little relevance to the clinical areas.

• Definition and Classification of Surgical Site Infection

One of the most important aspects of SSI surveillance is the definition of infection.¹³ It is crucial that a surveillance programme uses standardized definitions otherwise inaccurate and misleading results may be obtained and reported. In addition, to compare data over time it is essential that the definitions should remain unchanged so that baseline SSI rates may be established, patients' risk of developing SSI stratified, results of interventions analyzed and the possibility of inter hospital comparisons considered.¹⁴

The most widely used definition of SSI is that employed by the CDC's NNIS System (Table 1).¹⁵ The previous CDC definitions published in 1988 considered surgical wound infection (SWI) related to the skin incision only whereas the current definition now classifies SSIs into

Incisional or organ/space and has also introduced the change in terminology from SWI to SSI.¹⁵

SSI are classified based on the depth of involvement of the infection, which may be confined to the skin and subcutaneous tissues (superficial Incisional SSI), involve the deep soft tissue, such as the facial and muscular layers (deep Incisional SSI), or extend further beyond these anatomic boundaries (organ/space SSI). Incisional SSIs are further subdivided into primary and secondary for cases with more than one incision. For instance, a primary Incisional SSI involves the primary incision (e.g., chest incision for coronary artery bypass grafting), and a secondary Incisional SSI involves secondary incisions (e.g., leg incision for donor site in coronary artery bypass grafting).

EPIDEMIOLOGY

The development of multiple aspects of modern surgical care has led to significant improvements in the historical context described. Nevertheless, SSIs remain a frequent postoperative complication, developing in 3% to 20% of surgical procedures.¹⁶

The rate of SSI is highly variable depending on the specific operative procedure, with rates that can be even higher depending on the number of risk factors present. There is a substantial impact of SSI on both morbidity and mortality. However, establishing the exact impact of SSI is difficult because of the dependence on accuracy of reporting and the variability of patient follow-up. In the 1980s, it was observed that SSI led to a 10-day increase in hospital length of stay.¹⁷

Even a decade later, another study reported persistent delayed discharge from hospital and increased requirement for post-discharge care.¹⁸ In a study of 288,906 patients, in-hospital mortality for the patients with SSIs was 14.5% versus 1.8% of patients with no SSI. SSIs are estimated to be responsible for more than 8000 deaths annually in the United States.¹⁶

SSIs may be of even greater consequence in developing countries, because surveillance rates of SSI in a study conducted by the International Nosocomial Infection Control Consortium were higher for most surgical procedures compared with CDC-NHSN rates.¹⁹

SURVEILLANCE METHODS

A number of methods for the surveillance of Nosocomial infections have been developed and their sensitivity and specificity have been assessed. Surveillance has been described as a preventive measure for reducing such infections. A successful surveillance system that uses standard definitions, which feedback data on-site-specific, risk-adjusted SSIs rates may provide a measure of quality performance for surgeons and hospitals and

Table 1: National Healthcare Safety Network definitions for Surgical Site Infection

