



Improvement in Hyperglycemia Prevents Surgical Site Infection Irrespective of Insulin Therapy in Non-diabetic Patients Undergoing Gastrointestinal Surgery

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Abstract

Background Intensive glycemic control is recommended to prevent surgical site infections (SSI). Our aim was to evaluate retrospectively the effect of improvement in hyperglycemia irrespective of insulin use on the incidence of SSI in non-diabetic patients.

Methods The highest blood glucose (BG) concentration within 12 h (early peak BG) and the final BG from 12 to 24 h after surgery were evaluated in patients who underwent gastrointestinal surgery. Patients with an early peak BG of ≥ 150 mg/dL were divided into those with persistent (final BG of ≥ 150 mg/dL) and improved hyperglycemia (final BG of < 150 mg/dL). Patients without hyperglycemia and those with late-onset hyperglycemia were also assessed for SSI risk.

Results Overall, 1612 patients were studied (diabetes, $n = 293$). Although hyperglycemia increased the SSI rates in non-diabetic patients, no correlation was demonstrated in patients with diabetes at any cutoff final BG defining hyperglycemia except for 180 mg/dL. Hyperglycemia improved without insulin therapy in 283 of 512 non-diabetic patients who had early hyperglycemia. The adjusted standardized residual for those with SSI and persistent hyperglycemia was 5.2 ($P < 0.05$). In contrast, the absence of hyperglycemia was a significant preventive factor for SSI. In the multivariate analyses, persistent hyperglycemia was an independent risk factor for SSI (odds ratio 1.54; 95% confidence interval 1.03–2.31).

Conclusions Remission of hyperglycemia within 24 h after surgery prevented SSI in non-diabetic patients. Considering that hyperglycemia improved in approximately half of patients without insulin therapy, commencement of insulin dosing after two consecutive BGs of ≥ 150 mg/dL might be reasonable, especially in general wards.

Introduction

Several observational studies have shown an association between hyperglycemia and postoperative complications including surgical site infection (SSI) [1–5]. Acute hyperglycemia can significantly alter the innate immune responses to infection in surgical patients, resulting in SSI [6]. Some studies evaluated the highest blood glucose concentration (BG) after surgery as a cause of SSI [5]. However, SSI can be prevented by controlling the BG with insulin [1, 3, 7], and transient hyperglycemia immediately after completion of surgery may not cause SSI. A meta-

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analysis revealed that target BGs of both <110 and 110–150 mg/dL showed a significant benefit in reducing SSI for these intensive insulin protocols compared with a conventional protocol [1]. The guidelines established by the American College of Surgeons and Surgical Infection Society [8] recommended that the target BG should range from 110 to 150 mg/dL. However, there is compelling agreement that stress hyperglycemia should be treated in all surgical patients [9].

Stress hyperglycemia arises as a result of a cascade of normal physiologic responses to a surgical insult [6], and the effect of surgical stress can be attenuated over time, leading to achievement of the target BG the next morning in some non-diabetic patients who are not undergoing insulin therapy. Considering the significantly higher risk of hypoglycemia in association with an intensive insulin protocol, the target BG the morning after surgery might not be equal to the BG needed to start insulin therapy, especially in a non-surgical intensive care unit (ICU) setting. The present study was performed to investigate the effect of improvement in hyperglycemia irrespective of insulin therapy on SSI and assess the natural remission of hyperglycemia within 24 h after surgery in non-diabetic patients.

Methods

This retrospective study was conducted in three surgical wards from January 2014 to January 2016 and was approved by the Institutional Review Board of Hyogo College of Medicine (No. 2284). The inclusion criteria were age 15 years or older, performance of gastroenterological surgery, survival for at least 5 days, and measurement of BG at least twice. Patients receiving steroids were excluded because steroids can increase BG concentrations. The highest BG within 12 h (early peak BG) and the final BG from 12 to 24 h after surgery were evaluated. In accordance with hospital protocols, laboratory data, including BG, were measured at least when patients were admitted to the ward after surgery and the day after surgery. No BG measurements were taken with point-of-care testing of capillary blood; all BG data were measured using laboratory analyzers or arterial blood gas analyzers. Patients with a history of diabetes or hemoglobin A1c (HbA1c) > 6.5% were classified as having diabetes.

Hyperglycemia was defined by several cutoff BGs (110, 150, 180, and 200 mg/dL). SSI rates were evaluated in patients with and without diabetes according to final BG categories (<12.5th percentile, 12.5th–25th percentile, 25th–37.5th percentile, 37.5th–50th percentile, 50th–62.5th percentile, 62.5th–75th percentile, 75th–87.5th percentile, and \geq 87.5th percentile). For evaluation of the effect of BG control, patients with an early peak BG of \geq 150 mg/dL

were divided into those with persistent hyperglycemia (final BG of \geq 150 mg/dL) and improved hyperglycemia (final BG of <150 mg/dL). Patients without hyperglycemia throughout the postoperative period (within 24 h postoperatively) (early peak and final BG of <150 mg/dL) and those with late-onset hyperglycemia (early peak BG of <150 mg/dL, final BG of \geq 150 mg/dL) were also assessed for SSI risk. Commencement of insulin dosing according to peak BG categories (<110, 110–149, 150–179, 180–199 mg/dL, and \geq 200 mg/dL) was evaluated. Insulin was used at the clinician's discretion, and no interventions for BG control were mandated.

