NIH Public Access
Author Manuscript
Infect Control Hosp Epidemiol. Author manuscript; available in PMC 2013 April 06.

Published in final edited form as:
Infect Control Hosp Epidemiol. 2009 November ; 30(11): 1077–1083. doi:10.1086/606166.

Developing a Risk Stratification Model for Surgical Site Infection after Abdominal Hysterectomy

Margaret A. Olsen, PhD, MPH[1], **James Higham-Kessler, BA**[1], **Deborah S. Yokoe, MD**[2], **Anne M. Butler, MS**[1], **Johanna Vostok, BS**[3], **Kurt B. Stevenson, MD, MPH**[4], **Yosef Khan, MBBS, MPH**[4], **Victoria J. Fraser, MD**[1], and **the CDC Prevention Epicenter Program**

[1]Division of Infectious Diseases, Washington University School of Medicine, St. Louis, Missouri, St. Louis, MO

[2]Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA

[3]Harvard Pilgrim Health Care, Harvard Medical School, Boston, MA

[4]Department of Clinical Epidemiology, the Ohio State University Medical Center, and Division of Infectious Diseases, College of Medicine, The Ohio State University, Columbus, OH

Abstract

Objective—The incidence of surgical site infection (SSI) ranges widely from 2-21% after hysterectomy. There is insufficient understanding of risk factors to build a specific risk stratification index.

Methods—Retrospective case-control study of 545 abdominal and 275 vaginal hysterectomies from 7/1/03 - 6/30/05 at four institutions. SSIs were defined using CDC/NNIS criteria. Independent risk factors for abdominal hysterectomy were identified by logistic regression.

Results—There were 13 deep incisional, 53 superficial incisional, and 18 organ-space SSI after abdominal and 14 organ-space SSI after vaginal hysterectomy. Because risk factors for organ-space SSI were different in univariate analysis, further analyses focused on incisional SSI after abdominal hysterectomy. The maximum serum glucose within 5 days after operation was highest in patients with deep incisional SSI, lower in patients with superficial incisional SSI and lowest in uninfected patients (median 189, 156, and 141mg/dL, $p = .005$). Independent risk factors for incisional SSI included blood transfusion (odds ratio (OR) 2.4) and morbid obesity (body mass index (BMI) > 35, OR 5.7). Duration of operation > 75th percentile (OR 1.7), obesity (BMI 30-35, OR 3.0), and lack of private health insurance (OR 1.7) were marginally associated with increased odds of SSI.

Conclusions—Incisional SSI after abdominal hysterectomy was associated with increased BMI and blood transfusion. Longer operative time and lack of private health insurance were marginally associated with SSI. A specific risk stratification index could help to more accurately predict the risk of incisional SSI following abdominal hysterectomy.

Corresponding author: Margaret A. Olsen, PhD, MPH Campus Box 8051, 660 South Euclid Avenue Division of Infectious Diseases Washington University School of Medicine St. Louis, MO 63110-1093 Telephone: (314)-454-8320 Fax: (314)-454-5392 molsen@im.wustl.edu.

The following authors report no conflict of interest: MAO, JH-K, AMB, JV, KBS, YK, VJF. The following author received funding from Sage Products, Inc. for work on another study: DSY.

INTRODUCTION

More than 600,000 hysterectomies are performed annually in the U.S., with more than one-third of women having had the procedure by the age of 60.[1] In the 2000-2004 National Hospital Discharge Survey, abdominal hysterectomy accounted for about 2/3 of the procedures, and approximately 1/3 of the vaginal hysterectomies were performed laparoscopically.[1] The most common indications for hysterectomy were uterine leiomyoma, endometriosis, and uterine prolapse.[1]

The pooled rate of surgical site infection (SSI) reported by the National Healthcare Safety Network (NHSN) for 2006-7 after abdominal hysterectomy was 1.7%, and the rate after vaginal hysterectomy was 0.9%.[2] Rates of SSI after hysterectomy reported in individual studies range widely, depending on the approach and surgical method of hysterectomy (vaginal, abdominal, laparoscopic, etc.), the indication for operation, and use of antimicrobial prophylaxis. SSI rates also vary depending on the definitions used for surveillance and whether or not post-discharge surveillance was used to identify infections. Recently Reilly and colleagues reported that the SSI rate after abdominal hysterectomy doubled when patients completed questionnaires and interviews to identify wound signs and symptoms after hospital discharge compared to routine surveillance relying on only hospital data.[3]