Superficial Incisional Surgical Site Infection	
Infection occurs within 30 days after the operative procedure and involves only skin and subcutaneous tissue of the incision and patient has at least 1 of the following:	
A. Purulent drainage from the superficial incision	
B. Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision	
C. At least 1 of the following signs or symptoms of infection: pain or tenderness, localized swelling, redness, or heat, and superficial incision is deliberately opened by surgeon and is culture positive or not cultured. A culture-negative finding does not meet this criterion	
D. Diagnosis of superficial incisional SSI by the surgeon or attending physician	
Deep Incisional Surgical Site Infection	
Infection occurs within 30 days after the operative procedure if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operative procedure and involves deep soft tissues (e.g., facial and muscle layers) of the incision and patient has at least 1 of the following:	
A. Purulent drainage from the deep incision but not from the organ/space component of the surgical site	
B. A deep incision is deliberately opened by a surgeon and is culture-positive or not cultured when the patient has at least 1 of the following signs or symptoms: fever (>38° C) or localized pain or tenderness. A culture-negative finding does not meet this criterion	
C. An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopathology or radiologic examination	
D. Diagnosis of a deep Incisional SSI by a surgeon or attending physician	
Organ/Space Surgical Site Infection	
Infection occurs within 30 days after the operative procedure if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operative procedure and infection involves any part of the body, excluding the skin incision, fascia, or muscle layers, that is opened or manipulated during the operative procedure and patient has at least 1 of the following:	
A. Purulent drainage from a drain that is placed through a stab wound into the organ/space	
B. Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ/space	
C. An abscess or other evidence of infection involving the organ/space that is found on direct examination, during reoperation, or by histopathology or radiologic examination	
D. Diagnosis of an organ/space SSI by a surgeon or attending physician.	
From horantc, andrus m, dudeck ma. Cdc/nhsn surveillance definition of health care associated infection and criteria for specific types of infections in the acute care setting. (Am j) Infect Control 2008; 36:313-4.	

Table 2: The National Nosocomial Infections Surveillance (NNIS) risk index classification for predicting surgical site infection.²¹

Risk Factor	Score ascribed	
	0	1
Physical condition of patient according to the ASA classification	<3	=3
Class of contamination of surgical wound according to the NRC classification	Clean or potentially contaminated	Contaminated or infected
Length of surgery (in terms of the 75 percentile for the procedure)	≤75	>75

contribute to the prevention of hospital acquired infections.

For many years, wound contamination class was the only factor that was well described for predicting the risk for SSI. During the Study on the Efficacy of Nosocomial

Infection Control (SENIC) Project, an index was developed that provided a better assessment of the risk of SSIs than had the traditional wound classification system.

In 1991, a modification of the SENIC risk index by Culver *et al.* led to the National Nosocomial Infections

Table 3: Risk index classification of the American Society of Anesthesiology (ASA)²¹

Classification	Physical condition of the patient
1	Normally healthy
2	Discrete systemic disease.
3	Serious, non-incapacitating, systemic disease
4	Life-threatening, incapacitating systemic disease.
5	Moribund with death expected within 24 hrs.

Table 4: CDC wound classification system

Class/Classification	Potential for Contamination
Class I/clean	Surgical wounds that exhibit no infection or inflammation; operations not involving the entry of the uninfected respiratory, digestive, genital or urinary tracts. Operations in which aseptic conditions are fully maintained: surgical wounds are primarily closed and, if necessary, drained using a closed system. Surgical wounds after non-penetrating trauma injuries are included in this class if they fulfill the above criteria.
Class II/potentially contaminated	Surgeries involving opening of the respiratory, digestive, genital or urinary tracts under controlled contaminated conditions and without abnormal contamination. Operations involving biliary tract, appendix, vagina and oropharynx that exhibit no evidence of infection and where aseptic conditions are fully maintained are included in this class.
Class III/contaminated	Fresh (within 7 h of causal event), open trauma injuries. Surgical procedures with a major in sterile technique (open heart surgery), or with significant contamination from the gastrointestinal tract. Wounds with acute, non-purulent Inflammation are included in this class.
Class IV/infected/dirty	Old (more than 7 h after causal event) trauma injuries with devitalized tissue and with preexisting clinical infection or perforated viscera. This definition suggests that organisms giving rise to postoperative infection were present in the surgical area prior to the surgery.

Surveillance (NNIS) System risk index (Table 2). The NNIS index ranges from 0 to 3 with increasing risk and is raised by 1 point for each of 3 SSI predictors:²⁰

First, American Society of Anesthesiologists (ASA) classification (Table 3) (range, 1-5), as a measure of poor overall preoperative physical status of the patient.

Second, wound contamination class >2 (range, 1-4), corresponding to a contaminated or dirty-infected operation.

Third, duration of operation of >75th percentile (P75) for the specific procedure group, is associated with a greater risk of infection, for example, complexity of the case.

