

Perioperative Glycemic Control and Its Effect on Surgical Wound Healing

Raghunath Prasad¹, Narendra Kumar², Ambar Saxena³¹Assistant Professor, Department of General Medicine, Shri Kalyan Government Medical College and Attached Hospitals, Sikar, Rajasthan, India²Assistant Professor, Department of General Medicine, Shri Kalyan Government Medical College and Attached Hospitals, Sikar, Rajasthan, India³Assistant Professor, Department of General Surgery, Shri Kalyan Government Medical College and Attached Hospitals, Sikar, Rajasthan, India

Received: 01-05-2026 / Revised: 15-06-2026 / Accepted: 21-07-2026

Corresponding author: Dr. Ambar Saxena

Conflict of interest: Nil

Abstract

Background: Hyperglycemia during perioperative stress is a common metabolic consequence of surgical stress and has been associated with infectious complications and impairment in tissue repair. Higher glucose levels may hinder neutrophil function, endothelial response, collagen formation, and microvascular perfusion in the wound, ultimately affecting wound healing. In this study, perioperative glycemic control was assessed for SSI, delayed wound healing, wound dehiscence, and postoperative recovery after major abdominal surgery.

Methods: A prospective observational cohort study enrolled 260 adults who were undergoing abdominal surgery. Blood glucose was measured preoperatively, intraoperatively and 48 hours postoperatively. Patients were classified as having adequate perioperative glycemic control when glucose was <180 mg/dL and hyperglycemic when one or more clinically relevant perioperative measurements were ≥ 180 mg/dL. The surgical wounds were assessed for 30 days. The primary outcome was SSI, clinically significant wound dehiscence or delayed wound healing. Multivariable logistic regression was used to identify independent predictors.

Results: Of the 260 participants, 178 (68.5%) had glucose <180 mg/dL and 82 (31.5%) developed perioperative hyperglycemia. SSI was reported in 6.7% of the patients with properly controlled glucose and 20.7% of patients with hyperglycemic conditions ($p=0.001$). Time to heal was 9.6% compared to 24.4% ($p=0.002$), and wound dehiscence occurred in 3.4% compared to 9.8% ($p=0.036$). The wound complication rate was 14.6% compared to 36.6% ($p<0.001$). After accounting for this, glucose ≥ 180 mg/dL was still associated with wound complications independently (adjusted odds ratio 2.85; 95% CI 1.48-5.49; $p=0.002$).

Conclusion: Perioperative hyperglycemia was associated with poorer surgical wound outcomes and longer recovery. Structured glucose surveillance and safe correction of significant hyperglycemia may be an important modifiable component of perioperative wound-protection strategies.

Keywords: Perioperative Hyperglycemia; Glycemic Control; Surgical-Site Infection; Wound Healing; Diabetes Mellitus; Postoperative Complications.

DOI: 10.25258/ijcpr.18.8.310

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Surgical wound healing is a complex biological process that takes place through hemostasis, inflammation, cellular proliferation, angiogenesis, extracellular matrix deposition, epithelialization, and remodeling. The successful repair is dependent on local perfusion, oxygen availability, nutritional status, bacterial burden, surgical techniques, and metabolic environment. Hyperglycemia is especially important since it is common in surgery and is present in both patients with diabetes and those who have no prior diagnosis. The operative stress can activate the sympathetic nervous system

and hypothalamic-pituitary-adrenal axis and increase catecholamines, cortisol, glucagon, and inflammatory mediators. There have been clinical studies that link this metabolic response to postoperative morbidity. Frisch et al. reported that perioperative hyperglycemia in noncardiac surgery was associated with infectious complications, longer hospital and intensive care stays, renal dysfunction, and mortality [2]. In a large multicenter study, Kwon et al. showed that perioperative glucose levels above 180 mg/dL were associated with higher incidence of postoperative

infection and adverse outcomes and that appropriate insulin treatment mitigated some of these risks [3]. Ata et al. also found postoperative glucose elevation to be the major predictor of SSI in general surgical patients and showed a particularly clear relationship after colorectal procedures [4]. These findings suggest that perioperative glucose may provide prognostic information not only for diabetes diagnosis but for other diseases too.

The biological mechanisms are realistic and clinical implications are important. Hyperglycemia may reduce neutrophil chemotaxis, phagocytosis and intracellular killing; lead to oxidative stress and endothelial dysfunction; and may compromise capillary perfusion and tissue oxygenation. In parallel, prolonged metabolic dysregulation may affect fibroblast activity, collagen synthesis, matrix organization & epithelial repair. The surgical wound could be more susceptible to bacterial colonization and lose cellular and structural resources to heal as expected. And this mechanistic overlap is why dysglycemia may manifest as SSI, delayed epithelialization, poor tensile strength and wound separation rather than as infection alone.

