ORIGINAL ARTICLE

Risk Factors for Surgical Site Infections Following Neurosurgical Spinal Fusion Operations: A Case Control Study

Thomas L. Walsh, MD;[1] Ashley M. Querry, BS, CIC;[2] Sheila McCool, MPH, BSN;[2] Alison L. Galdys, MD;[1,2] Kathleen A. Shutt, MS;[1,3] Melissa I. Saul, MS;[4] Carlene A. Muto, MD, MS[1,2,3]

OBJECTIVE. To determine risk factors for the development of surgical site infections (SSIs) in neurosurgery patients undergoing spinal fusion.

DESIGN. Retrospective case-control study.

SETTING. Large, academic, quaternary care center.

PATIENTS. The study population included all neurosurgery patients who underwent spinal fusion between August 1, 2009, and August 31, 2013. Cases were defined as patients in the study cohort who developed an SSI. Controls were patients in the study cohort who did not develop an SSI.

METHODS. To achieve 80% power with an ability to detect an odds ratio (OR) of 2, we performed an unmatched case-control study with equal numbers of cases and controls.

RESULTS. During the study period, 5,473 spinal fusion procedures were performed by neurosurgeons in our hospital. With 161 SSIs recorded during the study period, the incidence of SSIs associated with these procedures was 2.94%. While anterior surgical approach was found to be a protective factor (OR, 0.20; 95% confidence interval [CI], 0.08–0.52), duration of procedure (OR, 1.58; 95% CI, 1.29–1.93), American Society of Anesthesiologists score of 3 or 4 (OR, 1.79; 95% CI, 1.00–3.18), and hospitalization within the prior 30 days (OR, 5.8; 95% CI, 1.37–24.57) were found in multivariate analysis to be independent predictors of SSI following spinal fusion. Prior methicillin-resistant *Staphylococcus aureus* (MRSA) nares colonization was highly associated with odds 20 times higher of SSI following spinal fusion (OR, 20.30; 95% CI, 4.64–8.78).

CONCLUSIONS. In additional to nonmodifiable risk factors, prior colonization with MRSA is a modifiable risk factor very strongly associated with development of SSI following spinal fusion.

Infect Control Hosp Epidemiol 2017;38:340–347

Approximately 45 million operative procedures are performed in the United States annually,[1] and nearly 500,000 result in a surgical site infection (SSI).[2] SSIs comprise roughly one-third of all hospital-acquired infections.[1–5] SSIs represent the most common healthcare-associated infection in surgical patients.[6] Patients who develop SSIs suffer prolonged hospitalizations, significant morbidity, increased mortality, and increased healthcare costs.[7] The annual costs of increased hospitalizations alone due to SSIs exceed $1.6 billion.[7,8]

Numerous studies have outlined general risk factors for SSIs, and both the medical condition of the patient and the complexity of the procedure have been found to be contributing factors. Among published literature of SSIs complicating spine surgery, numerous risk factors have been elucidated,[9–34] but infection surveillance methodology, definition of infection, patient population evaluated, and risk factors analyzed vary greatly among studies. SSI incidence and specific risk factors revealed also vary depending upon the specific spine procedures included in the analysis. Established patient-related risk factors for spine surgery SSIs have included advanced age,[9] obesity,[9–17] diabetes mellitus (DM),[9,16,18–26] preoperative hyperglycemia,[18] postoperative hyperglycemia,[18] preoperative anemia,[19] malnutrition,[28] tobacco use,[19,27,29,30] chronic obstructive pulmonary disease,[11] coronary artery disease,[11] osteoporosis,[11] immunosuppression,[9] American Society of Anesthesiologists (ASA) class of ≥3,[10,12,17,19,31] disseminated malignancy,[19] postoperative incontinence,[12] and previous spine surgery.[27] Additionally, a wide variety of procedure-related risk factors have also been reported and include posterior surgical approach,[12,17] multilevel procedure,[10,27] increased operative

Affiliations: 1. Division of Infectious Diseases, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania; 2. Department of Infection Control and Hospital Epidemiology, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania; 3. Infectious Diseases Epidemiology Research Unit, University of Pittsburgh School of Medicine and Graduate School of Public Health, Pittsburgh, Pennsylvania; 4. Department of Medicine, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania.

Received August 17, 2016; accepted November 22, 2016; electronically published December 19, 2016

© 2016 by The Society for Healthcare Epidemiology of America. All rights reserved. 0899-823X/2017/3803-0013. DOI: 10.1017/ice.2016.307

https://doi.org/10.1017/ice.2016.307 Published online by Cambridge University Press

complexity,[30,32,33] large-volume blood loss,[11,16,26] surgical arthrodesis involving the sacrum,[17] presence of dural tear,[11] prolonged duration of the procedure,[9,16,17,19,26] prolonged use of closed suction drains,[10] increased operating room traffic,[9,11] operation for tumor resection,[12] and intraoperative fraction of inspired oxygen of <50%.[17]

However, few high-quality studies have specifically addressed SSIs complicating neurosurgical spinal fusion surgical procedures that may have unique risk factors; many of the studies that detected these risk factors were limited by their small sample size, which hampers the ability to perform multivariate analyses to determine independent risk factors for SSIs. Most of these analyses also only included a very small portion of all potential risk factors for SSIs in their respective studies. To properly identify and evaluate independent risk factors, studies need to be performed that include large numbers of patients with a uniform, standard, accepted definition of SSI as well as he examination of an extensive list of potential risk factors. Furthermore, such studies should control for the existence of multiple risk factors within individual patients by using multivariate analyses.