SSI was diagnosed based on definitions stated in the guidelines issued by the National Nosocomial Infections Surveillance System [10]. The wounds were inspected daily by a nurse, once a week by a surveillance member, once during the hospital stay by an attending physician, and at the 4-week postoperative follow-up visits. The criteria for the diagnosis of SSI included an infection that occurred within 30 days after the operation and at least one of the following: (1) purulent discharge from the incision or from a drain placed into the organ/space; (2) organisms could be isolated by culturing fluid or tissue from the incision or the organ/space; (3) an open wound with signs and symptoms of infection; and (4) an abscess or other evidence of infection found on examination of the incision or the organ/space. In this series, the 75th percentile was used for the definition of prolonged surgery in each procedure, massive intraoperative hemorrhage, and long preoperative hospital stay.

Adjusted standardized residuals for performing post hoc tests were used in the comparison of SSI rate among the four BG control groups. Absolute values greater than the critical value of 1.96 have raw *P* values of less than 0.05. Univariate analyses for risk factors associated with SSI were performed by the Chi-square test, and potential confounders were examined by cross-tabulation. Variables selected by these univariate analyses (*P* < 0.1) were subsequently entered into a logistic regression model to estimate the size of the association [odds ratio (OR)] and the 95% confidence interval (CI). SPSS v.21.0 for Windows (IBM Corp., Armonk, NY, USA) was used for all analyses, and the level of significance was set at *P* < 0.05.

Results

Overall, 1899 patients underwent gastrointestinal surgery, of whom 1612 (male, *n* = 1043, diabetes, *n* = 293) were included in the analyses. Patients who did not survive at least 5 days after surgery (one patient), did not have the required two measurements of BG (127 patients) or were receiving steroids (159 patients) were excluded. The mean

age was 59.6 ± 17.8 years. Patients with an American Society of Anesthesiologists physical status score of ≥ 3 comprised 21.3% of the total patients. Preoperative hemoglobin A1c levels of the patients with diabetes were as follows: 61 with $<6.5\%$, 111 with 6.5–6.9%, 81 with 7.0–7.9%, and 40 with $\geq 8.0\%$. In total, 136 patients were admitted to the ICU after surgery. Colorectal surgery was the most frequently performed surgery ($n = 572$), followed by small intestinal surgery ($n = 324$), hepatobiliary pancreatic surgery ($n = 329$), gastric surgery ($n = 279$), esophageal surgery ($n = 49$), and other surgeries ($n = 59$). The 28-day mortality rate was 0.37%.

SSI occurred in 231 patients (14.3%), 67 of whom had incisional SSI, whereas 164 had organ/space SSI. The SSI rate was 14.0% in non-diabetic patients and 16.0% in patients with diabetes. Median length of hospital stay was 20 days, and there was a significant difference between patients with and without SSI (41 days versus 18 days, $P < 0.001$).

The incidence of hyperglycemia as assessed by the early peak BG in non-diabetic patients was significantly lower than that in patients with diabetes at each cutoff value (110 mg/dL: 79.4% vs. 94.9%; 150 mg/dL: 38.8% vs. 74.1%; 180 mg/dL: 16.1% vs. 52.2%; 200 mg/dL: 7.0% vs. 35.2%; $P < 0.001$ each). The number of patients with diabetes who were treated by insulin according to the peak BG categories was 5/15 (33.3%) for <110 mg/dL, 21/61 (34.4%) for 110–149 mg/dL, 32/64 (50.0%) for 150–179 mg/dL, 30/50 (60.0%) for 180–199 mg/dL, and 86/103 (83.5%) for ≥ 200 mg/dL. However, a low performance rate was observed in non-diabetic patients [0/272 (0.0%), 1/535 (0.2%), 3/300 (0.1%), 4/120 (3.3%), and 8/92 (8.7%), respectively].

