

Risk factors for surgical site infection of pilon fractures

Tingting Ren,¹ Liang Ding,^{1,*} Feng Xue,^{1,**} Zhimin He,¹ Haijun Xiao¹

¹Shanghai Jiao Tong University, South Campus, School of Medicine, Renji Hospital, Department of Intensive Care Unit, Shanghai, China. ²Shanghai Fengxian Central Hospital, Department of Orthopaedics, Shanghai, China.

OBJECTIVES: Pilon fracture is a complex injury that is often associated with severe soft tissue damage and high rates of surgical site infection. The goal of this study was to analyze and identify independent risk factors for surgical site infection among patients undergoing surgical fixation of a pilon fracture.

METHODS: The medical records of all pilon fracture patients who underwent surgical fixation from January 2010 to October 2012 were reviewed to identify those who developed a surgical site infection. Then, we constructed univariate and multivariate logistic regressions to evaluate the independent associations of potential risk factors with surgical site infection in patients undergoing surgical fixation of a pilon fracture.

RESULTS: A total of 519 patients were enrolled in the study from January 2010 to October 2012. A total of 12 of the 519 patients developed a surgical site infection, for an incidence of 2.3%. These patients were followed for 12 to 29 months, with an average follow-up period of 19.1 months. In the final regression model, open fracture, elevated postoperative glucose levels (≥ 125 mg/dL), and a surgery duration of more than 150 minutes were significant risk factors for surgical site infection following surgical fixation of a pilon fracture.

CONCLUSIONS: Open fractures, elevated postoperative glucose levels (≥ 125 mg/dL), and a surgery duration of more than 150 minutes were related to an increased risk for surgical site infection following surgical fixation of a pilon fracture. Patients exhibiting the risk factors identified in this study should be counseled regarding the possible surgical site infection that may develop after surgical fixation.

KEYWORDS: Pilon fracture; Surgical site infection; Open reduction; Internal fixation; External fixation.

Ren T, Ding L, Xue F, He Z, Xiao H. Risk factors for surgical site infection of pilon fractures. *Clinics*. 2015;70(6):419-422

Received for publication on February 12, 2015; First review completed on March 19, 2015; Accepted for publication on March 19, 2015

E-mail: dl1900@sina.com / xuefengky@163.com

*Corresponding author

**Co-corresponding author

■ INTRODUCTION

The pilon fracture, which constitutes 1–5% of all lower extremity fractures and 7–10% of all tibial fractures (1), extends from the distal tibial metaphysis into the ankle. It is a complex injury that is often associated with severe soft tissue damage. This combination of osseous and soft tissue trauma, which is associated with high rates of infection, has long challenged surgeons. Mccann et al. (2) observed a 14.3% rate of superficial infection and a 2.0% rate of deep infection in their series of 49 pilon fractures treated definitively via surgical fixation. White et al. (3) reported that six patients developed a deep wound infection (four after an open fracture and two after a closed fracture) in their series of ninety-five type 43.C pilon fractures treated via surgical fixation. However, prior investigations were limited

methodologically by small sample sizes with little adjustment for confounders.

Therefore, we conducted this retrospective study to assess the incidence of surgical site infection (SSI) in patients with a pilon fracture, to determine whether various perioperative and postoperative laboratory findings and independent risk factors are associated with the development of SSIs, and to quantify the contribution of independent risk factors to the probability of SSI development in patients with a pilon fracture.

■ MATERIAL AND METHODS

Data collection

Institutional review board approval was granted for this investigation before medical record and radiographic review. All patients who were treated for a pilon fracture at our institution from January 2010 to October 2012 were retrospectively identified using our electronic database. The inclusion criteria were as follows: patient age of 18 years or greater; underwent surgical fixation of a pilon fracture during the study period; and at least 12 months of clinical and radiographic follow-up. The exclusion criteria included the following: pregnancy, lactation, sepsis, human immunodeficiency virus-1 (HIV) infection, any site infection within

Copyright © 2015 CLINICS – This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

No potential conflict of interest was reported.

DOI: 10.6061/clinics/2015(06)06



the past thirty days, underwent initial fracture surgery at an outside hospital or presented with SSIs after being treated at another hospital, metastatic fractures, death within 30 days of initial surgery, and severe concomitant disease of other systems.