The CDC wound classification system defines wound class based on risk and is divided into 4 categories: clean, clean-contaminated, contaminated, and dirty (Table 4)²¹ With clean wounds, the expected risk is from microbes located directly on the surface of the skin, or introduced from the external environment. With increasing wound class, there is increased exposure to microorganisms that are present on internal structures of the body, such as epithelial surfaces of the gastrointestinal tract and genitourinary tract. In the early epidemiologic studies, the SSI rate increased with wound class (I: 2.1%; II: 3.3%; III: 6.4%; IV: 7.1%).²²

MICROBIOLOGY

SSI is caused by microorganisms introduced into the surgical wound at the time of the operative procedure.

Most of these microorganisms come from the patient's endogenous flora, but occasionally the pathogenic organisms are acquired from an exogenous source, such as the air in the operating room, surgical equipment, implants or gloves, or even medications administered during the operative procedure.^{23,24} When there is an unexplained local outbreak of SSI, investigations performed by infection control personnel may be useful in uncovering an exogenous source.

Large, cross-institutional surveys involving all surgical specialties have revealed that a small number of gram-positive cocci and gram-negative bacilli are responsible for most SSIs. The NNIS system categorized 17,671 isolates obtained from patients with SSI from 1986 to 1996.²⁵ Over one half of the isolates were gram-positive cocci; *Staphylococcus aureus* was the most commonly isolated organism, followed by coagulase-negative staphylococci, and *Enterococcus* spp. Approximately one third of the isolates were gram-negative bacilli, with *Escherichia coli*, *Pseudomonas aeruginosa*, and *Enterobacter* spp being the most frequently encountered gram-negative organisms. About 5% of the isolates were anaerobic bacteria.

More recent surveys involving multiple²⁶ or single institutions^{27,28} have these general findings, although the specific distribution of organisms differs somewhat, probably reflecting different types of surgical practices at individual institutions. This general pattern significant

masks variability in the microbiology of SSI according to the type of operative procedure.^{23,24} For patients undergoing clean procedures, Staphylococci predominate as the cause of SSI, since these microorganisms are present on the skin at the site of most incisions. However, gram-negative and other enteric organisms colonize the skin at certain sites, including the axilla, perineum and groin; patients having incisions in those areas may have a SSI caused by gram-negative organisms. Thus, patients undergoing coronary artery bypass surgery are likely to have gram-positive organisms as the cause of a sternal wound infection, but are frequently found to have gram-negative organism as the cause of a leg wound infection.²⁹ With clean-contaminated or contaminated wounds, bacteria from the respiratory, gastrointestinal, genital, or urinary tracts contribute to the infection. For instance, gram-negative bacilli and anaerobic organisms are frequent causes of SSI following procedures involving the lower gastrointestinal tract.²³ Nonetheless, organisms derived from the skin may still contribute to these infections.

In a recent trial of prophylactic antibiotics for subjects undergoing colorectal procedures, 11% of all isolates obtained from subjects with SSI were staphylococci, most of which were *S. aureus*.³⁰ With Class IV (dirty-infected) wounds, it is generally assumed that pathogenic organisms already present in the operative field will be responsible for a subsequent SSI.²³ Finally, it should be noted that unique microbiological patterns may pertain to certain highly specialized procedures; for instance, enterococci are frequently found to be the pathogens causing SSI after liver transplantation.³¹ The most significant change in the microbiology of SSI has been the increased involvement of resistant microorganisms in these infections.