Perioperative glucose management is thus far the key to SSI prevention. The Centers for Disease Control and Prevention recommend intraoperative glycemic control with blood glucose below 200 mg/dL [5]. The American Diabetes Association and Endocrine Society also recommend structured inpatient glucose monitoring and treatment for clinically significant hyperglycemia while avoiding unnecessary hypoglycemia [6,7]. Research from randomized trials and meta-analyses show that lower perioperative glucose targets may decrease SSI. The same is true for perioperative glucose monitoring and treatment, which may greatly increase hypoglycemia in critically ill patients [8]. So the goal is not just to normalize glucose in general but to prevent long-term or severe hyperglycemia in a safe treatment range. The present study investigated the relationship between perioperative glycemic control and early surgical wound healing in adults undergoing major abdominal surgery.

The primary objective was to compare the incidence of composite wound healing complications among patients that had perioperative glucose <180 mg/dL and patients with glucose $\geq$ 180 mg/dL. Secondary goals were to compare SSI, delayed wound healing, wound dehiscence, time to satisfactory healing, postoperative length of stay, and independent clinical predictors of adverse wound outcomes.

Materials and Methods

Study design and setting: A prospective observational cohort study was conducted in consecutive adult patients who received major abdominal surgery and required postoperative inpatient observation. The patients were prospectively screened and enrolled according to predefined inclusion and exclusion criteria. The study design allowed to systematically monitor perioperative glycemic exposure and follow up postoperative wound outcomes without altering standard therapy decisions.

Participants: People age 18 years or older who had major abdominal surgery in an elective or urgent manner were included. People with and without diabetes mellitus were included. The patients were excluded if they had diabetic ketoacidosis or hyperosmolar hyperglycemic state, active infection at the intended operative site, established sepsis before surgery, pregnancy, long-term systemic corticosteroid or powerful immunosuppressive therapy, severe neutropenia, terminal illness, or poor postoperative follow up. Procedures that did not have a traditional surgical incision and reoperation within 48 hours for unrelated technical complications were excluded.

Perioperative assessment and glucose monitoring: At the planning stage, age, sex, body mass index (BMI), smoking status, diabetes history, antihyperglycemic treatment, comorbidities, American Society of Anesthesiologists physical status, operative indication, operative duration, blood loss, and transfusion requirement were recorded. Hemoglobin (HbA1c) was measured preoperatively if clinically available. Blood glucose was measured before surgery, during induction, at intraoperative intervals, on arrival in the post-anesthesia care unit (around 6 hours after surgery), and then based on clinical status in the first 48 postoperative hours.

Definition of glycemic exposure: We classified patients prior to the main analysis. Good perioperative glycemic control was defined as glucose below 180 mg/dL in the monitored perioperative period. Perioperative hyperglycemia was defined as one or more clinically relevant values of 180 mg/dL or more. The 180 mg/dL threshold is a common value in both inpatient and perioperative care to determine clinically relevant hyperglycemia and is not the likely point for very intensive glucose normalization. Insulin and nutritional management were selected by the treating clinical team to follow practice of the clinical team.

Wound assessment and study outcomes: The surgical wounds were examined in their hospitalization and follow up at around postoperative days 7, 14, and 30. SSI was classified according to accepted CDC definitions.

Investigators observed erythema, purulent or serous discharge, wound separation, antibiotic escalation, drainage, debridement, and secondary closure. Delayed wound healing was defined as not showing sufficient epithelialization or clinically appropriate progression of wound closure at postoperative day 14 in a wound that was intended for primary healing. The main outcome was a combination of SSI, clinically significant wound dehiscence, and delayed wound healing within 30 days. Secondary outcomes were the individual wound endpoints, wound-related reintervention, time to satisfactory healing, reported hypoglycemia below 70 mg/dL, and postoperative hospital stay.

Statistical Analysis: Continuous variables were reported as mean $\pm$ standard deviation or median and quartile distribution. Categorical variables were reported as frequencies and percentages. Between groups were compared using independent samples t test or Mann-Whitney U test for continuous and chi-square test or Fisher exact test for categorical data. All clinically relevant variables and factors related to wound complications on univariable analysis were included in multivariable logistic regression. Adjusted odds ratios (aORs) were reported with 95% confidence intervals. We tested the model collinearity and calibration before we could interpret it. A two-sided p value < 0.05 was considered statistically significant.