The following information was extracted from the medical record and radiographic review: age, gender, medical comorbidities, fracture patterns (The AO Foundation and Orthopaedic Trauma Association (AO/OTA) classification) (4), fracture type, time to definitive surgical treatment, surgical approach, fixation type, polytrauma, operative characteristics, duration of surgery, estimated blood loss, use of a bone graft, temporary use of external fixation before definitive surgery (treated in two stages), infection at other sites, and causes of injuries.

One investigator retrospectively reviewed the medical records. These data underwent extensive logic checks by other investigators to identify illogical or impossible data. All illogical data were repeatedly reviewed by comparing both the written and the electronic patient medical records.

According to the Centers for Disease Control and Prevention (CDC) criteria, SSIs are defined as infections occurring within 30 days after a surgical operation (or within one year if an implant is left in place after the procedure) and affecting either the incision or deep tissue at the operation site (5). Incisional SSIs were categorized into those involving only the skin and subcutaneous tissue (superficial incisional SSI) and those involving deeper soft tissues of the incision (deep incisional SSI). The SSI category can be determined based on clinical or laboratory findings (wound culture).

Polytrauma was defined as trauma to more than one of the following systems: musculoskeletal, abdominal, cardiothoracic, urogenital, vascular, and central nervous systems. Multiple isolated orthopedic injuries were not classified as polytrauma unless they were associated with hemodynamic instability.

Data analysis

Biostatisticians at our institution completed statistical analysis of the collected data. All potential risk factors were evaluated for a univariate association with SSIs using independent-samples *t* tests for continuous variables and chi-square or Fisher exact tests for categorical or discrete variables. Unadjusted odds ratios and 95% confidence intervals (CIs) were calculated and presented for discrete variables. A multivariate logistic regression was then used to evaluate the independent associations of each potentially explanatory variable. All variables that had been previously identified in the literature, those with clinical and/or biologic plausibility, and those with a univariate *p*-value of ≤ 0.10 were considered to be eligible for inclusion in the multivariate model. Using a forward, stepwise procedure, variables that displayed a *p*-value of ≤ 0.10 remained in the final model, and significant factors were defined as those variables which displayed a *p*-value of ≤ 0.05 . Adjusted odds ratios and their respective 95% CIs according to the final model were reported.

RESULTS

From January 2010 to October 2012, a total of 519 patients were enrolled in the study. A total of 12 of the 519 cases developed SSIs, for an incidence of 2.3%. The patients were followed for 12 to 29 months, with an average follow-up period of 19.1 months. Patients at our institution are treated

according to published CDC/National Nosocomial Infections Surveillance System guidelines for preventing surgical site infections. Using a dose based on the patient's weight, 1 to 2 g of cefazolin, a first-generation cephalosporin, was administered to each patient within one hour prior to the skin incision. During the first twenty-four hours following wound closure, the surgery patients were treated with a prophylactic antibiotic regimen per this protocol. Twenty-two of the SSIs were treated conservatively with local wound care and systemic antibiotic therapy. Nine patients required surgical wound debridement, eleven required hardware removal, and three ultimately required free myocutaneous flap coverage of the wound. No patient required amputation.

Clinical variables

Based on univariate analysis, we observed that smoking history, but not, age, gender, obesity, medical history, causes of injuries, side of injury, polytrauma, infection at other sites, associated ipsilateral distal fibular fracture, or ASA score, was significantly associated with SSI.

Association of therapy-related variables with SSI

The results of univariate analysis of the therapy-related variables are displayed in Table 1. We found that the fracture grade, the fracture patterns (AO/OTC classification), the duration of surgery, the use of a drain, the use of a bone graft, and the total number of persons in the operating room were significantly associated with SSI.

Association of laboratory variables with SSI

Table 2 illustrates the laboratory variables associated with SSI. Elevated postoperative glucose levels (≥ 125 mg/dL) were significantly associated with SSI.

Multivariate analysis

Table 3 shows the results of multivariate logistic regression analysis assessing the association of each risk factor with SSI after adjusting for all other potential risk factors. In the final model, we identified three factors that independently predicted SSI: 1) open fracture (odds ratio, 7.42; 95% CI: 1.94 to 28.34, $p=0.003$); 2) duration of surgery (odds ratio, 1.06; 95% CI: 1.02 to 1.11, $p=0.009$); and 3) postoperative glucose level ≥ 125 mg/dL (odds ratio, 1.47; 95% CI: 1.18 to 1.84, $p=0.001$).