An increased occurrence of infections due to MRSA has also been recognized in studies of subjects undergoing cardiac, orthopedic, or plastic surgery procedures.³⁴⁻³⁷ The emergence of the USA300 clone of MRSA, commonly referred to as community-acquired MRSA, may further impact the microbiology of SSI. This strain is recognized as being responsible for significant numbers of serious hospital-acquired staphylococcal infections;^{38,39} a preliminary report also suggests its frequent involvement as a cause of SSI.⁴⁰ The gram-negative bacilli isolated from patients with SSI also demonstrate increased resistance.^{41,42} These resistant organisms likely result from prior exposure of the patient to the health care environment or broad spectrum antimicrobial therapy. The increasing resistance of gram-negative organisms causing SSI parallels their increasing resistance when they cause other nosocomial infections.⁴³

CONCLUSION

Surveillance systems aim to provide feedback to hospitals and stimulate infection control activities. An adequate method for risk adjustment is important for the comparison of hospital's specific rates. Risk indexes for surgical wound infection such as SENIC and NNIS were developed to provide comparative healthcare associated infection data within the hospitals that at least partially adjusted for patients' intrinsic and extrinsic risk factors. The SENIC risk index predicted SSI risk for all surgical patients better than stratifying by wound class alone. However, the SENIC index did not stratify risk by individual operative procedures. The NNIS surgical wound infection risk index is a modification of the SENIC risk index. Some inaccuracies associated with the SENIC index are corrected with NNIS index. Researchers in a number of countries have found that the NNIS risk index performed favorably for prediction of SSI. Not all experts concede that the NNIS risk index is the best method for the risk stratification of all surgical procedures. For example, several studies have shown that the NNIS risk index does not necessarily work well for patient undergoing cardiothoracic procedures. Future work should be directed to improvement of the sensitivity and the specificity of these indexes. Despite the insufficient follow-up parameters included in the NNIS index, authors have clearly demonstrated an increase of the actual rates of SSI using the NNIS index for surveillance. The healthcare worker-based (practices of an individual healthcare) or institution-based (practices of an entire hospital) factors may be other responsible causes for higher SSI rates. Scarcely when NNIS and SENIC is used together to predict the SSI they forecast the development of infection better. But there is a lot of other factors that affect the development of SSI, so for excellent surveillance risk index those factors known by everyone must be added to risk index scales.

ACKNOWLEDGEMENTS

The authors would like to thank the following people for their contribution to the manuscript: Mr. Sourabh kosey, Dr. H.P.S. Sandhu, Dr. Ashwani Kumar and Mr. Tarun Gautam for their guidance and valuable help.

Specially I would like to thank Sh. Parveen Garg, Chairman, ISF college of pharmacy, Moga for providing excellent research facilities.

CONFLICTS OF INTEREST

There is no conflict of interest by author on manuscript.

ABBREVIATION

ASA:	American Society of Anesthesiologists
CDC:	Centre of disease control and prevention
MRSA:	Methicillin-resistant <i>Staphylococcus aureus</i>

NNIS:	National Nosocomial infection Surveillance
SENIC:	Study of efficacy of nosocomial infection control and prevention
SSI:	Surgical site infection
SWI:	Surgical wound infection