Results

We have reported on 260 patients who were observed in perioperative glucose monitoring and 30 days of wound assessment. 178 of them (68.5%) had glucose levels below 180 mg/dL and 82

(31.5%) developed perioperative hyperglycemia. Those in the hyperglycemic group were more likely to have diabetes and had higher BMI and HbA1c. The wound contamination was similar across age, sex, smoking status, and wound contamination. Hyperglycemia was observed in a metabolically higher risk population but not only for patients with a prior diagnosis of diabetes.

Glycemic differences between groups were visible prior to surgery and more pronounced intraoperatively and in the early postoperative period. Hyperglycemic patients had higher peak intraoperative glucose, higher 6-hour postoperative glucose and a higher mean glucose in the first 24 hours. Glycemic variability was also greater. Documented hypoglycemia was uncommon and was similar across groups. The wound differences did not, however, suggest a significantly higher percentage of treatment-related low glucose in the well-controlled group.

Wound events were also consistent with glucose ≥ 180 mg/dL. SSI was reported in 20.7% of hyperglycemic patients and 6.7% in well-controlled patients. Delayed healing occurred in 24.4% as compared to 9.6% and wound dehiscence in 9.8% as opposed to 3.4%. The overall wound complication rate was 36.6% in the hyperglycemic group compared to 14.6% in the controlled group. Hyperglycemia was associated with longer times to satisfactory healing of wounds and longer postoperative hospitalization. Multivariable analysis confirmed perioperative glucose ≥ 180 mg/dL as an independent predictor of wound complications after adjusting for metabolic and operative covariates.

Table 1: Baseline demographic and perioperative characteristics

Variable	Glucose <180 mg/dL (n=178)	Glucose ≥ 180 mg/dL (n=82)	p value
Age, years, mean $\pm$ SD	54.6 $\pm$ 13.4	56.2 $\pm$ 12.8	0.36
Male sex, n (%)	98 (55.1)	47 (57.3)	0.74
Diabetes mellitus, n (%)	47 (26.4)	45 (54.9)	<0.001
BMI, kg/m ² , mean $\pm$ SD	26.8 $\pm$ 4.1	28.9 $\pm$ 5.2	0.004
HbA1c, %, mean $\pm$ SD	6.4 $\pm$ 1.0	7.6 $\pm$ 1.5	<0.001
ASA physical status III, n (%)	44 (24.7)	31 (37.8)	0.030
Current smoking, n (%)	53 (29.8)	27 (32.9)	0.61
Contaminated wound class, n (%)	22 (12.4)	15 (18.3)	0.20
Operative duration >120 min, n (%)	63 (35.4)	39 (47.6)	0.061

The baseline characteristics of perioperative hyperglycemia were consistent with a well-established metabolic risk. Diabetes prevalence, BMI, HbA1c and ASA III were all higher in patients with glucose ≥ 180 mg/dL, while age, sex, smoking and wound contamination were not

significantly different. This indicates that glycemic elevation occurred in a more vulnerable subgroup but the relative similarity of some operative variables led to further adjustments to determine if glucose itself was still associated with impaired wound healing.

Table 2: Perioperative glycemic profile

Glycemic parameter	Glucose <180 mg/dL	Glucose ≥180 mg/dL	p value
Preoperative glucose, mg/dL	126±22	171±41	<0.001
Maximum intraoperative glucose, mg/dL	153±18	214±31	<0.001
6-hour postoperative glucose, mg/dL	145±21	208±36	<0.001
Mean 24-hour postoperative glucose, mg/dL	137±19	196±29	<0.001
Glycemic coefficient of variation, %	18±7	27±10	<0.001
Glucose <70 mg/dL, n (%)	9 (5.1)	6 (7.3)	0.47

Hyperglycemic patients, however, exhibited sustained glucose levels from preoperative to first day of postoperative rather than a temporary increase. Higher mean values were associated with larger variability, indicating a metabolically unstable perioperative profile. Hypoglycemia was

not common and did not significantly differ between groups. These findings suggest that long-term glucose exposure and variability should be monitored in perioperative blood glucose treatment rather than one admission level for the risk of wound complications.