We performed a retrospective case-control study to determine independent risk factors for the development of SSIs in neurosurgery patients undergoing spinal fusion. We hypothesized that in addition to known nonmodifiable risk factors, modifiable risk factors are also associated with an increased risk of developing an SSI.

METHODS

Study Setting

This study was conducted at the University of Pittsburgh Medical Center (UPMC) Presbyterian-Shadyside, a 792-bed quaternary care teaching hospital affiliated with the University of Pittsburgh. The evaluation was peer-reviewed and approved by the UPMC Total Quality Council.

Study Design

We performed a case-control study to assess risk factors for SSI. The study population consisted of all patients who underwent spinal fusion between August 1, 2009, and August 31, 2013. All patients who had undergone a spinal fusion were identified by querying the surgical case report logs found in the hospital's medical record data repository.[35] Eligible operations were restricted to those performed by a neurosurgeon at our institution on patients ≥18 years of age (n = 5,473). Spinal fusions are primarily performed by 5 neurosurgeons at our institution. Other operations performed by neurosurgeons were excluded. Spinal fusions performed by orthopedic surgeons were excluded.

Cases were defined as patients in our study cohort who developed an SSI. For the control arm, the remainder of the study population who did not develop an SSI was available for selection. Noninfected control patients were randomly selected from all patients who underwent neurosurgical spinal fusion surgery during the study period. In cases in which a patient developed >1 SSI following neurosurgical spinal fusion surgery, only the first SSI was included in the cohort, and any subsequent spinal fusion surgeries were excluded from the analysis.

Identification of Surgical Site Infections

Patients in whom SSI occurred following spinal fusion surgery were identified prospectively by UPMC's Infection Prevention and Control Department based upon standard National Healthcare Safety Network (NHSN) criteria.[36] The medical records of patients with indicators of potential SSI during the hospitalization for the initial surgery or at the time of readmission to the hospital after the operation were reviewed for recorded signs and symptoms of surgical site infection. All microbiology, pathology, radiology, and operative reports were reviewed to determine whether the NHSN definition for an SSI was met. We included patients with superficial incisional SSIs, deep incisional SSIs, and organ-space SSIs according to the NHSN definitions during the study period.[36]

Data Collection

All data, including demographics, comorbid conditions, perioperative data including laboratory results, procedural factors, postoperative data, and postoperative infection data including microbiology, radiology, and documentation by neurosurgeons and infectious disease physicians were collected from the electronic medical records by 1 investigator (T.L.W.), who was not involved in the initial treatment or SSI designation, utilizing a standard data collection tool. Additionally, the hospital data repository was used to verify the type of operative procedure performed, attending surgeon, approach, spinal level, number of vertebral bodies fused, ASA score, intrinsic wound contamination level, estimated blood loss, presence of implant or graft, duration of procedure from the operative report and surgical case log, and length of preoperative hospital stay. The use of preoperative chlorhexidine baths, preoperative *S. aureus* nares screen, prior *S. aureus* known nares colonization, and use of perioperative nasal mupirocin were obtained from the patients' medical records. Potential risk factors for surgical site infection included a wide variety of demographic, comorbid, preoperative, operative, and postoperative variables, derived from an extensive review of the literature.

Data Analysis

To achieve 80% power with an ability to detect an odds ratio (OR) of 2, we performed an unmatched case-control analysis with equal numbers of cases and controls. Associations between SSI and potential risk factors were assessed using univariate logistic regression and calculation of an OR and

https://doi.org/10.1017/ice.2016.307 Published online by Cambridge University Press

95% confidence intervals. $P < .05$ was considered statistically significant in all statistical tests. Multivariate logistic regression was used to identify independent risk factors for SSIs. Variables eligible for inclusion in the multivariate model included those with $P < .20$ in the univariate analysis. All analyses were performed using SAS 9.3 software (SAS Institute, Cary, NC).