There were 679 non-diabetic patients and 37 diabetic patients who never developed hyperglycemia, 287 non-diabetic patients and 55 diabetic patients with early hyperglycemia with resolution, 225 non-diabetic patients and 162 diabetic patients with persistent hyperglycemia, and 128 non-diabetic patients and 39 diabetic patients with late-onset hyperglycemia. Among 512 non-diabetic patients with an early peak BG of ≥ 150 mg/dL, hyperglycemia improved in 55.3% of patients without insulin therapy. BG significantly decreased from 177.6 ± 30.1 to 127.5 ± 14.4 mg/dL without insulin use in patients with improved hyperglycemia ($P < 0.001$). The SSI rate was 24.9% in non-diabetic patients with persistent hyperglycemia, 14.3% in those with improved hyperglycemia, 14.1% in those with late-onset hyperglycemia, and 10.2% in those without hyperglycemia (Fig. 1). The adjusted standardized residual for those with SSI and persistent hyperglycemia was 5.2 ($P < 0.05$). In contrast, the adjusted standardized residual for those with SSI and without hyperglycemia was -4.1 ; thus, the absence of

hyperglycemia was a significant preventive factor for SSI ($P < 0.05$). In diabetic patients, persistent hyperglycemia also increased SSI rate compared with other BG control groups (the adjusted standardized residual, 2.6) (Fig. 1).

A significant difference was found between the early peak BG and final BG (150.7 ± 46.2 vs. 144.2 ± 40.6 mg/dL, respectively, $P < 0.001$). The SSI rate was compared between patients with and without hyperglycemia according to the different cutoff early peak BG within 12 h and final BG (Fig. 2). Below and above cutoff final BG values discriminated the SSI rate between patients better than did below and above cutoff early peak BG values. When a cutoff level of 200 mg/dL was adopted, the SSI rate was not significantly different between patients with below and above cutoff BG values for the early peak BG (17.4% vs. 13.9%, respectively, $P = 0.187$); however, a significantly higher SSI rate was demonstrated for the final BG (22.3% vs. 13.5%, $P = 0.004$).

In non-diabetic patients, the SSI rates increased incrementally with higher final BG categories (<12.5 th percentile, 8.0%; 12.5th–25th percentile, 9.0%; 25th–37.5th percentile, 11.3%; 37.5th–50th percentile, 11.0%; 50th–62.5th percentile, 14.8%; 62.5th–75th percentile, 14.7%; 75th–87.5th percentile, 20.5%; ≥ 87.5 th percentile, 21.8%). However, patients with diabetes did not show an increase in the SSI rate in a dose–response manner as the BG categories increased (Fig. 3). Hyperglycemia assessed by final BG was associated with higher SSI rates in non-diabetic patients. However, no correlation between hyperglycemia and the SSI rate was found in patients with diabetes at any cutoff final BG values defining hyperglycemia except for 180 mg/dL (Fig. 4). Hence, further evaluation of the effect of BG control on the SSI rate was conducted only in non-diabetic patients in this study.

Table 1 shows the results of univariate analyses for the risk factors associated with SSI in non-diabetic patients. Common risk factors including American Society of Anesthesiologist physical status classification ≥ 3 (SSI rate, 18.5%), prolonged surgery (SSI rate, 23.3%), and wound class ≥ 3 (SSI rate, 22.8%) were identified. Persistent hyperglycemia and a final BG of ≥ 150 mg/dL were associated with an increased risk of SSI, whereas the absence of hyperglycemia throughout the postoperative period was associated with a decreased risk of SSI. In the multivariate analyses, independent risk factors for SSI were persistent hyperglycemia (OR 1.54; 95% CI 1.03–2.31), prolonged surgery, blood transfusion, preoperative infections, hepatectomy with biliary tract resection and other types of hepatobiliary pancreatic surgery, and esophageal surgery. Small intestinal surgery and laparoscopic surgery were associated with a lower risk (Table 2).

The various gastrointestinal surgical interventions carried out in the study cohort were found to carry varying