Reported SSI rates tend to be higher for the abdominal approach compared to vaginal hysterectomy, and higher in the pre-prophylaxis era than more recently. Before routine use of prophylactic antibiotic regimens, SSI rates reported for abdominal hysterectomy were 9% or higher in all[4-8] but one publication.[9] During the last decade, reported SSI rates after abdominal hysterectomy have ranged from 1.7 to 11%,[10-17] while SSI rates reported in individual studies after vaginal hysterectomy (i.e., vaginal cuff cellulitis) ranged from 3.1 to 4.8%.[12,15,17] Thus there appears to be a wider range of SSI rates reported from individual institutions after abdominal hysterectomy than after vaginal hysterectomy.

Very few studies have determined risk factors for SSI after hysterectomy using standard definitions for SSI and multivariable analysis. Risk factors for SSI identified by multivariable analysis in previous studies include obesity,[7,17] lower serum albumin,[14] depth of subcutaneous tissue,[6] abdominal approach,[4,17] open vaginal cuff,[7] younger age,[4] non-private patient status,[4] and inadequate antimicrobial prophylaxis,[4,7,14,17] although only 3 used standard criteria to define SSI.[6,14,17] It is essential to identify independent risk factors in order to create a risk index specific to abdominal hysterectomy. The National Nosocomial Infection Surveillance (NNIS) risk index, most commonly used by hospital epidemiologists, performs better as a risk stratification method between different types of operations rather than within an individual type of operation.[18,19] A risk index tailored to abdominal hysterectomy would allow for more accurate comparison of SSI rates across institutions, which would help determine the reasons for the wide range of reported SSI rates in individual studies.

We performed a retrospective multi-hospital analysis of risk factors for SSI after abdominal hysterectomy as part of a multicenter surveillance study through the CDC Prevention Epicenter Program.

METHODS

Study population

We conducted a retrospective case-control study of women who had hysterectomy (ICD-9-CM procedure codes 68.39, 68.4, 68.6, 68.51, 68.59, and 68.7) performed at four participating CDC Prevention Epicenter hospitals from July 1, 2003 through June 30, 2005. Laparoscopically assisted abdominal hysterectomies (ICD-9-CM procedure code 68.31) were excluded. This study was done in concert with a CDC Prevention Epicenter project to determine the validity of enhanced surveillance based on ICD-9-CM diagnosis codes and antimicrobial utilization to identify inpatient SSI (Yokoe et al., manuscript in progress).[20] Approval for this study was obtained from all Institutional Review Boards at the participating centers.

Identification of Case and Control Patients

Initial case patients with SSI within 30 days after operation were identified at all participating hospitals by routine infection control surveillance. For hospitals with more than 200 procedures performed during the study period, 200 patients without SSI identified by routine surveillance were selected for comparison (by selecting every nth procedure, where "n" = total number of procedures/200, rounded to the nearest integer). Medical records for the original surgical admission and all subsequent hospital inpatient readmission(s) within 60 days after operation were reviewed for case and control patients. Signs, symptoms and potential risk factors for SSI were abstracted and entered directly into a Microsoft Access® database. Patients initially selected as controls based on routine surveillance who were subsequently determined to have an SSI based on CDC/NNIS definitions[21] were defined as case patients in the analyses. Any patients determined to be prisoners were excluded from evaluation at one institution.

Risk factor data

Information on potential risk factors collected from cases and controls included age at date of hysterectomy, weight, height, type of health insurance, current smoking, diabetes, congestive heart failure, indication for hysterectomy (ovarian, uterine, cervical, or other cancer, or not cancer related), preoperative glucose within 24 hours before incision, preoperative creatinine, postoperative glucose within 5 days after operation, postoperative creatinine (during operative admission), blood transfusion during or after operation (during operative admission), duration of operation, and type of operation (based on ICD-9-CM procedure codes).