DISCUSSION

An ideal analysis of the cause of postoperative infection would necessarily consider innumerable variables including patient characteristics, operating room environment, operative site preparation and draping, antibiotic administration, surgical technique and postoperative care. Although it is impossible to exhaustively examine every variable that might affect the rate of SSI, this study focused on a broad array of clinical, laboratory and therapy-related variables, specifically sought the therapy-related variables over which the surgeon can reasonably control.

SSIs are one of the most common major complications in patients with pilon fracture. Some authors have reported a variable infection rate from 0–55% (6–8). Compared to historical reports, the infection rate in our study was 2.3%. In previous reports, long duration of surgery and open fracture have attracted much attention as predictors of prolonged wound healing and infection (9–11). We also

**Table 1** - Distribution of a subset of clinical and therapy-related variables in patients with surgical site infection and in controls.

Risk factor	SSI group (N=12)	Non-SSI group (N=507)	Odds ratio (95% confidence interval)	p-value
Smoking history‡	4	65	3.40 (1.00–11.61)	0.039†
Fracture grade‡				
Closed	3	418	(Reference)	
Gustilo I	2	49	5.69 (0.93–34.87)	<0.001†
Gustilo II	5	32	21.77 (4.98–95.24)	
Gustilo III	2	8	34.83 (5.10–237.86)	
AO/OTC‡				
43.A	2	200	(Reference)	0.013†
43.B	7	278	2.52 (0.52–12.25)	
43.C	3	29	10.35 (1.66–64.56)	
Duration of surgery (min)*	156.67 ± 38.57	132.36 ± 24.78	-	0.001†
Use of a drain‡	6	212	1.39 (0.44–4.37)	0.570
Time from injury to surgery (days)*	8.12 ± 3.23	8.55 ± 3.67	-	0.688
Estimated blood loss (mL)*	149.39 ± 37.13	143.32 ± 33.88	-	0.541
Bone graft use‡	3	34	4.64 (1.20–17.93)	0.015†
Temporary external fixation (treated in two stages)‡	1	123	0.28 (0.04–2.22)	0.201
Tourniquet use‡	8	385	0.63 (0.19–2.14)	0.459
Definitive fixation‡				0.985
External fixation with limited internal fixation	1	43	(Reference)	
Open reduction internal fixation (ORIF)	11	464	1.02 (0.13–8.09)	
Surgery approach (ORIF group only, N=475)‡				0.934
Anterior	2	66	(Reference)	
Anterolateral	3	103	0.96 (0.16–5.91)	
Anteromedial	5	228	0.72 (0.14–3.82)	
Posteromedial	0	18	-	
Medial	1	29	1.14 (0.10–13.05)	
Posterolateral	0	20	-	
Total no. of persons in the operating room (x11)*	8.17 ± 2.44	5.89 ± 1.38	-	0.001†

* The values are presented as the means and the standard deviation. †Significant at $p=0.05$.

‡The values are presented as the number of patients or controls followed by the percentage in parentheses.

Table 2 - Comparison of laboratory findings in patients with and without surgical site infection.

Perioperative laboratory findings	SSI group (N=12)	Non-SSI group (N=507)	Odds ratio (95% confidence interval)	p-value
Albumin (preop.) (g/dL)*	3.41 ± 0.35	3.49 ± 0.39	-	0.482
Glucose level(mg/dL)*				
Preop.	107.81 ± 10.01	104.56 ± 9.79	-	0.257
Postop.	122.08 ± 9.37	97.71 ± 8.91	-	<0.001†
Glucose level of ≥ 125 mg/dL‡				
Preop.	1	13	3.46(0.42–28.78)	0.223
Postop.	5	19	18.35(5.33–63.13)	<0.001†
Hemoglobin*				
Preop.	12.03 ± 2.51	11.86 ± 2.37	-	0.806
Postop.	11.12 ± 1.75	11.24 ± 1.61	-	0.799
Hematocrit*				
Preop.	34.54 ± 10.12	35.62 ± 9.89	-	0.709
Postop.	31.67 ± 5.86	31.83 ± 6.11	-	0.929
Lymphocyte*				
Preop.	1.89 ± 0.48	1.92 ± 0.53	-	0.846
Postop.	1.71 ± 0.62	1.69 ± 0.57	-	0.905

* The values are expressed as the means and the standard deviation. †Significant at $p=0.05$.