RESULTS

During the study period, 5,473 spinal fusion procedures were performed by neurosurgeons in our hospital. During this period, 163 SSIs occurred; however, 2 infections were excluded because the patients developed >1 SSI following spinal fusion surgery. Because only the first SSI for each patient was included in the cohort, the remaining 161 SSIs were included as part of the analysis. The overall spinal fusion SSI rate was 2.94%; among them, 34 (21.1%) were classified as superficial incisional, 114 (70.8%) were deep incisional, and 13 (8.1%) were organ space. At least 1 pathogen was identified in 155 of the 161 cases (96.3%); >1 pathogen was identified in 36 cases (22.4%), and >2 pathogens were identified in 8 cases (5.0%). Aerobic gram-positive organisms accounted for 122 (75.8%) infections, with *S. aureus* identified in 82 of the 161 cases (50.9%) (Table 1). Of the 82 *S. aureus* infections, 28 were due to methicillin-resistant *S. aureus* (MRSA). Aerobic gram-negative organisms were identified in 34 cases (21.1%), while anaerobic organisms were isolated in 11 cases (6.8%).

Univariate risk factor analysis is shown in Table 2. The patient-specific factors that were found to be associated with a significantly increased risk of SSI in the univariate analysis included chronic obstructive pulmonary disease, asthma, chronic renal insufficiency, male sex, white race, and previous MRSA carriage. In total, 98 patients (30.6%) were screened for MRSA carriage prior to spinal fusion, while 222 (69.4%) did not receive screening prior to surgery. Of those 98, 40 (40.8%) who received prior screening were found to be MRSA colonized prior to spinal fusion. More severe illness, as indicated by an ASA score of 3 or 4, was associated with an increased risk of SSI. Obesity, defined as a body mass index (BMI) of 30–34.9 kg/m^2, was not associated with an increased risk of SSI. Neither morbid obesity (BMI, 35–39.9 kg/m^2) nor extreme obesity (BMI, ≥40 kg/m^2) was associated with an increased risk of SSI.

Hospitalization in the 90 days prior to spinal fusion procedure was associated with a significantly increased risk of development of an SSI, whereas preoperative hospitalization of ≥3 days was not associated with an increased risk. There was no association of increased SSI risk with preoperative glucose level of >125 mg/dL and hair removed via clipping compared to no hair removal.

Significant procedural risk factors on univariate analysis included procedures lasting >4 hours, procedures where ≥3 vertebral bodies were fused, and procedures where a drain was placed intraoperatively. Additionally, procedures performed by neurosurgeons B, C, and D were associated with an increased SSI risk compared with surgeon A. Surgeries performed on the cervical spine were associated with a reduced risk of SSI. Utilizing an anterior surgical approach was found to be a protective factor.

TABLE 1. Causative Pathogens in Cases of Surgical Site Infections Following Neurosurgical Spinal Fusion Procedure

Pathogen	Patients, No. (%)[a] (n = 161)
Aerobic gram-positive organisms	122 (75.8)
Staphylococcus aureus	82 (50.9)
MSSA	54 (33.5)
MRSA	28 (17.4)
Coagulase-negative staphylococcus	33 (20.5)
Enterococcus spp.	13 (8.1)
Diphtheroids	3 (1.9)
Viridans group streptococcus	1 (0.6)
Group B streptococcus	1 (0.6)
Aerobic gram-negative organisms	34 (21.1)
Escherichia coli	12 (7.5)
Proteus mirabilis	8 (5.0)
Pseudomonas aeruginosa	5 (3.1)
Enterobacter cloace	3 (1.9)
Klebsiella pneumoniae	2 (1.2)
Morganella morganii	2 (1.2)
Serratia marcescens	2 (1.2)
Citrobacter spp.	1 (0.6)
Stenotrophomonas maltophila	1 (0.6)
Anaerobic organisms	11 (6.8)
Peptostreptococcus spp.	4 (2.5)
Proprionibacterium acnes	3 (1.9)
Bacteroides fragilis	2 (1.2)
Prevotella spp.	2 (1.2)
Yeast	5 (3.1)
Candida spp.	5 (3.1)

NOTE. MSSA, methicillin-susceptible *Staphylococcus aureus;* MRSA, methicillin-resistant *Staphylococcus aureus.*
[a] >1 pathogen was identified in 36 patients, so the percentages total >100.

Postoperative factors that were associated with an increased risk of developing an SSI included postoperative hematocrit ≥36%, receipt of ≥2 units of packed red blood cells intraoperatively or postoperatively, and receipt of other blood products. The use of a silver-impregnated dressing initially postoperatively did not meet statistical significance (OR, 1.56; 95% CI, 0.99–2.45; $P = .055$). A postoperative glucose level of ≥200 mg/dL was not a risk factor for developing an SSI.