Data analysis

De-identified data were analyzed using SPSS 14.0 (SPSS, Inc., Chicago, IL) and SAS v9.1 (SAS Institute Inc., Cary, NC). Comparisons for categorical variables were made using the Chi-square for trend test, and comparisons for continuous variables were made using the Student's T-test or Mann-Whitney U test, as appropriate. All variables with $p < 0.20$ in the univariate analysis or with *a priori* clinical significance were evaluated by stepwise logistic regression. Lack of private insurance (i.e., Medicaid, Medicare, or no health insurance) was forced into the logistic regression model as a proxy for socioeconomic status. Missing values for body mass index, glucose, and creatinine were imputed using multiple imputation with the SAS procedure PROC MI. Ten datasets were generated, each of which had an imputed value for the variables with missing values. The 10 datasets were analyzed using PROC LOGISTIC, and the results were combined for inference using PROC MIANALYZE.[22,23] After identification of the main effects, clinically relevant interactions between variables were tested for inclusion in the model, with $p < 0.05$ the criterion for inclusion. Model fit was assessed using the C statistic. All tests were two-tailed and $p < 0.05$ was considered significant.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

RESULTS

Eighty-four patients were identified with SSI following abdominal hysterectomy, 14 patients had organ-space SSI following vaginal hysterectomy (non-laparoscopic; 8 vaginal cuff cellulitis and 5 intra-abdominal infection) and 1 patient had organ-space SSI (intra-abdominal infection) following laparoscopic vaginal hysterectomy from the four participating hospitals. Fifty-three of the 84 SSI after abdominal hysterectomy were superficial incisional (63.1%), 13 (15.5%) were deep incisional, and 18 (21.4%) were classified as organ-space SSI (5 vaginal cuff cellulitis and 13 intra-abdominal infection). A total of 722 control patients without SSI were randomly selected for comparison to the SSI case patients (261 vaginal and 461 abdominal hysterectomy).

Table 1 shows the case mix of procedures by participating hospital. Hospitals C and D had much larger proportions of abdominal hysterectomies compared to Hospitals A and B. Over 80% of the hysterectomies at Hospital C were performed in patients with a diagnosis of cancer, while the proportion of operations performed for cancer at Hospitals A and B were much smaller (4% and 11%, respectively). Of 275 vaginal hysterectomies performed at the 4 hospitals, 57 (20.7%) were laparoscopically assisted.

Univariate associations between demographics, underlying comorbidities, and operative characteristics and organ-space SSI are shown in Table 2, while the comparisons with these factors and incisional SSI (superficial incisional and deep incisional) are shown in Table 3. Women with organ-space SSI were younger and more likely to smoke, and less likely to have private health insurance and operations for cancer than women without SSI (Table 2). Women with incisional SSI after abdominal hysterectomy had higher median body mass index and were more likely to have diabetes and perioperative blood transfusion than women without SSI after abdominal hysterectomy (Table 3). Women with incisional SSI after abdominal hysterectomy were also more likely to lack private health insurance.

In univariate analysis, the maximum perioperative serum glucose levels and creatinine levels were significantly higher in women with deep incisional SSI and superficial incisional SSI compared to women without SSI after abdominal hysterectomy (Table 3). Duration of operation was also significantly longer for women with deep incisional SSI and superficial incisional SSI compared to women without SSI after abdominal hysterectomy (Table 3).

Of the 64 women with incisional SSI and perioperative glucose measurements available for analysis, 4 had their maximum glucose measurement prior to operation. Of the remaining 60 women with a postoperative glucose available, 12 had postoperative serum glucose measurements that could have been obtained within two days before the diagnosis of incisional SSI. Of these 12 women, 7 had a maximum postoperative serum glucose measurement > 150 mg/dL within 5 days of the operation. For these 7 subjects, the date of maximum postoperative glucose measurement was available for 5; all of the maximum postoperative serum glucose values were obtained within 48 hours after operation and at least 2 days prior to the diagnosis of incisional SSI.