REFERENCES

1. Mahesh CB, Shiva Kumar S, Suresh BS, Chidanand SP, Vishwanath Y. A prospective study of surgical site infections in a teaching hospital. *Journal of Clinical and Diagnostic Research* 2010 October; 4: 3114-9.
2. US. Department of Health and Human Services. Centers for Disease Control and Prevention, National Center for Health Statistics. *Vital and Health Statistics, Detailed Diagnoses and Procedures. National Hospital Discharge Survey* Hyattsville, Maryland: DHHS Publication; 1997.
3. Emori TG, Gaynes RP. An overview of nosocomial infections, including the role of the microbiology laboratory. *Clinmicrobiol Rev.* 1993; 6(4): 428-42.
4. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. The Hospital Infection Control Practices Advisory Committee. Guideline for prevention of surgical site infection. *Infect Control hospedemiol.* 1999; 27(2): 247-78.
5. Bratzler DW, Dellinger EP, Olsen KM. Clinical practice guidelines for antimicrobial prophylaxis in surgery. *Am J Health Syst Pharm.* 2013; 70(3): 195-283.
6. Haley RW, Culver DH, White JW. The efficacy of infection surveillance and control programs in preventing nosocomial infections in US hospitals. *Am J Epidemiol.* 1985; 121(1): 182-205.
7. Poulsen KB, Jepsen OB. Failure to detect a general reduction of surgical wound infections in Danish hospitals. *Dan Med Bull.* 1995; 42(5): 485-8.
8. Haley RW. The scientific basis for using surveillance and risk factor data to reduce nosocomial infections rates. *J Hosp Infect.* 1995; 30(1): 3-14.
9. Cardo DM, Falk PS, Mayhall CG. Validation of surgical wound surveillance. *Infect Control hospedemiol.* 1993; 14(04): 211-5.
10. Lee JT. Contemporary wound infection surveillance issues. *New Horizons* 1998; 6(Suppl): S20-9.
11. The Infection Control Standards Working Party. Standards in infection control in hospitals. Joint publication by the Association of Medical Microbiologists. Hospital Infection Society, Infection Control Nurses Association and the Public Health Laboratory Service. HMSO; 1993.
12. Roy MC, Peri TM. Basics of surgical-site infection surveillance. *Infect Control hospedemiol.* 1997; 18(09): 659-68.
13. Penin GB, Ehrenkranz NJ. Priorities for surveillance and cost-effective control of postoperative infection. *Arch Surg.* 1988; 123(11): 1305-8.
14. National Nosocomial Infections Surveillance System. Nosocomial infections rates for interhospital comparison: limitations and possible solutions. A report from the National Nosocomial Infections Surveillance (NNIS) System. *Infect Control hospedemiol.* 1991; 12(1): 609-21.
15. 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 hospedemiol.* 1992; 13(10): 606-8.
16. Klevens RM, Edwards JR, Richards CL Jr. Estimating health care associated infections and deaths in U.S. hospitals. *Pub Health Rep.* 2007; 122(2): 160-6.
17. Cruse PJ, Foord R. The epidemiology of wound infection. A 10-year prospective study of 62,939 wounds. *Surgclin North Am.* 1980; 60(1): 27-40.
18. Dipiro JT, Martindale RG, Bakst A. Infection in surgical patients: effects on mortality, hospitalization, and postdischarge care. *Am J Health Syst Pharm.* 1998; 55(8): 777-81.
19. Rosenthal VD, Richtmann R, Singh S. Surgical site infections, International Nosocomial Infection Control Consortium (INICC) report, data summary of 30 countries, 2005-2010. *Infect Control hospedemiol.* 2013; 34(6): 597-604.
20. Culver DH, Horan TC, Gaynes RP, Martone WJ, Jarvis WR. Surgical wound infection rates by wound class, operative procedure, and patient risk index. National Nosocomial Infections Surveillance System. *Am J Med.* 1991; 91(3): S152-7.
21. Ercole FF, Starling CEF, Chianca TCM, Carneiro M. Applicability of the National Nosocomial Infections Surveillance System Risk Index for the Prediction of Surgical Site Infections: A Review. *The Brazilian Journal of Infectious Diseases* 2007; 11(1): 134-41.
22. Culver DH, Horan TC, Gaynes RP. Surgical wound infection rates by wound class, operative procedure, and patient risk index. National Nosocomial Infections Surveillance System. *Am J Med.* 1991; 91(3B): 152S-7.