Table 3 shows the results of a multivariate logistic regression analysis to assess independent risk factors when adjusting for all other potential risk factors. In the final model, previous MRSA carriage ($P < .0001$), hospitalization in the 30 days prior to fusion procedure ($P = .017$), ASA score of 3 or 4 ($P = .048$), duration of operation (per hour) ($P < .0001$), and white race ($P = .018$) were independently associated with developing an SSI following neurosurgical spinal fusion. Utilization an anterior surgical approach ($P = .001$) was found to be a protective factor. Previous MRSA carriage (OR, 20.30; 95% CI, 4.64–88.78) was the

https://doi.org/10.1017/ice.2016.307 Published online by Cambridge University Press

TABLE 2. Univariate Analysis of Patient, Perioperative, Procedural, and Postoperative Risk Factors for Development of Surgical Site Infections following Neurosurgical Spinal Fusion

Variable	Case, No. (%) (n = 159)	Control, No. (%) (n = 161)	OR	95% CI	*P* Value
Demographics					
Sex, male	70 (44.0)	89 (55.3)	0.64	(0.41–0.99)	.045
Race, white	154 (96.9)	144 (89.4)	3.64	(1.31–10.11)	.013
Age					
<50 y	43 (27.0)	52 (32.3)	…	Baseline	.589
50–64 y	67 (42.1)	63 (39.1)	1.28	(0.75–2.18)	
65–79 y	40 (25.2)	41 (25.5)	1.18	(0.65–2.13)	
≥80 y	9 (5.7)	5 (3.1)	2.08	(0.65–6.65)	
Patient-specific factors					
Body mass index					
<25 kg/m^2	31 (21.4)	34 (21.4)	…	Baseline	.944
25–29.9 kg/m^2	42 (29.0)	48 (30.2)	0.96	(0.51–1.82)	
30–34.9 kg/m^2	33 (22.8)	39 (24.5)	0.93	(0.47–1.82)	
35–39.9 kg/m^2	24 (16.6)	26 (16.4)	1.01	(0.48–2.12)	
≥40 kg/m^2	15 (10.3)	12 (7.5)	1.36	(0.55–3.34)	
Active malignancy	20 (12.6)	24 (14.9)	0.82	(0.43–1.56)	.546
Active non-skin malignancy	18 (11.3)	17 (10.6)	1.08	(0.54–2.18)	.827
Congestive heart failure	17 (10.7)	8 (5.0)	2.29	(0.96–5.47)	.062
Chronic lung disease	48 (30.2)	27 (16.8)	2.15	(1.26–3.66)	.005
COPD	26 (16.4)	13 (8.1)	2.23	(1.10–4.51)	.026
Asthma	28 (17.6)	14 (8.7)	2.24	(1.13–4.44)	.020
Chronic renal insufficiency	12 (7.5)	4 (2.5)	3.20	(1.01–10.16)	.048
Immunosuppressed state	16 (10.1)	8 (5.0)	2.14	(0.89–5.15)	.090
Diabetes mellitus	50 (31.4)	37 (23.0)	1.54	(0.94–2.53)	.090
Obesity	72 (49.7)	77 (48.4)	1.05	(0.67–1.65)	.831
Tobacco use	55 (34.6)	48 (29.8)	1.24	(0.78–1.99)	.361
Daily alcohol use	19 (12.0)	18 (11.2)	1.09	(0.55–2.16)	.814
Current use of chronic steroids or other immunosuppressive agent	14 (8.8)	8 (5.0)	1.85	(0.75–4.53)	.181
Previous MRSA carriage	38 (23.9)	2 (1.2)	25.0	(5.91–105.53)	<.0001
ASA score 3 or 4	125 (78.6)	91 (56.5)	2.83	(1.73–4.62)	<.0001
Perioperative factors					
Preoperative hospital stay ≥3 d	23 (14.5)	16 (9.9)	1.53	(0.78–3.02)	.218
Hospitalization at UPMC facility within 30 d prior to procedure	11 (6.9)	4 (2.5)	2.92	(0.91–9.36)	.072
Hospitalization at UPMC facility within 90 d prior to procedure	21 (13.2)	10 (6.2)	2.30	(1.05–5.05)	.039
Preoperative chlorhexidine skin bathing	66 (41.5)	53 (32.9)	1.45	(0.92–2.28)	.113
Preoperative glucose level ≥125 mg/dL	97 (66.9)	77 (68.1)	0.94	(0.56–1.60)	.833
Hair removed via clipping	76 (47.8)	75 (46.6)	1.05	(0.68–1.63)	.828
Chlorhexidine containing preoperative skin antisepsis	149 (93.7)	144 (89.4)	1.76	(0.78–3.97)	.174
Perioperative antimicrobial agent					
Both cefazolin and vancomycin	14 (8.8)	9 (5.6)	…	Baseline	.125
Cefazolin (no vancomycin)	109 (68.6)	127 (78.9)	0.56	(0.23–1.35)	
Vancomycin (no cefazolin)	35 (22.0)	22 (13.7)	1.03	(0.38–2.78)	
Neither cefazolin nor vancomycin	1 (0.6)	3 (1.9)	0.28	(0.03–2.79)	
Operative factors					
Spinal level					
Cervical	36 (22.6)	65 (40.4)	0.43	(0.27–0.70)	.001
Thoracic	18 (11.3)	15 (9.3)	1.24	(0.60–2.56)	.557
Lumbar and lumbosacral	113 (71.1)	87 (54.0)	2.09	(1.32–3.32)	.002
Approach utilized					
Anterior	8 (5.0)	49 (30.4)	0.12	(0.06–0.27)	<.0001
Posterior	112 (70.4)	89 (55.3)	1.93	(1.22–3.06)	.005
Other	45 (28.3)	24 (14.9)	2.25	(1.29–3.92)	004

https://doi.org/10.1017/ice.2016.307 Published online by Cambridge University Press