Independent risk factors for incisional SSI were identified by multivariable logistic regression. In order to include all subjects in the analysis, multiple imputation was used to create values for missing body mass index, serum glucose and creatinine. The risk factors retained in the final model are shown in Table 4. Independent risk factors included perioperative blood transfusion and morbid obesity (BMI > 35). Duration of operation greater than the 75th percentile, lack of private health insurance, and obesity (BMI 30-35) were marginally associated with increased risk of incisional SSI. In a preliminary multivariable model that excluded patients with missing glucose testing results, perioperative serum glucose > 180 mg/dL was marginally associated with increased risk of

NIH-PA Author Manuscript

NIH-PA Author Manuscript

incisional SSI ($p = .056$). In the final multivariable model after imputing missing values, perioperative serum glucose > 180 mg/dL was no longer associated with significantly increased odds of SSI ($p = .145$).

DISCUSSION

Identification of independent risk factors for SSI is essential in order to develop operation-specific risk stratification indices. In this multicenter study performed at four hospitals in the CDC Prevention Epicenter Program, we identified two independent risk factors for incisional SSI after abdominal hysterectomy, blood transfusion and morbid obesity. Duration of operation greater than the 75th percentile, obesity, and lack of private health insurance were marginally associated with increased odds of SSI in the multivariable analysis.

We determined independent risk factors for only abdominal incisional SSI in this study, since in univariate analysis the risk factors appeared to be different for organ-space SSI compared to incisional SSI. Crude risk factors for organ-space SSI included lack of private health insurance, current smoking, and younger age, while cancer as the indication for operation was associated with significantly lower risk of organ-space SSI. Since these risk factors were sufficiently different from the incisional SSI risk factors in subsequent analyses we focused on risk factors for superficial incisional and deep incisional SSI after abdominal hysterectomy.

Morbid obesity was associated with the greatest odds of incisional SSI, with a dose-response in risk as the BMI category increased. Obesity has been found to be a risk factor for SSI by many investigators, in particular after abdominal and gynecologic operations.[24] Obesity has also been shown to be independently associated with SSI after hysterectomy (vaginal or abdominal)[17] and specifically after abdominal hysterectomy.[6,7] In our current study receiving a blood transfusion was associated with significantly increased odds of incisional SSI. Persson and colleagues found blood loss of more than 1 liter during operation to be a risk factor for SSI[8], and Shapiro and colleagues also found blood loss to be associated with increased risk of SSI in univariate analysis.[25] Both of these studies included vaginal and abdominal hysterectomies, and neither included blood loss in a multivariable model to control for confounding factors. We and others have previously found excessive blood loss requiring transfusion to be associated with increased risk of SSI after a variety of operations,[26-29] although whether this association is due to the underlying anemia, transfusion-related immunomodulation,[30] or residual confounding is not clear.

Longer duration of operation and lack of private health insurance were marginally associated with increased odds of SSI after abdominal hysterectomy. Duration of operation greater than the 75th percentile is part of the standard NNIS risk index. It is not clear whether the risk associated with longer operations is truly due to the length of the operation, or if longer duration is at least in part a proxy for the complexity of the operation or operative skill.

We used lack of private health insurance as a proxy for low socioeconomic status in this study. Previously Shapiro et al. found the clinic service (in comparison to private service) to be an independent risk factor for SSI after vaginal or abdominal hysterectomy.[4] In general lower socioeconomic status is considered to be a risk factor for infection after gynecologic operations,[31] but the reasons for this association are not known.