‡The values are expressed as the number of patients or controls followed by the percentage in parentheses.

identified associations between these two risk factors and SSI. Diabetic patients have been documented to exhibit greater complication rates for both open and closed treatment of ankle fractures, with infection rates in diabetic patients ranging from 10% to 60% (12,13). In our study, we identified an elevated postoperative glucose level (≥ 125 mg/dL) as a risk factor for SSI. Hyperglycemia during the immediate postoperative period was an independent risk factor for developing infection among patients regardless of their history of diabetes, and the risk of infection correlated with the degree of glucose elevation (14).

It has been demonstrated that postoperative hyperglycemia can impede wound healing and can predispose patients to infection caused by ischemia secondary to microvascular abnormalities (15,16). Thus, acute elevations in glucose levels temporally related to the surgical procedure are important.

Open fracture is a well-accepted risk factor for deep infection due to more extensive soft-tissue injury and frank contamination of the wound by skin and ambient flora (2). Therefore, it is not surprising that in our study, this clinical variable was found to be an independent predictor of



Table 3 - Multivariate Logistic Regression Analysis of Risk Factors for SSI.

Risk factor	Adjusted odds ratio (95% confidence interval)	p-value
Open fracture	7.42 (1.94–28.34)	0.003†
Duration of surgery	1.06 (1.02–1.11)	0.009†
Postoperative glucose level (≥ 125 mg/dL)	1.47 (1.18–1.84)	0.001†

† Significant at $p=0.05$.

postoperative SSI. The presence of open fractures should continue to alert the treating surgeon to a heightened risk profile.

Other factors such as smoking, fracture patterns, and bone graft use only showed a trend towards significance based on univariate analysis and, thus, were not included in the multivariate model. It is possible that in a larger study with more infection events, these variables could be more definitively shown to contribute to higher rates of SSI.

Although we believe that this information can be useful to surgeons treating patients with pilon fractures, we acknowledge certain limitations of our investigation. First, the electronic medical records greatly facilitated this study but resulted in the exclusion of certain factors that may have been associated with SSI development, including nutritional status, the precise timing of preoperative antibiotic administration, and the skill of the operating teams. Second, we assumed that a patient who was not diagnosed with an infection within 30 days and did not return specifically for treatment of an infection within the next 12 months did not experience an infection. Therefore, a few patients who were diagnosed elsewhere with an infection between 1 and 12 months postoperatively may have been missed in our study. Third, the reliability and the accuracy of SSI classification have been questioned, but the sensitivity of the various surveillance methods reported in the literature has ranged from 80% to 90%, and the specificity has been nearly 100% (5,17). Additionally, this study did not include an analysis of the skill of the senior surgeon as a risk factor for SSI. Meticulous identification of the anatomy and preservation of the soft tissue are essential in pilon fracture surgery, and surgical experience is critical for recognizing and protecting these structures. Technical complications such as wound edge necrosis, hematoma, and wound infection occur as a consequence of several factors, including surgical inexperience. We believe that these operations should be limited to specially trained surgeons in this field.

In conclusion, open fractures, elevated postoperative glucose levels (≥ 125 mg/dL), and a long surgical duration were related to an increased risk for SSI following surgical fixation of a pilon fracture. Patients exhibiting the risk factors identified in this study should be counseled regarding possible SSI that may arise after surgical fixation.

AUTHOR CONTRIBUTIONS

Ren T, Ding L and Xue F conceived and designed the study. He Z and Xiao H analyzed and interpreted the data. Ren T and Ding L drafted the manuscript and critically revised the manuscript for intellectual content. Xue F inspected and approved the final version of the manuscript. All of the authors were involved in manuscript preparation.