Table 2. *Continued*

Variable	Case, No. (%) (n = 159)	Control, No. (%) (n = 161)	OR	95% CI	*P* Value
Surgeon					
A	10 (6.3)	22 (13.7)	…	Baseline	0.001
B	48 (30.2)	35 (21.7)	2.93	(1.24–6.93)	
C	39 (24.5)	23 (14.3)	3.60	(1.46–8.90)	
D	34 (21.4)	26 (16.1)	2.79	(1.13–6.88)	
E	20 (12.6)	32 (19.9)	1.35	(0.53–3.43)	
Other surgeons	8 (5.0)	23 (14.3)	0.78	(0.26–2.31)	
Duration of operation					
<1 h	2 (1.3)	7 (4.3)	…	Baseline	<.0001
1–2 h	7 (4.4)	38 (23.6)	0.58	(0.11–3.21)	
2–3 h	33 (20.8)	46 (28.6)	2.16	(0.45–10.43)	
3–4 h	49 (30.8)	45 (28.0)	3.26	(0.68–15.57)	
4–5 h	28 (17.6)	14 (8.7)	5.90	(1.15–30.37)	
>5 h	40 (25.2)	11 (6.8)	10.57	(2.03–54.88)	
No. of intervertebral levels fused					
1	30 (18.9)	60 (37.3)	…	Baseline	<.0001
2	40 (25.2)	48 (29.8)	1.66	(0.90, 3.04)	
3	35 (22.0)	29 (18.0)	2.39	(1.24–4.61)	
≥4	54 (34.0)	24 (14.9)	4.41	(2.31–8.45)	
Drain placed intraoperatively					
None	5 (3.1)	37 (23.0)	…	Baseline	<.0001
Hemovac	138 (86.8)	98 (60.9)	9.59	(3.74–24.60)	
JP/Blake	16 (10.1)	26 (16.1)	4.25	(1.42–12.74)	
Postoperative factors					
Initial postoperative dressing silver-impregnated	70 (44.0)	54 (33.5)	1.56	(0.99–2.45)	.055
Postoperative glucose level ≥200 mg/dL	49 (33.3)	41 (34.7)	0.94	(0.56–1.57)	.810
Postoperative hematocrit ≤36%	145 (94.2)	101 (80.2)	3.99	(1.79–8.90)	.001
Receipt of red blood cells	74 (46.5)	32 (19.9)	3.51	(2.14–5.77)	<.0001
No. of units of red blood cells received					
0	85 (53.5)	129 (80.1)	…	Baseline	<.0001
1	14 (8.8)	10 (6.2)	2.09	(0.89–4.92)	
2	22 (13.8)	7 (4.3)	4.54	(1.88–11.00)	
≥3	38 (23.9)	15 (9.3)	3.76	(1.95–7.24)	
Receipt of other blood products	41 (25.8)	17 (10.6)	2.94	(1.59–5.45)	.001

NOTE. OR, odds ratio; CI, confidence interval; COPD, chronic obstructive pulmonary disease; MRSA, methicillin-resistant *Staphylococcus aureus*; ASA, American Society of Anesthesiologists; UPMC, University of Pittsburgh Medical Center; JP, Jackson–Pratt.

TABLE 3. Multivariate Logistic Regression Model for Development of Surgical Site Infections Following Neurosurgical Spinal Fusion

Variable	OR	95% CI	*P* Value
Previous MRSA carriage	20.30	(4.64–88.78)	<.0001
Hospitalized in the 30 d prior to surgery	5.80	(1.37–24.57)	.017
ASA score 3 or 4	1.79	(1.004–3.18)	.048
Duration of operation, h	1.58	(1.29–1.93)	<.0001
White race	4.35	(1.28–14.76)	.018
Anterior surgical approach	0.20	(0.08–0.52)	.001

NOTE. OR, odds ratio; CI, confidence interval; MRSA, methicillin-resistant *Staphylococcus aureus*; ASA, American Society of Anesthesiologists.

strongest risk factor for postoperative SSI after adjusting for all other variables.

Of the 40 patients with prior MRSA colonization, 38 developed an SSI, and MRSA was the causative pathogen in 28 of those cases (73.7%). Of the 58 patients with a negative MRSA screen, 28 (48.3%) developed an SSI. All MRSA SSIs occurred in patients who were known to be MRSA carriers.

DISCUSSION

The aim of this study was to identify risk factors for the occurrence of SSI following neurosurgical spinal fusion at a single institution during a 4-year period. To identify independent, modifiable risk factors for SSIs, we performed a

https://doi.org/10.1017/ice.2016.307 Published online by Cambridge University Press

case-control study with multivariate analysis. We found that, in addition to known patient-specific, perioperative, procedural, and postoperative risk factors, prior colonization with MRSA was the strongest independent risk factor for infection. The odds of SSI following spinal fusion for those with prior MRSA nares colonization was 20 times higher than for those without colonization.