In univariate analysis there was a trend towards increasing perioperative serum glucose in patients with superficial incisional SSI and deep incisional SSI, compared to women with no SSI after abdominal hysterectomy. We used a perioperative window of 24 hours before to 5 days after operation for assessment of maximum serum glucose measurements. It is unlikely that active infection could have explained the high glucose values, since the onset of incisional SSI occurred at least 2 days after the highest serum glucose measure in all but 2 cases (with missing dates of glucose measurement). Serum glucose did not remain as an independent risk factor in the multivariable model. In part this may be due to the relatively large number of patients without laboratory results for serum glucose in the perioperative period. We used multiple imputation to impute a set of plausible glucose values that represent the uncertainty about the correct value, but it is possible that higher glucose would have remained associated with increased risk of incisional SSI if serum glucose results had been available for all patients. Determination of the risk of incisional SSI associated with perioperative hyperglycemia will require more complete glucose testing of patients at risk for hyperglycemia (e.g., with obesity, family history of diabetes) before and after operation. Given the increasingly widespread epidemics of obesity and diabetes in the U.S., there are increasing numbers of hospitalized patients with undiagnosed and untreated diabetes.[32,33] Earlier and more accurate diagnosis of diabetes preoperatively is necessary. Additional studies to evaluate the relationship between perioperative glucose control and SSI and wound complications are needed.

Limitations of this study include the retrospective observational nature of the study, which precluded collecting some potential risk factors for SSI (e.g., adequacy of preoperative skin antisepsis, operative hemostasis, etc.). Also, the collection of data from four hospitals necessitated restricting data collection to a relatively small number of risk factors, in order to ensure as complete and accurate data collection as possible. In addition, the surveillance strategy we used excluded SSI diagnosed and treated solely in outpatient settings. Since we only reviewed hospital records it is possible that some individuals classified as control uninfected patients had SSI, resulting in misclassification of the outcome, potentially resulting in bias of results towards the null.

Advantages of this study included the multicenter nature, with collection of data from patients admitted for hysterectomy to four academic hospitals. Inclusion of data from a variety of different hospital types, including hospitals with different patient populations and different indications for hysterectomy expands the generalizability of the results to other academic medical centers. In addition we used standardized definitions for SSI and enhanced surveillance to identify infections during the operative admission and readmission to the hospital. We focused analysis on risk factors only for incisional SSI after abdominal hysterectomy, since the risk factors associated with organ-space SSI appear to differ from risk factors for incisional SSI after hysterectomy.

In summary, we identified morbid obesity and perioperative blood transfusion as independent risk factors for superficial incisional and deep incisional SSI after abdominal hysterectomy. Obesity, longer duration of operation, and lack of private health insurance were marginally associated with increased odds of incisional SSI. Additional studies are needed to determine the association of perioperative hyperglycemia with SSI after abdominal hysterectomy. The risk factors identified in this study can be used in the future to create a risk stratification index specific for abdominal hysterectomy and incisional SSI.

Acknowledgments

We gratefully acknowledge Marian Mamayek, Susan Marino, and Cherie Hill for assistance with data collection and the databases. This study was supported by funding from the CDC/NIH Prevention Epicenter Program (5U01CI000333 to Washington University PE, 5U01CI000344 to the Eastern Massachusetts PE, 5U01CI000328 to The Ohio State University PE). Drs. Olsen and Fraser were partially supported by NIH Career Development Awards (K01AI065808 (Olsen) and K24AI067794 (Fraser)). Mr. Higham-Kessler was supported by a WUSM training grant (T35HL007815).