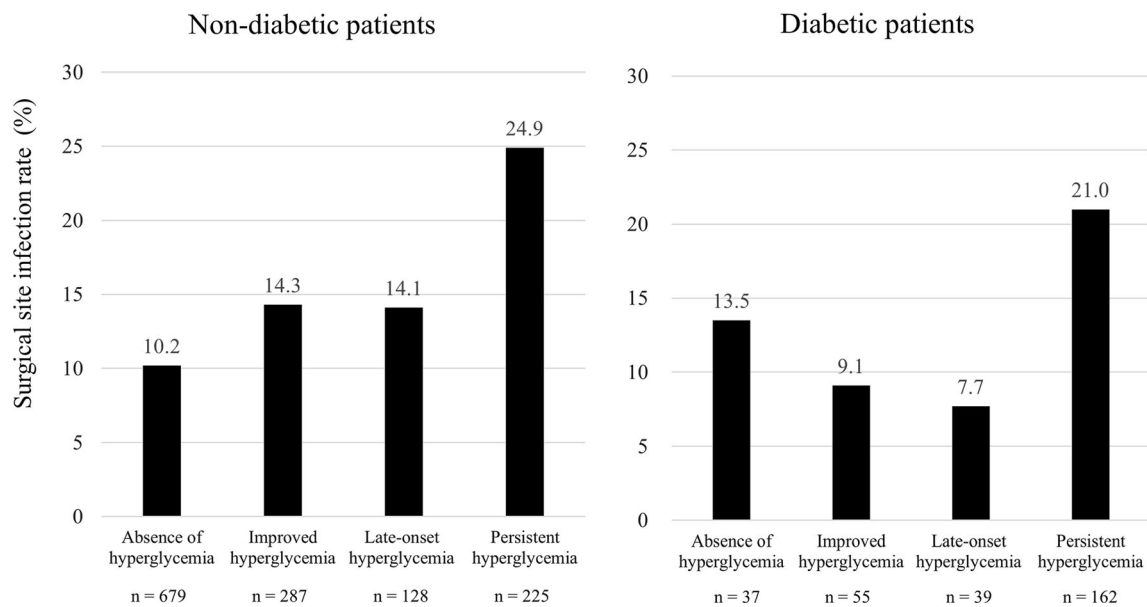


Fig. 1 Surgical site infection rates in non-diabetic patients and diabetic patients with improved hyperglycemia, with persistent hyperglycemia, with late-onset hyperglycemia, and the absence of hyperglycemia. In patients with and without diabetes, adjusted standardized residual was 2.6 and 5.2, respectively, in the persistent hyperglycemia group, -1.5 and 0.1 in the late-onset hyperglycemia group, -1.6 and 0.2 in the improved hyperglycemia group, and -0.4 and -4.1 in the absence of hyperglycemia group. Absolute values greater than the critical value (1.96) have *P* values of less than 0.05

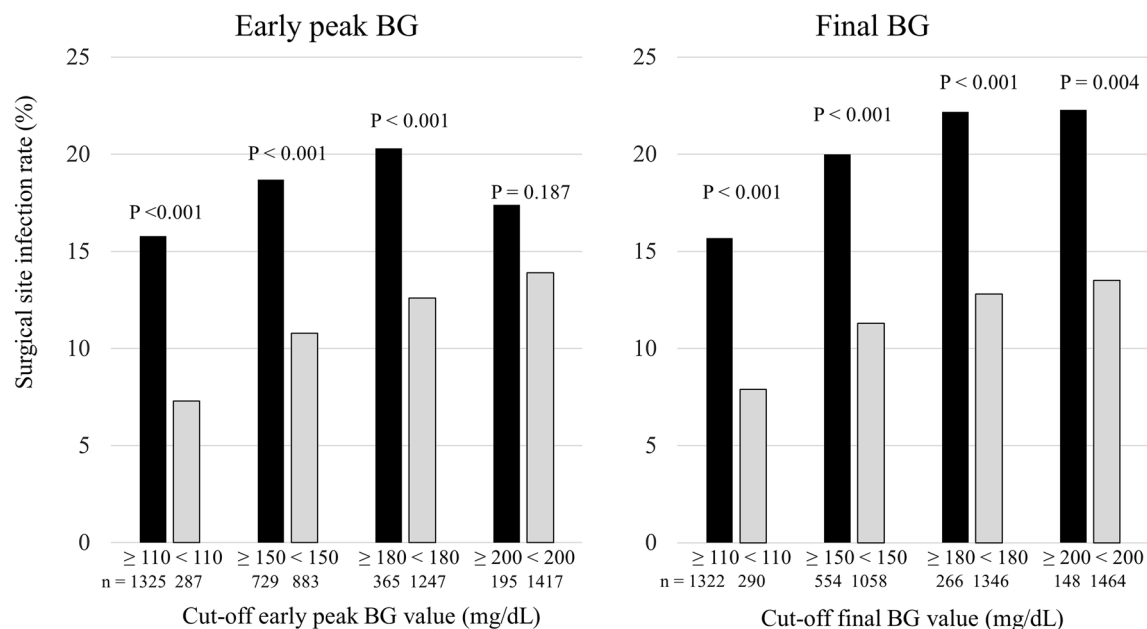


Fig. 2 Surgical site infection rates in patients with and without hyperglycemia defined by different cutoff values: comparison between peak blood glucose concentration (BG) within 12 h and final BG within 24 h after surgery. Black bar: below cutoff BG; gray bar: above cutoff BG

risks of SSI (13.2% for colorectal surgery; 6.9% for small intestine, 15.4% for gastric surgery; 32.4% for esophageal surgery; 21.8% for hepatobiliary pancreatic surgery; and 9.6% for other surgery). Therefore, the risks of SSI

according to category of BG control were further investigated for each surgical procedure (Table 3). Persistent hyperglycemia (OR 1.88, 95% CI 0.94–3.78, *P* = 0.072) in patients undergoing colorectal surgery, the absence of