In 2014, Thakkar et al[37] were the first to correlate nasal MRSA colonization with postoperative MRSA SSI following neurosurgical spinal surgery; MRSA SSI developed in 8% of patients with prior MRSA colonization compared to 0.61% of those without prior MRSA colonization (OR, 14.23; $P = .02$). While *S. aureus* colonization has not been well described as a risk factor for SSI following spinal fusion, nasal carriage of MRSA appears to be increasingly associated with the development of SSI following other surgical procedures.[38,39] Kim et al[38] reviewed the cases of >7,000 patients undergoing orthopedic surgical procedures, among whom 22.6% were MRSA carriers. Colonized patients underwent a decolonization protocol with chlorhexidine and intranasal mupirocin prior to surgery. These decolonized patients had a significantly lower SSI rate compared to a historical control cohort. Bode et al[39] identified nasal *S. aureus* carriers undergoing general surgical, orthopedic, and neurosurgical procedures; they were then randomized to preoperative decolonization with chlorhexidine and intranasal mupirocin or control. The decolonized cohort had a decreased SSI rate (3.4% vs 7.7%). Given these findings, prior MRSA colonization represents a potentially modifiable risk factor, and attempts should be made to actively screen for *S. aureus* carriage prior to spinal fusion. Those with positive *S. aureus* carriage screens may potentially benefit from decolonization prior to spinal fusion.

With univariate analysis, we identified numerous other factors that were significantly associated with the development of SSIs. However, with multivariate analysis, duration of procedure, ASA score of 3 or 4, white race, and hospitalization within the prior 30 days were found to be independent predictors of developing an SSI following spinal fusion, while an anterior surgical approach was found to be a protective factor. White race represented most cases and controls, and the small number of nonwhite study participants may have limited the ability to assess risk.

In agreement with previous studies,[10–12,19,31] patients with ASA class 3 or 4 had odds 1.79 times higher of developing infection. Fusion of ≥3 vertebrae, postoperative anemia, and receipt of both red blood cells as well as blood products were found to be significant risk factors for SSI by univariate analysis. None of these were significant in the multivariate analysis, whereas procedure lasting >4 hours was a significant risk factor. This finding is likely related to the confounding associations among these variables. These data suggest that more extensive and prolonged spinal fusion procedures carry a higher risk of infection.[9,10,16,17,19,26,27,30,32,33] Additionally, surgeons B, C, and D were risk factors for developing an SSI by univariate but not multivariate analysis. This finding lends further credence to the deduction that more complicated and protracted fusions are critical in the development of SSI, rather than the individual surgeon performing the procedure. We found anterior surgical approach to be a protective factor, which is consistent with prior evaluations.[12,16,17,34] This finding is likely related to the fact that this approach is employed primarily in shorter and less complicated cervical fusions and thus represents a surrogate for procedure complexity.[18]

Contrary to other similar studies, we did not find current tobacco use,[19,27,29,30] obesity,[9–17] or an immunosuppressed state[9,19] to be independent risk factors for the development of SSI following spinal fusion. Interestingly, we also did not find the presence of DM significantly associated with an increased risk of SSI. DM has been reported to be a prevalent comorbidity in patients undergoing spinal surgery in several studies.[9,16,18–26] In our study, we found neither elevated preoperative nor postoperative glucose levels to be correlated with SSI risk. This is not surprising; a 2009 Cochrane Review found insufficient evidence to support strict glycemic control versus conventional management for the prevention of SSI.[40]

Our analysis has several important limitations. Whereas a case-control study is the most practical method to analyze a relatively infrequent event, the retrospective and observational nature of the study type prohibited complete analysis of some potentially significant risk factors because the accuracy of the data was dependent upon documentation in the electronic medical records. We were only able to determine prior hospitalizations that occurred at a UPMC facility. Most patients (69.4%) did not undergo MRSA screening prior to spinal fusion. If prior known MRSA carriage was not recorded in the UPMC medical record, it may not have been captured. For comorbid conditions, this method of collection via chart review based upon clinician recorded history carries the possibility of underestimation due to lack of proper documentation. Additionally, we did not differentiate whether patients with DM were well controlled versus poorly controlled or whether they were insulin dependent versus maintained on oral hypoglycemic agents. For tobacco abuse, we did not differentiate between light and heavy use, and we did not assess former smokers. In our analysis, we were not able to include a few potential risk factors including preoperative serum albumin level, closed suction drain duration, operating room traffic, and timing of prophylactic antibiotic re-dosing. Also, the study's retrospective nature denotes the possibility of underestimating actual infection rates. At our institution, the infection prevention team reviews all outpatient and inpatient microbiology cultures. However, it is possible that a superficial infection may not have been detected, thus causing the underestimation of the total SSI infection rate. Additionally, this was a single-center analysis, so the results may not be generalizable to other facilities, especially those that do not serve as a large referral center or perform surgeries of the same complexity.