References

1. Whiteman MK, Hillis SD, Jamieson DJ, Morrow B, Podgornik MN, Brett KM, et al. Inpatient hysterectomy surveillance in the United States, 2000-2004. Am J Obstet Gynecol. 2008; 198:34–37. [PubMed: 17981254]
2. Edwards JR, Peterson KD, Andrus ML, Dudeck MA, Pollock DA, Horan TC. National Healthcare Safety Network (NHSN) Report, data summary for 2006 through 2007, issued November 2008. Am J Infect Control. 2008; 36:609–26. [PubMed: 18992647]
3. Reilly J, Allardice G, Bruce J, Hill R, McCoubrey J. Procedure-specific surgical site infection rates and postdischarge surveillance in Scotland. Infect Control Hosp Epidemiol. 2006; 27:1318–23. [PubMed: 17152029]
4. Shapiro M, Munoz A, Tager IB, Schoenbaum SC, Polk BF. Risk factors for infection at the operative site after abdominal or vaginal hysterectomy. N Engl J Med. 1982; 307:1661–66. [PubMed: 6755254]
5. Kandula PV, Wenzel RP. Postoperative wound infection after total abdominal hysterectomy: a controlled study of the increased duration of hospital stay and trends in postoperative wound infection. Am J Infect Control. 1993; 21:201–4. [PubMed: 8239050]
6. Soper DE, Bump RC, Hurt WG. Wound infection after abdominal hysterectomy: effect of the depth of subcutaneous tissue. Am J Obstet Gynecol. 1995; 173:465–69. [PubMed: 7645622]
7. Korn AP, Grullon K, Hessol N, Lin P, Siopak J. Does vaginal cuff closure decrease the infectious morbidity associated with abdominal hysterectomy? J Am Coll Surg. 1997; 185:404–7. [PubMed: 9328390]
8. Persson E, Bergstrom M, Larsson PG, Moberg P, Platz-Christensen JJ, Schedvins K, et al. Infections after hysterectomy. A prospective nation-wide Swedish study. The Study Group on Infectious Diseases in Obstetrics and Gynecology within the Swedish Society of Obstetrics and Gynecology. Acta Obstet Gynecol Scand. 1996; 75:757–61. [PubMed: 8906013]
9. Taylor G, Herrick T, Mah M. Wound infections after hysterectomy: opportunities for practice improvement. Am J Infect Control. 1998; 26:254–57. [PubMed: 9638288]
10. Ahmed F, Wasti S. Infectious complications following abdominal hysterectomy in Karachi, Pakistan. Int J Gynaecol Obstet. 2001; 73:27–34. [PubMed: 11336718]
11. Chongsomchai C, Lumbiganon P, Thinkhamrop J, Ounchai J, Vudhikamraksa N. Placebo-controlled, double-blind, randomized study of prophylactic antibiotics in elective abdominal hysterectomy. J Hosp Infect. 2002; 52:302–6. [PubMed: 12473477]
12. Davies A, Hart R, Magos A, Hadad E, Morris R. Hysterectomy: surgical route and complications. Eur J Obstet Gynecol Reprod Biol. 2002; 104:148–51. [PubMed: 12206928]
13. Eason E, Wells G, Garber G, Hemmings R, Luskey G, Gillett P, et al. Antisepsis for abdominal hysterectomy: a randomised controlled trial of povidone-iodine gel. BJOG. 2004; 111:695–99. [PubMed: 15198760]
14. Rodriguez JF, Trobo AR, Garcia MV, Martinez MJ, Millan CP, Vazquez MC, et al. The effect of performance feedback on wound infection rate in abdominal hysterectomy. Am J Infect Control. 2006; 34:182–87. [PubMed: 16679174]
15. Leung PL, Tsang SW, Yuen PM. An audit on hysterectomy for benign diseases in public hospitals in Hong Kong. Hong Kong Med J. 2007; 13:187–93. [PubMed: 17548906]
16. Anderson DJ, Chen LF, Sexton DJ, Kaye KS. Complex surgical site infections and the devilish details of risk adjustment: important implications for public reporting. Infect Control Hosp Epidemiol. 2008; 29:941–46. [PubMed: 18808342]
17. Molina-Cabrillana J, Valle-Morales L, Hernandez-Vera J, Lopez-Carrio I, Garcia-Hernandez JA, Bolanos-Rivero M. Surveillance and risk factors on hysterectomy wound infection rate in Gran Canaria, Spain. Eur J Obstet Gynecol Reprod Biol. 2008; 136:232–38. [PubMed: 17337107]
18. Gaynes RP. Surgical-site infections and the NNIS SSI Risk Index: room for improvement. Infect Control Hosp Epidemiol. 2000; 21:184–85. [PubMed: 10738986]
19. Geubbels EL, Grobbee DE, Vandenbroucke-Grauls CM, Wille JC, de Boer AS. Improved risk adjustment for comparison of surgical site infection rates. Infect Control Hosp Epidemiol. 2006; 27:1330–39. [PubMed: 17152031]