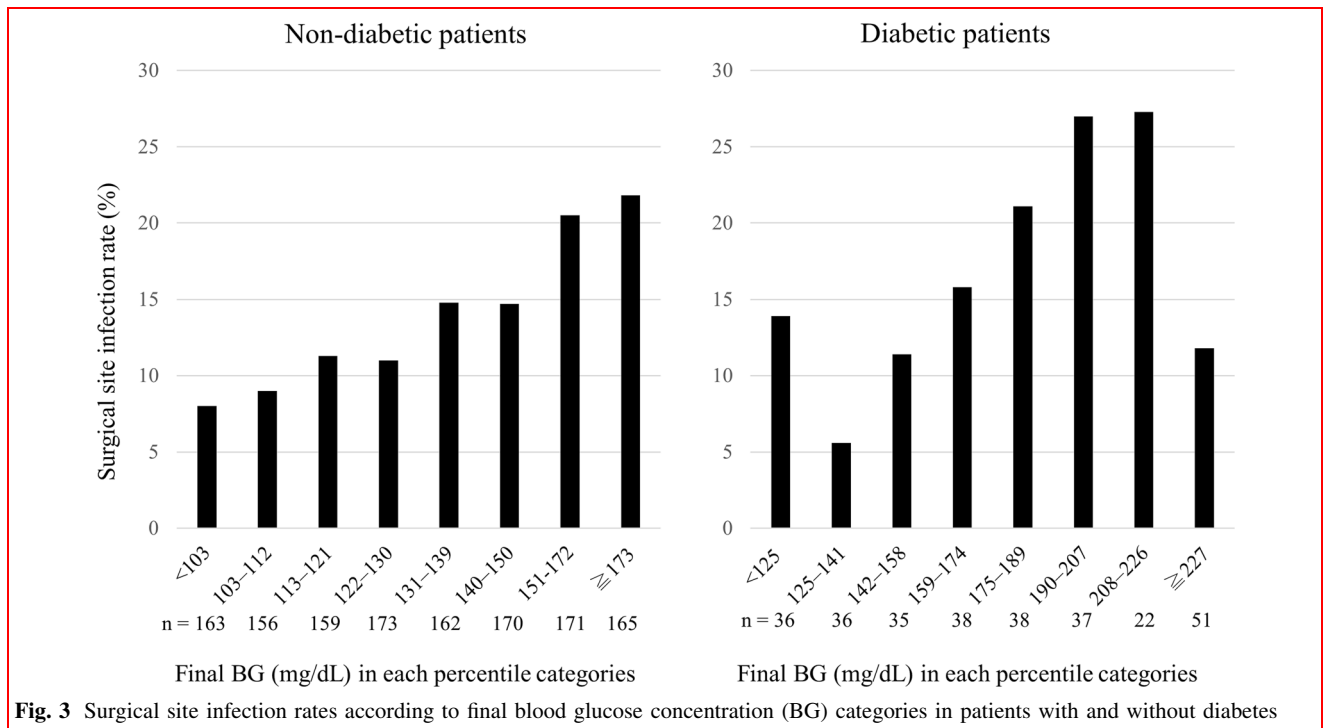


Fig. 3 Surgical site infection rates according to final blood glucose concentration (BG) categories in patients with and without diabetes