REFERENCES

- Egol KA, Wolinsky P, Koval KJ. Open reduction and internal fixation of tibial pilon fractures. *Foot Ankle Clin.* 2000;5(4):873–85.
- McCann PA, Jackson M, Mitchell ST, Atkins RM. Complications of definitive open reduction and internal fixation of pilon fractures of the distal tibia. *Int Orthop.* 2011;35(3):413–8, <http://dx.doi.org/10.1007/s00264-010-1005-9>.
- White TO, Guy P, Cooke CJ, Kennedy SA, Droll KP, Blachut PA, et al. The results of early primary open reduction and internal fixation for treatment of OTA 43.C-type tibial pilon fractures: a cohort study. *J Orthop Trauma.* 2010;24(12):757–63.
- Marsh JL1, Slongo TF, Agel J, Broderick JS, Creevey W, DeCoster TA, et al. Fracture and Dislocation Classification Compendium - 2007: Orthopaedic Trauma Association Classification, Database and Outcomes Committee. *J Orthop Trauma.* 2007;21(10 Suppl):S1–133, <http://dx.doi.org/10.1097/00005131-200711101-00001>.
- Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Hospital. Infection Control Practices Advisory Committee. *Infect Control Hosp Epidemiol.* 1999;20(4):250–78;quiz 279–80, <http://dx.doi.org/10.1086/iche.1999.20.issue-4>.
- Joveniaux P, Ohl X, Harisboure A, Berrichi A, Labatut L, Simon P, et al. Distal tibia fractures: management and complications of 101 cases. *Int Orthop.* 2010;34(4):583–8, <http://dx.doi.org/10.1007/s00264-009-0832-z>.
- Tarkin IS, Clare MP, Marcantonio A, Pape HC. An update on the management of high-energy pilon fractures. *Injury.* 2008;39(2):142–54, <http://dx.doi.org/10.1016/j.injury.2007.07.024>.
- Zelle BA, Bhandari M, Espiritu M, Koval KJ, Zlowodzki M. Treatment of distal tibia fractures without articular involvement: a systematic review of 1125 fractures. *J Orthop Trauma.* 2006;20(1):76–9, <http://dx.doi.org/10.1097/01.bot.0000202997.45274.a1>.
- Colman M, Wright A, Gruen G, Siska P, Pape HC, Tarkin I. Prolonged operative time increases infection rate in tibial plateau fractures. *Injury.* 2013;44(2):249–52, <http://dx.doi.org/10.1016/j.injury.2012.10.032>.
- Jones KB, Maiers-Yelden KA, Marsh JL, Zimmerman MB, Estin M, Saltzman CL. Ankle fractures in patients with diabetes mellitus. *J Bone Joint Surg Br.* 2005;87(4):489–95, <http://dx.doi.org/10.1302/0301-620X.87B4.15724>.
- Miller AG, Margules A, Raikin SM. Risk factors for wound complications after ankle fracture surgery. *J Bone Joint Surg Am.* 2012;94(22):2047–52, <http://dx.doi.org/10.2106/JBJS.K.01088>.
- Schatzker J, McBroom R, Bruce D. The tibial plateau fracture. The Toronto experience 1968–1975. *Clin Orthop Relat Res.* 1979;(138):94–104.
- Stark E, Stucken C, Trainer G, Tornetta P 3rd. Compartment syndrome in Schatzker typeVI plateau fractures and medial condylar fracture-dislocations treated with temporary external fixation. *J Orthop Trauma.* 2009;23(7):502–6, <http://dx.doi.org/10.1097/BOT.0b013e3181a18235>.
- Latham R, Lancaster AD, Covington JF, Pirolo JS, Thomas CS Jr. The association of diabetes and glucose control with surgical-site infections among cardiothoracic surgery patients. *Infect Control Hosp Epidemiol.* 2001;22(10):607–12, <http://dx.doi.org/10.1086/iche.2001.22.issue-10>.
- Theuma P, Fonseca VA. Novel cardiovascular risk factors and macrovascular and microvascular complications of diabetes. *Curr Drug Targets.* 2003;4(6):477–86, <http://dx.doi.org/10.2174/1389450033490939>.
- Sheetz MJ, King GL. Molecular understanding of hyperglycemia's adverse effects for diabetic complications. *JAMA.* 2002;288(20):2579–88, <http://dx.doi.org/10.1001/jama.288.20.2579>.
- Smyth ET, Emmerson AM. Surgical site infection surveillance. *J Hosp Infect.* 2000;45(3):173–84, <http://dx.doi.org/10.1053/jhin.2000.0736>.