20. Yokoe DS, Noskin GA, Cunningham SM, Zuccotti G, Plaskett T, Fraser VJ, et al. Enhanced identification of postoperative infections among inpatients. Emerg Inf Dis. 2004; 10:1924–30.
21. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for the prevention of surgical site infection, 1999. Infect Control Hosp Epidemiol. 1999; 20:247–78.
22. Allison, PD. Missing Data. Sage Publications, Inc.; Thousand Oaks, CA: 2001.
23. SAS Institute, Inc.. SAS OnlineDoc, Version 8. SAS Institute, Inc.; 2001. http://support.sas.com/documentation/onlinedoc/v82/whatsnew/zenid-96.htm
24. Thomas EJ, Goldman L, Mangione CM, Marcantonio ER, Cook EF, Ludwig L, et al. Body mass index as a correlate of postoperative complications and resource utilization. Am J Med. 1997; 102:277–83. [PubMed: 9217597]
25. Anteby EY, Yagel S, Hanoch J, Shapiro M, Moses AE. Puerperal and intrapartum group A streptococcal infection. Infect Dis Obstet Gynecol. 1999; 7:276–82. [PubMed: 10598916]
26. Olsen MA, Lock-Buckley P, Hopkins D, Polish LB, Sundt TM, Fraser VJ. The risk factors for deep and superficial chest surgical-site infections after coronary artery bypass graft surgery are different. J Thorac Cardiovasc Surg. 2002; 123:136–45. [PubMed: 12091819]
27. Olsen MA, Lefta M, Dietz JR, Brandt KE, Aft R, Matthews R, et al. Risk factors for surgical site infection after major breast operation. J Am Coll Surg. 2008; 207:326–35. [PubMed: 18722936]
28. Walz JM, Paterson CA, Seligowski JM, Heard SO. Surgical site infection following bowel surgery: a retrospective analysis of 1446 patients. Arch Surg. 2006; 141:1014–18. [PubMed: 17043280]
29. Banbury MK, Brizzio ME, Rajeswaran J, Lytle BW, Blackstone EH. Transfusion increases the risk of postoperative infection after cardiovascular surgery. J Am Coll Surg. 2006; 202:131–38. [PubMed: 16377506]
30. Vamvakas EC, Blajchman MA. Transfusion-related immunomodulation (TRIM): an update. Blood Rev. 2007; 21:327–48. [PubMed: 17804128]
31. Hager, DW.; Larsen, JW. Postoperative infections: prevention and management.. In: Rock, JA.; Jones, HW., III, editors. Te Linde's Operative Gynecology. 10th ed.. Lippincott Williams & Wilkins; Philadelphia, PA: 2008. p. 190-202.
32. Wexler DJ, Nathan DM, Grant RW, Regan S, Van Leuvan AL, Cagliero E. Prevalence of elevated hemoglobin A1c among patients admitted to the hospital without a diagnosis of diabetes. J Clin Endocrinol Metab. 2008; 93:4238–44. [PubMed: 18697862]
33. Charfen MA, Ipp E, Kaji AH, Saleh T, Qazi MF, Lewis RJ. Detection of Undiagnosed Diabetes and Prediabetic States in High-risk Emergency Department Patients. Acad Emerg Med. 2009; 16:394–402. [PubMed: 19302369]

Table 1

Characteristics of Operations and Patients at Participating Hospitals

Hospital	Total No. of Hysterectomies Included in Study	No. of Abdominal Procedures (%)	No. of Cancer-Related Procedures (%)	No. without Private Health Insurance (%)
A	198	80 (40.4)	8 (4.0)	19 (9.6)
B	161	42 (26.1)	18(11.2)	23 (14.3)
C	198	183 (92.4)	160 (80.8)	81 (40.9)
D	263	240 (91.3)	115 (43.7)	57 (21.7)
Total	820	545 (66.5)	301 (36.7)	180 (22.0)