23. Mangram AJ, Horan TC, Pearson ML. Guideline for prevention of surgical site infection. *Infect Control hospedemiol.* 1999; 20(7): 250-78.
24. Nichols RL. Preventing surgical site infections: a surgeon's perspective. *Emerg Infect Dis.* 2001; 7(2): 220-4.
25. Anonymous. National Nosocomial Infections Surveillance (NNIS) report, data summary from October 1986-April 1996, issued May 1996. A report from the National Nosocomial Infections Surveillance (NNIS) System. *Am J Infect Control.* 1996; 24(1): 380-8.
26. Anderson DJ, Sexton DJ, Kanafani ZA. Severe surgical site infection in community hospitals: epidemiology, key procedures, and the changing prevalence of methicillin-resistant *Staphylococcus aureus*. *Infect Control hospedemiol.* 2007; 28(09): 1047-53.
27. Weiss CA, Statz CL, Dahms RA. Six years of surgical wound infection surveillance at a tertiary care center: review of the microbiologic and epidemiological aspects of 20,007 wounds. *Arch Surg* 1999; 134(10): 1041-8.
28. Cantlon CA, Stemper ME, Schwan W. Significant pathogens isolated from surgical site infections at a community hospital in the Midwest. *Am J Infect Control.* 2006; 34(8): 526-9.
29. L'Ecuyer PB, Murphy D, Little JR. The epidemiology of chest and leg wound infections following cardiothoracic surgery. *Clin Infect Dis.* 1996; 22(3): 424-9.
30. Itani KM, Wilson SE, Awad SS. Ertapenem versus cefotetan prophylaxis in elective colorectal surgery. *N Engl J Med.* 2006; 355(25): 2640-51.
31. Garcia Prado ME, Matia EC, Ciuro FP. Surgical site infection in liver transplant recipients: impact of the type of perioperative prophylaxis. *Transplantation* 2008; 85(12): 1849-54.
32. Jernigan JA. Is the burden of *Staphylococcus aureus* among patients with surgical-site infections growing? *Infect Control hospedemiol.* 2004; 25(06): 457-60.
33. Naylor AR, Hayes PD, Darke S. A prospective audit of complex wound and graft infections in Great Britain and Ireland: the emergence of MRSA. *Eur J vasc endovasc surg.* 2001; 21(4): 289-94.
34. Sharma M, Berriel-Cass D, Baran J Jr. Sternal surgical-site infection following coronary artery bypass graft: prevalence, microbiology, and complications during a 42-month period. *Infect Control hospedemiol.* 2004; 25(06): 468-71.
35. Kourbatova EV, Halvosa JS, King MD. Emergence of community-associated methicillin-resistant *Staphylococcus aureus* USA 300 clone as a cause of health care-associated infections among patients with prosthetic joint infections. *Am J Infect Control.* 2005; 33(7): 385-91.
36. Merrer J, Girou E, Lortat-Jacob A. Surgical site infection after surgery to repair femoral neck fracture: a French multicenter retrospective study. *Infect Control hospedemiol.* 2007; 28(10): 1169-74.
37. Zoumalan RA, Rosenberg DB. Methicillin-resistant *Staphylococcus aureus*-positive surgical site infections in face-lift surgery. *Arch Facial plastsurg* 2008; 10(2): 116-23.
38. Davis SA, Rybak MJ, Muhammad A. Characteristics of patients with healthcare-associated infection due to SCCmec Type IV methicillin-resistant *Staphylococcus aureus*. *Infect Control hospedemiol.* 2006; 27(10): 1025-31.
39. Popovich KJ, Weinstein RA, Hota B. Are community-associated methicillin-resistant *Staphylococcus aureus* (MRSA) strains replacing traditional nosocomial MRSA strains? *Clin Infect Dis.* 2008; 46(6): 787-94.

40. Manian FA, Griesnauer S. Community-associated methicillin-resistant *Staphylococcus aureus* (MRSA) is replacing traditional health care-associated MRSA strains in surgical-site infections among inpatients. *Clin Infect Dis*. 2008; 47(2): 434–5.
41. Gaynes R, Edwards JR. National Nosocomial Infections Surveillance System. Overview of nosocomial infections caused by gram-negative bacilli. *Clin Infect Dis*. 2005; 41(6): 848–54.
42. Kusachi S, Sumiyama Y, Arima Y. Isolated bacteria and drug susceptibility associated with the course of surgical site infections. *J Infect Chemother*. 2007; 13(3): 166–71.
43. Anonymous. National Nosocomial Infections Surveillance (NNIS) report, data summary from January 1992 through June 2004, issued October 2004. A report from the NNIS System. *Am J Infect Cont*. 2004; 32(8): 470–85.