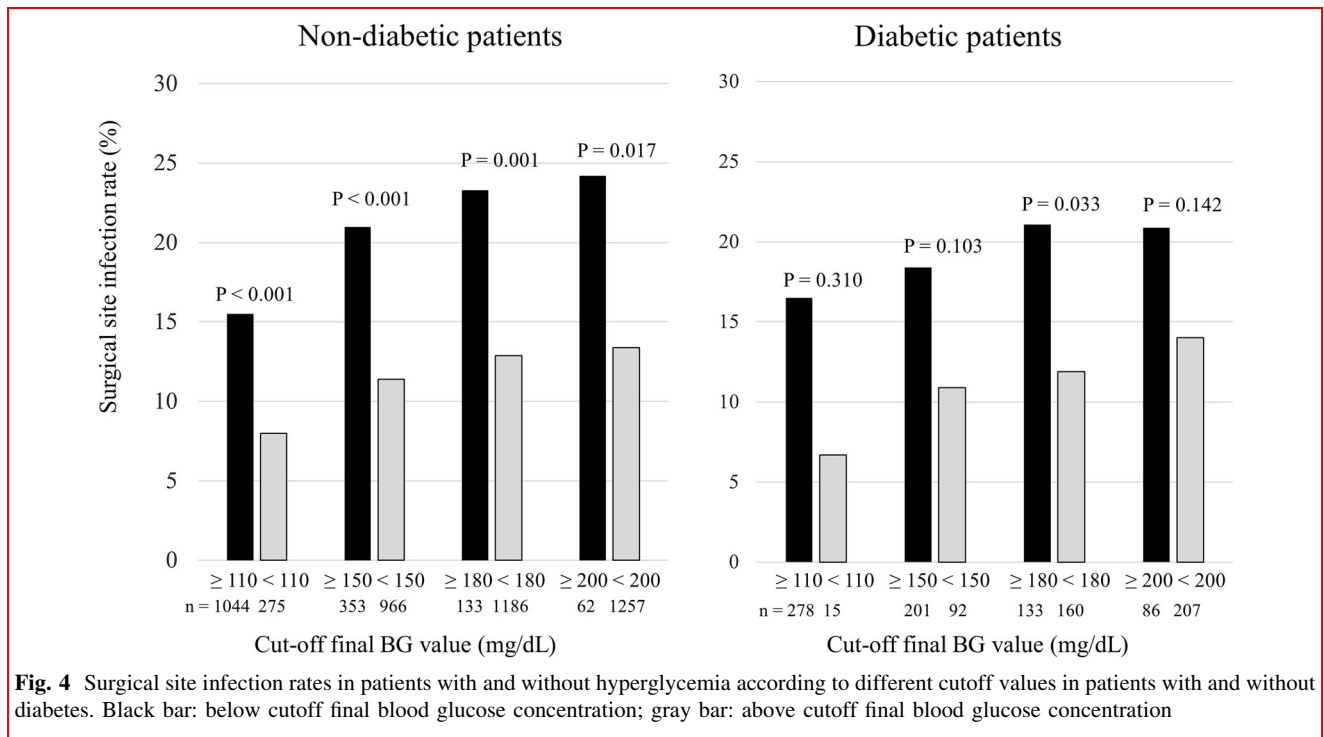


Fig. 4 Surgical site infection rates in patients with and without hyperglycemia according to different cutoff values in patients with and without diabetes. Black bar: below cutoff final blood glucose concentration; gray bar: above cutoff final blood glucose concentration

hyperglycemia (OR 0.39, 95% CI 0.15–0.99, $P = 0.042$) and late-onset hyperglycemia (OR 2.96, 95% CI 1.01–8.72, $P = 0.040$) in patients undergoing small intestine surgery, and persistent hyperglycemia (OR 4.14, 95% CI 1.94–8.83,

$P < 0.001$), late-onset hyperglycemia (OR 0.25, 95% CI 0.06–1.07, $P = 0.052$), and improved hyperglycemia (OR 0.18, 95% CI 0.04–0.78, $P = 0.011$) for those undergoing gastric surgery were selected by univariate analysis for

Table 1 Risk factors associated with surgical site infection in non-diabetic patients: univariate analyses

Factors	SSI, no of patients (%)		P value
	Yes	No	
<i>(a) Demographics and comorbidity</i>			
Male	119/829 (14.4)	65/490 (13.3)	0.581
Age \geq 65	98/558 (17.6)	86/761 (11.3)	0.001
Hypertension	53/304 (17.4)	131/1015 (12.9)	0.046
Chronic renal dysfunction	9/34 (26.5)	175/1285 (13.6)	0.043
Chronic hepatic dysfunction	30/175 (17.1)	154/1144 (13.5)	0.191
Cardiac disease	21/121 (17.4)	163/1198 (13.6)	0.257
ASA physical status classification \geq 3	42/227 (18.5)	142/1092 (13.0)	0.030
<i>(b) Preoperative factors</i>			
Smoking	71/476 (14.9)	113/843 (13.4)	0.447
Prolonged preoperative hospital stay	51/288 (17.7)	133/1031 (12.9)	0.037
Immunosuppressant	32/254 (12.6)	152/1065 (14.3)	0.489
Biological products	0/15 (0.0)	184/1304 (14.1)	0.249
Anti-cancer drug	8/30 (26.7)	176/1289 (13.7)	0.057
Preoperative serum albumin $<$ 3.0 g/dL	32/151 (21.2)	152/1168 (13.0)	0.006
Preoperative anemia	101/516 (19.6)	83/803 (10.3)	$<$ 0.001
Body mass index \geq 25	23/176 (13.1)	161/1143 (14.1)	0.890
Preoperative infection	43/89 (48.3)	141/1230 (11.5)	$<$ 0.001
Preoperative BG \geq 150 mg/dL	7/41 (17.1)	177/1278 (13.8)	0.558
<i>(c) Primary surgery</i>			
Colorectal surgery	64/485 (13.2)	120/834 (14.4)	0.547
Construction or closure of stoma	41/264 (15.5)	143/1055 (13.6)	0.407
Surgery for small intestine	21/304 (6.9)	163/1015 (16.1)	$<$ 0.001
Gastric surgery	34/221 (15.4)	150/1098 (13.7)	0.500
Esophageal surgery	12/37 (32.4)	172/1282 (13.4)	0.001
Hepatectomy without biliary tract resection	12/87 (13.8)	172/1232 (14.0)	0.965
Hepatectomy combined with biliary tract resection	16/36 (44.4)	168/1283 (13.1)	$<$ 0.001
Surgery for pancreas	11/33 (33.3)	173/1286 (13.5)	0.003
Cholecystectomy	1/45 (2.2)	183/1274 (14.4)	0.021
Other hepatobiliary pancreatic surgery	8/19 (42.1)	176/1300 (13.5)	0.002
Other surgery	5/52 (9.6)	179/1267 (14.1)	0.357
<i>(d) Intra- and postoperative factors</i>			
Wound class \geq 3	23/101 (22.8)	161/1218 (13.2)	0.008
Prolonged surgery	63/270 (23.3)	121/1049 (11.5)	$<$ 0.001
Emergent surgery	19/133 (14.3)	165/1186 (13.9)	0.906
Laparoscopic surgery	11/185 (5.9)	173/1134 (15.3)	0.001
Massive intraoperative bleeding	80/304 (26.3)	104/1015 (10.2)	$<$ 0.001
Perioperative blood transfusion	59/167 (35.3)	125/1152 (10.9)	$<$ 0.001
Intraoperative hypothermia ($<$ 36 °C)	36/243 (14.8)	148/1076 (13.8)	0.667
Admission to intensive care unit	34/136 (25.0)	150/1183 (12.7)	$<$ 0.001
Insulin treatment	4/16 (25.0%)	180/1303 (13.8)	0.263
<i>(e) Classification of blood glucose concentration control</i>			
Absence of hyperglycemia	69/679 (10.2)	115/640 (18.0)	$<$ 0.001
Improved hyperglycemia	41/287 (14.3)	143/1032 (13.9)	0.853
Persistent hyperglycemia	56/225 (24.9)	128/1094 (11.7)	$<$ 0.001
Late-onset hyperglycemia	18/128 (14.0)	166/1191 (13.9)	0.939