Table 2

Risk Factors for Organ-Space SSI after Hysterectomy

Risk Factor	SSI Case Patients n= 32 (%)	Control Uninfected Patients n = 722 (%)	*p*
Insurance status			
Private or Medicare	22 (68.8)	641 (88.8)	
None	4 (12.5)	24 (3.3)	.007
Medicaid	6 (18.8)	57 (7.9)	.020
Current smoking	14 (43.8)	106 (14.7)	<.001
Congestive heart failure	2 (6.3)	11 (15)	.102
Diabetes	2 (6.3)	82 (11.4)	.566
Cancer	6 (18.8)	258 (35.7)	.049
Perioperative blood transfusion	7 (21.9)	25 (78.1)	.301
Vaginal hysterectomy	14 (43.8)	261 (36.1)	.382
Continuous Risk Factors	**SSI Case Patients Median (Range)**	**Control Uninfected Patients Median (Range)**	***p***
Age (years)	45 (33-72)	52 (20-92)	.001
Body Mass Index[a]	26.5 (20.5-54.5)	27.5 (15.8-67.3)	.228
Duration of operation (minutes)[b]	147 (44-369)	146 (33-476)	.769
Creatinine, mg/dL (highest during operative admission)[c]	0.7 (0.3-6.7)	0.7 (0.3-5.1)	.598
Glucose, mg/dL (highest from 24 hours before to 5 days after operation)[d]	129 (80-315)	140 (81-500)	.690

[a] Weight in kg/(height in m^2). 56 control patients missing values for weight and/or height

[b] 1 control patient had missing values for operation start and stop times.

[c] 9 case patients and 281 control patients were missing data for creatinine in the operative admission.

[d] 23 case patients and 435 control patients were missing data for serum glucose in the operative admission.

NIH-PA Author Manuscript
NIH-PA Author Manuscript

Table 3

Risk Factors for Incisional SSI after Abdominal Hysterectomy

Risk Factor	**Deep Incisional SSI Case Patients n= 13 (%)**	**Superficial Incisional Case Patients n = 53 (%)**	**Control Uninfected Patients n = 461 (%)**	***p*** [a]
Insurance status				
Private or Medicare	9 (69.2)	37 (69.8)	402 (87.2)	
None	2 (l5.4)	5 (9.4)	17 (3.7)	.002
Medicaid	2 (l5.4)	11 (20.8)	42 (9.1)	
Current smoking	4 (30.8)	11 (20.8)	72 (15.6)	.097
Congestive heart failure	0	3 (4.5)	10 (2.2)	.483
Diabetes	6 (46.2)	14 (26.4)	67 (14.5)	<.001
Cancer	8 (61.5)	29 (54.7)	232 (50.3)	.333
Perioperative blood transfusion	5 (38.5)	22 (41.5)	89 (19.3)	<.001
Continuous Risk Factors	**Deep Incisional SSI Case Patients**	**Superficial Incisional Case Patients**	**Control Uninfected Patients**	***p*** [b]
Age (median, range)	61 (29-80)	50 (34-88)	51 (20-92)	.254
Body Mass Index[c] – median (range)	33.3 (25.1-64.8)	37.6 (15.7-68.2)	29.7 (15.8-67.3)	<.001
Duration of operation in minutes - mean (SD)	216.6 (73.1)	190.6 (75.1)	154.4 (64.9)	<.001[d]
Creatinine, mg/dL - median (range)	0.93 (0.7-1.7)	0.80 (0.5-3.1)	0.71 (0.3-5.1)	.001[e]
Glucose, mg/dL - median (range)	189 (103-399)	156 (108-500)	141 (82-500)	.005[f]

[a]Chi-square for trend.

[b]Kruskal-Wallis test

[c]Weight in kg/(height in m^2). 2 case patients and 24 control patients missing values for weight and/or height.

[d]One-way ANOVA.

[e]92 patients were missing values for serum creatinine.

[f]94 patients were missing values for serum glucose.

Table 4

Independent Risk Factors for Incisional SSI After Abdominal Hysterectomy

Risk Factor	Adjusted OR (95% Confidence Interval)	*p*
Perioperative blood transfusion	2.4 (1.4,4.4)	.003
Duration of operation > 75%	1.7 (1.0,3.0)	.074
Medicaid or no health insurance	1.7 (0.9,2.9)	.076
Body Mass Index		
< 25	Reference	
25-30	2.4 (0.8,7.2)	.104
30-35	3.0 (1.0,9.6)	.058
> 35	5.7 (2.1,15.6)	.001

Model C-statistic = 0.729