Table 3: Surgical wound and postoperative outcomes

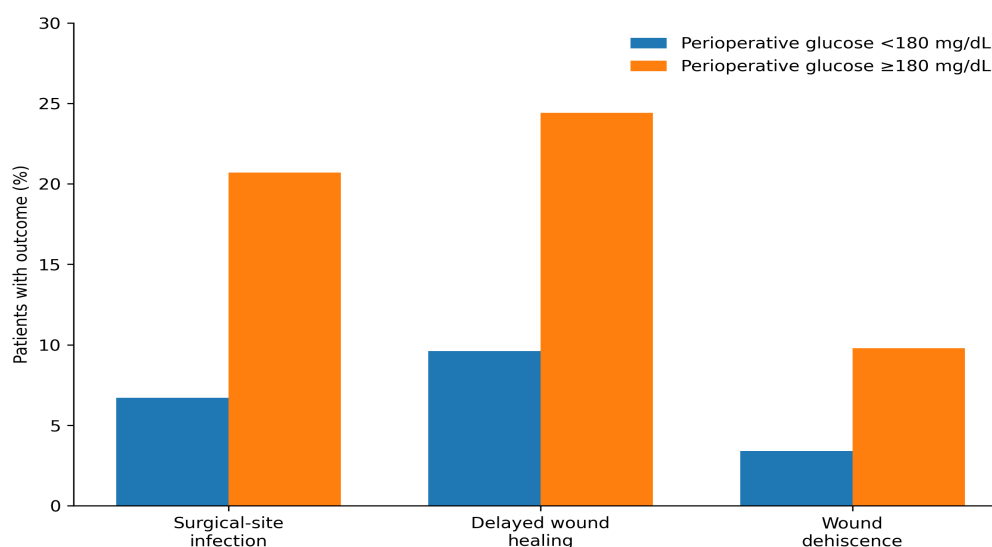
Outcome	Glucose <180 mg/dL (n=178)	Glucose ≥180 mg/dL (n=82)	p value
Surgical-site infection	12 (6.7%)	17 (20.7%)	0.001
Delayed wound healing	17 (9.6%)	20 (24.4%)	0.002
Wound dehiscence	6 (3.4%)	8 (9.8%)	0.036
Seroma requiring intervention	10 (5.6%)	9 (11.0%)	0.12
Wound-related reintervention	5 (2.8%)	6 (7.3%)	0.10
Composite wound complication	26 (14.6%)	30 (36.6%)	<0.001
Time to satisfactory healing, days, median (IQR)	11 (10–13)	15 (12–19)	<0.001
Postoperative hospital stay, days, median (IQR)	5 (4–7)	7 (5–10)	<0.001

Perioperative glucose ≥180 mg/dL was associated with clinically significant deterioration in different aspects of wound recovery.

SSI was more than three times as frequent and delayed healing and wound dehiscence were also much higher. This was reflected in a substantially

higher composite wound-complication rate and approximately four more days to satisfactory healing.

This indicates that dysglycemia affects infectious and reparative aspects of wound healing, and can lead to longer postoperative hospitalization.

**Figure 1: Surgical Wound Outcomes According To Perioperative Glycemic Control**

The graphic comparison shows that there is an overall excess of adverse wound outcomes in patients with perioperative glucose ≥180 mg/dL. The greatest absolute differences were for surgical-

site infection and delayed wound healing while wound dehiscence showed the same direction of association despite being less frequent. The same increases across different wound endpoints suggest

that the effects of infection are not only infectious in nature and that postoperative tissue repair is

worse in the case of clinically significant perioperative hyperglycemia.

Table 4: Multivariable predictors of composite wound-healing complications

Predictor	Adjusted OR	95% CI	p value
Perioperative glucose ≥ 180 mg/dL	2.85	1.48–5.49	0.002
Contaminated wound class	2.41	1.21–4.80	0.012
Operative duration >120 min	1.89	1.04–3.45	0.038
HbA1c $\geq 8.0\%$	1.72	0.91–3.26	0.096
BMI ≥ 30 kg/m ²	1.62	0.88–2.99	0.120
Current smoking	1.39	0.75–2.59	0.290
Established diabetes diagnosis	1.18	0.60–2.32	0.630

After adjusting for metabolic and operative factors, perioperative glucose ≥ 180 mg/dL was the strongest independent predictor of wound complications with nearly three times higher adjusted odds.

Contaminated wounds and prolonged operations were also independently associated with adverse

healing. However, diabetes diagnosis itself was not significant in the model when perioperative glucose was taken into account.

This suggests that the immediate glycemic environment may be more predictive for early wound recovery than the diagnosis of diabetes alone.