ASA American Society of Anesthesiologist

Table 2 Risk factors associated with surgical site infections in non-diabetic patients: multivariate analyses

Factors	Odds ratio	95% confidence interval
Persistent hyperglycemia	1.54	1.03–2.31
Prolonged surgery	1.81	1.23–2.65
Perioperative blood transfusion	2.36	1.56–3.57
Preoperative infections	5.04	3.10–8.19
Hepatectomy combined with biliary tract resection	2.79	1.31–5.96
Other hepatobiliary pancreatic surgeries	3.57	1.28–9.92
Small intestinal surgery	0.54	0.32–0.90
Laparoscopic surgery	0.51	0.26–1.00

multivariate analysis. Multivariate analysis identified persistent hyperglycemia as an independent risk factor for SSI in patients undergoing gastric surgery (OR 5.25, 95% CI 2.25–12.25, $P < 0.001$).

Discussion

It is commonly accepted that hyperglycemia affects the immune system via impaired chemotaxis, phagocytosis, overproduction of reactive oxygen species, free fatty acids, and inflammatory mediators [11]. These pathophysiologic changes also cause cellular damage and vascular dysfunction. Substantial evidence indicates that correction of hyperglycemia with insulin therapy reduces complications, including SSI, in patients undergoing cardiac and general surgery [1–5]. To determine the ability of BG control to prevent SSI, SSI rates in patients whose hyperglycemia improved were compared with those of patients with persistent hyperglycemia. In non-diabetic patients, the SSI rates increased in parallel with higher final BG categories. However, no correlation was found between hyperglycemia and SSI rates in patients with diabetes at each level of hyperglycemia. This result is consistent with other reports [12, 13]. In patients with diabetes, it is difficult to identify the target BG needed to prevent SSI; thus, the degree of BG control must be determined individually. Hence, in this study only the effect of hyperglycemia control in non-diabetic patients was evaluated.

Persistent hyperglycemia throughout the postoperative period (within 24 h) was associated with a risk of SSI in non-diabetic patients. Early-onset transient hyperglycemia within 12 h (improved hyperglycemia group) did not increase the SSI rate. In the subgroup analysis, persistent hyperglycemia was an independent risk factor in patients undergoing gastric surgery. These findings demonstrate the importance of hyperglycemia control in patients with an early-onset BG of ≥ 150 mg/dL. However, the risk of hypoglycemia should be considered in patients undergoing

intensive insulin therapy with a target BG of <150 mg/dL [1], especially patients admitted to the general surgical ward. Stress hyperglycemia typically resolves as surgical stress abates [6]. Approximately half of patients with an episode of early-onset hyperglycemia attained the target BG range within 24 h after surgery without insulin therapy. BG significantly decreased from 177.6 to 127.5 mg/dL without insulin use in patients with improved hyperglycemia. This raises the question of whether moderate-grade hyperglycemia such as a BG of ≥ 150 mg/dL should be treated with insulin in all patients undergoing surgery. Commencement of insulin dosing after confirmation of two consecutive BGs of ≥ 150 mg/dL or single BG of ≥ 180 – 200 mg/dL [14, 15] might be reasonable, especially in the non-surgical ICU setting. After starting insulin administration, the target BG should be set at <150 mg/dL.