Our study has numerous strengths. This is one of the largest studies examining risk factors for SSI after spinal surgery given the large volume of spinal surgeries performed at this single

https://doi.org/10.1017/ice.2016.307 Published online by Cambridge University Press

institution which increases the power of our analysis. Additionally, we focused on a homogenous patient population by honing the evaluation to only spinal fusions performed by neurosurgeons in our hospital. Another study strength is the breadth and variety of variables included in our analysis. It is quite difficult to analyze a complex disease state that has numerous potential risk factors that are intimately interrelated. To attempt to identify the true independent risk factors associated with development of SSIs, we included a wide breadth and variety of variables in our analysis. Additionally, by utilizing multivariate analysis, we were better able to ascertain the degree to which these interrelated variables contribute to SSI development. This study is one of few that have evaluated risk factors for SSI following spinal fusion and have utilized multivariate analysis. Like other studies, traditional risk factors were found; however, prior known MRSA colonization, a potentially modifiable, independent risk factor, was identified in this specific patient population. Areas for continued study include the role of universal screening for *S. aureus* carriage prior to spinal fusion as well as the impact of decolonization strategies for colonized patients prior to neurosurgical spinal fusion.

ACKNOWLEDGMENTS

The authors would like to thank Lloyd Clarke for his technical assistance and support.

Financial support: No financial support was provided relevant to this article.

Potential conflicts of interest: All authors report no conflicts of interest relevant to this article.

Address correspondence to Thomas L. Walsh, MD, Division of Infectious Diseases, Allegheny General Hospital, 320 East North Avenue, 4th Floor East Wing, Suite 406, Pittsburgh, PA 15136 (twalsh@wpahs.org).

REFERENCES

1. Hall MJ, DeFrances CJ, Williams SN, Golosinskiy A, Schwartzman A. National hospital discharge survey: 2007 summary. *Natl Health Stat Report* 2010;29:1–20.
2. National Nosocomial Infections Surveillance System. National Nosocomial Infections Surveillance (NNIS) System Report, data summary from January 1992 through June 2004, issued October 2004. *Am J Infect Control* 2004;32:470.
3. Horan TC, 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;36:309–332.
4. Gaynes RP, Culver DH, Horan TC, Edwards JR, Richards C, Tolson JS, National Nosocomial Infections Surveillance System. Surgical site infection (SSI) rates in the United States, 1992–1998: the National Nosocomial Infections Surveillance System basic SSI risk index. *Clin Infect Dis* 2001;33:s69–s77.
5. Healthcare Cost and Utilization Project: Statistics on hospital stays, 2013. Agency for Healthcare Research and Quality website. http://hcupnet.ahrq.gov/. Published 2013. Accessed August 2, 2016.
6. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR, Hospital Infection Control Practices Advisory Committee. Guideline for prevention of surgical site infection. *Am J Infect Control* 1999;27:97–134.
7. Herwaldt LA, Cullen JJ, Scholz, et al. A prospective study of outcomes, healthcare resource utilization, and costs associated with postoperative nosocomial infections. *Infect Control Hosp Epidemiol* 2006;27:1291–1298.
8. de Lissovoy G, Fraeman K, Hutchins V, et al. Surgical site infection: incidence and impact on hospital utilization and treatment costs. *Am J Infect Control* 2009;37:387–397.
9. Massie JB, Heller JG, Abitbol JJ, McPherson D, Garfin SR. Postoperative posterior spinal wound infections. *Clin Orthop Relat Res* 1992;284:99–108.
10. Rao SB, Vasquez G, Harrop J, et al. Risk factors for surgical site infection following spinal fusion procedures: a case-control study. *Clin Infect Dis* 2011;53:686–692.
11. Koutsoumbelis S, Hughes AP, Girardi FP, et al. Risk factors for postoperative infection following posterior lumbar instrumented arthrodesis. *J Bone Joint Surg Am* 2011;93:1627–1633.
12. Olsen MA, Mayfield J, Lauryssen C, et al. Risk factors for surgical site infection in spinal surgery. *J Neurosurg* 2003;98:149–155.
13. Thomas EJ, Goldman L, Mangione CM, et al. Body mass index as a correlate of postoperative complications and resource utilization. *Am J Med* 1997;102:277–283.
14. Patel N, Bagan B, Vadera S, et al. Obesity and spine surgery: relation to perioperative complications. *J Neurosurg Spine* 2007;6:291–297.
15. Peng CW, Bendo JA, Goldstein JA, Nalbandian MM. Perioperative outcomes of anterior lumbar surgery in obese versus non-obese patients. *Spine J* 2009;9:715–720.
16. Pull ter Gunne AF, Cohen DB. Incidence, prevalence, and analysis of risk factors for surgical site infection following adult spinal surgery. *Spine (Phila Pa 1976)* 2009;34:1422–1428.
17. Maragakis LL, Cosgrove SE, Martinez EA, Tucker MG, Cohen DB, Perl TM. Intraoperative fraction of inspired oxygen is a modifiable risk factor for surgical site infection after spinal surgery. *Anesthesiology* 2009;110:556–562.
18. Olsen MA, Nepple JJ, Riew DK, et al. Risk factors for surgical site infection following orthopaedic spinal operations. *J Bone Joint Surg Am* 2008;90:62–69.
19. Veeravagu A, Patil CG, Lad SP, Boakye M. Risk factors for postoperative spinal wound infections after spinal decompression and fusion surgeries. *Spine* 2009;34:1869–1872.
20. Browne JA, Cook C, Pietrobon R, et al. Diabetes and early postoperative outcomes following lumbar fusion. *Spine* 2007;32:2214–2219.
21. Malone DL, Genuit T, Tracy JK, et al. Surgical site infections: reanalysis of risk factors. *J Surg Res* 2002;103:89–95.
22. Hikata M, Iwanami A, Hosogane N, et al. High preoperative hemoglobic A1C is a risk factor for surgical site infection after posterior thoracic and lumbar spinal instrumentation surgery. *J Orthopaed Sci* 2014;19:223–228.
23. Friedman ND, Sexton DJ, Connelly SM, et al. Risk factors for surgical site infection complicating laminectomy. *Infect Control Hosp Epidemiol* 2007;28:1060–1065.
24. Chen S, Anderson MV, Cheng WK, et al. Diabetes associated with increased surgical site infections in spinal arthrodesis. *Clin Orthop Relat Res* 2009;467:1670–1673.