This study had several limitations. First, it was a single-center, retrospective study. Second, there is a significant risk of bias as insulin treatment was not standardized, timing was variable, and the patient cohort/surgical interventions were heterogeneous. Third, the small number of surgical patients with diabetes in Japan might have contributed to the lack of a significant relationship between hyperglycemia and the SSI rate in patients with diabetes. Fourth, because glycemic control was dependent upon the attending physician's decisions, the performance rate of insulin treatment was low. Because of this, however, the natural course of postoperative hyperglycemia without insulin therapy was evaluated in this study. Finally, in our study the definition of stress hyperglycemia was not clear. HbA1c was not measured in patients without a history of diabetes unless they had hyperglycemia before surgery; thus, it was not possible to differentiate patients with stress hyperglycemia from those with diabetes that was not diagnosed before surgery. In conclusion, persistent hyperglycemia within 24 h after surgery is an independent risk factor for SSI, and remission of hyperglycemia until the morning after surgery is recommended in non-diabetic patients with postoperative hyperglycemia (≥ 150 mg/dL).

Table 3 Risk of surgical site infection according to blood glucose control category for each surgical procedure: univariate and multivariate analyses

Classification of blood glucose control	Colorectal surgery		Surgery for small intestine		Gastric surgery		Esophageal surgery		Hepatobiliary pancreatic surgery		Other surgery	
	OR 95% CI (<i>P</i> value)	Adjusted OR 95% CI (<i>P</i> value)	OR 95% CI (<i>P</i> value)	Adjusted OR 95% CI (<i>P</i> value)	OR 95% CI (<i>P</i> value)	Adjusted OR 95% CI (<i>P</i> value)	OR 95% CI (<i>P</i> value)	Adjusted OR 95% CI (<i>P</i> value)	OR 95% CI (<i>P</i> value)	Adjusted OR 95% CI (<i>P</i> value)	OR 95% CI (<i>P</i> value)	Adjusted OR 95% CI (<i>P</i> value)
Absence of hyperglycemia	0.69 0.41–1.17 (0.164)	–	0.39 0.15–0.99 (0.042)	1.13 0.27–4.67 (0.865)	1.05 0.47–2.35 (0.899)	–	2.44 0.41–14.47 (0.367)	–	0.55 0.27–1.13 (0.100)	–	0.34 0.05–2.27 (0.342)	–
Improved hyperglycemia	1.14 0.64–2.03 (0.663)	–	1.38 0.30–6.37 (0.656)	–	0.18 0.04–0.78 (0.011)	0.40 0.08–2.00 (0.267)	0.50 0.11–2.31 (0.476)	–	0.99 0.48–2.03 (0.973)	–	3.81 0.54–27.08 (0.202)	–
Persistent hyperglycemia	1.88 0.94–3.78 (0.072)	1.50 0.69–3.24 (0.303)	1.72 0.20–14.43 (0.480)	–	4.14 1.94–8.83 (<0.001)	5.25 2.25–12.25 (<0.001)	1.27 0.32–5.06 (0.732)	–	1.30 0.66–2.56 (0.445)	–	–	–
Late-onset hyperglycemia	0.87 0.30–2.55 (1.000)	–	2.96 1.01–8.72 (0.040)	1.39 0.41–4.69 (0.595)	0.25 0.06–1.07 (0.052)	0.41 0.08–2.17 (0.297)	–	–	2.59 0.87–7.67 (0.102)	–	2.69 0.24–30.19 (0.410)	–

OR odds ratio, 95% CI 95% confidence interval

Included variables (*P* < 0.1) in the adjusted model were chronic renal dysfunction, preoperative serum albumin ≤3.0 g/dL, preoperative anemia, preoperative infection, wound class ≥3, massive intraoperative bleeding, perioperative blood transfusion, construction or closure of stoma and persistent hyperglycemia in colorectal surgery; physical status classification ≥3, preoperative infection, wound class ≥3, prolonged surgery, construction or closure of stoma, the absence of hyperglycemia and late-onset hyperglycemia in surgery for small intestine; and age ≥65, chronic hepatic dysfunction, cardiac disease, physical status classification ≥3, prolonged preoperative hospital stay, preoperative infection, laparoscopic surgery, perioperative blood transfusion, the absence of hyperglycemia, persistent hyperglycemia, and late-onset hyperglycemia in gastric surgery

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Compliance with ethical standards

Conflict of interest All authors declare that they have no conflicts of interest.

Informed consent Because of retrospective study, informed consent was not obtained. This study was approved by the Institutional Review Board of Hyogo College of Medicine (No. 2284).

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