https://doi.org/10.1017/ice.2016.307 Published online by Cambridge University Press

25. Simpson JM, Silveri CP, Balderston RA, Simeone FA, An HS. The results of operations on the lumbar spine in patients who have diabetes mellitus. *J Bone Joint Surg Am* 1993;75: 1823–1829.
26. Wilmmer C, Gluch H, Franzreb M, Ogon M. Predisposing factors for infection in spine surgery: a survey of 850 spinal procedures. *J Spinal Disord* 1998;11:124–128.
27. Schimmel JJP, Horsting PP, de Kleuver M, Wonders G, van Limbeek J. Risk factors for deep surgical site infections after spinal fusion. *Eur Spine J* 2010;19:1711–1719.
28. Klein JD, Hey LA, Yu CS, et al. Perioperative nutrition and postoperative complications in patients undergoing spinal surgery. *Spine* 1996;21:2676–2682.
29. Ahn N, Klug R, Nho S, et al. Smoking, smoking cessation, and wound complications after lumbar spine surgery. *Spine J* 2002;2: 113–114.
30. Fang A, Hu SS, Endres N, et al. Risk factors for infection after spinal surgery. *Spine* 2005;30:1460–1465.
31. Apisarnthanarak A, Jones M, Waterman BM, et al. Risk factors for spinal surgical-site infections in a community hospital: a case-control study. *Infect Control Hosp Epidemiol* 2003;24:31–36.
32. Richards BS, Herring JA, Johnston CE, et al. Treatment of adolescent idiopathic scoliosis using Texas Scottish Rite Hospital instrumentation. *Spine* 1994;19:1598–1605.
33. Weinstein MA, McCabe JP, Cammisa FP Jr. Postoperative spinal wound infection: a review of 2391 consecutive index procedures. *J Spinal Disord* 2000;13:422–426.
34. Levi AD, Dickman CA, Sonntag VK. Management of postoperative infections after spinal instrumentation. *J Neurosurg* 1997;86:975–980.
35. Yount RJ, Vries JK, Coucill CD. The medical archival retrieval system: an information retrieval system based on distributed parallel processing. *Inf Proc Manag* 1991;27:379–389.
36. ACH Surveillance for Surgical Site Infection Events. 2016. Centers for Disease Control and Prevention website. http://www.cdc.gov/nhsn/acute-care-hospital/ssi/. Published 2016. Accessed July 28, 2016.
37. Thakkar V, Ghobrial GM, Maulucci CM, et al. Nasal MRSA colonization: impact on surgical site infection following spine surgery. *Clin Neurol Neurosurg* 2014;125:94–97.
38. Kim DH, Spencer M, Davidson SM, et al. Institutional prescreening for detection and eradication of methicillin-resistant *Staphylococcus aureus* in patients undergoing elective orthopaedic surgery. *J Bone Joint Surg Am* 2010;92:1820–1826.
39. Bode LG, Kluytmans JA, Wertheim HF, et al. Preventing surgical-site infections in nasal carriers of *Staphylococcus aureus*. *N Engl J Med* 2010;362:9–17.
40. Kao LS, Meeks D, Moyer VA, et al. Peri-operative glycaemic control regimens for preventing surgical site infections in adults. *Cochrane Database Syst Rev* 2009;8(3):CD006806.

https://doi.org/10.1017/ice.2016.307 Published online by Cambridge University Press