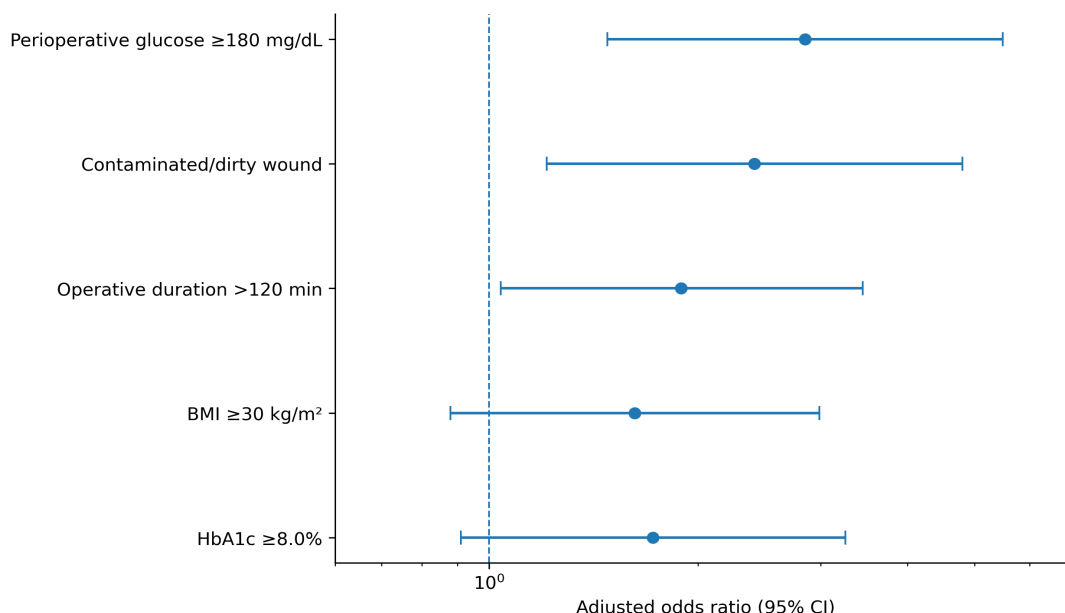


Figure 2: Adjusted Predictors of Postoperative Wound-Healing Complications

The forest plot shows the relative contribution of clinically relevant predictors after adjustment. Perioperative hyperglycemia was the greatest independent indicator of wound complications, followed by contaminated wound class and longer operative duration.

Although high HbA1c and obesity were estimated to be above unity, their confidence intervals crossed one. These findings suggest a multifactorial model of wound failure and suggest that perioperative glucose elevation is a more modifiable exposure to prognostic outcomes that is more relevant than a set of baseline characteristics.

Discussion

This study showed a clear association of perioperative hyperglycemia with impaired surgical wound healing. Patients with glucose >180 mg/dL also had more SSI, delayed healing and wound dehiscence and it took longer for closure of the wound to occur. The association was confirmed with diabetes, BMI, wound contamination, operative period and other covariates adjusted. These findings are consistent with Frisch et al. who linked perioperative hyperglycemia to infectious morbidity and adverse outcomes after noncardiac surgery [2], and multicenter analysis of Kwon et al., who found that glucose >180 mg/dL was associated with an increased adjusted infection risk [3]. Similarly, postoperative glucose elevation was a major predictor of SSI, especially after colorectal

procedures [4]. The wound-specific pattern is supported by several other studies. Bellusse et al. reported that hyperglycemia at the end of abdominal surgery and early postoperative period independently increased SSI risk [11].

McConnell et al. found significantly higher SSI rates in diabetic colorectal patients with mean postoperative glucose >200 mg/dL [12]. Kotagal et al. showed a dose-response relationship between perioperative hyperglycemia and adverse events, even in patients who did not have diabetes [13]. Richards et al. also found higher SSI rates in hyperglycemic nondiabetic orthopedic trauma patients [19]. These studies suggest stress hyperglycemia is not to be dismissed just because diabetes is absent.

This is biologically linked with host defense and tissue repair. Hyperglycemia impairs neutrophil chemotaxis, phagocytosis and intracellular bacterial killing, compresses oxidative stress; changes endothelial nitric oxide signaling; and can impair microvascular perfusion at the wound level. At the wound level, it may be associated with reduced fibroblast efficiency and disordered collagen deposition and impaired angiogenesis and slower epithelialization. As such, the fact that delayed healing and dehiscence were also seen as a result of SSI is biologically consistent and supports dysglycemia as the cause of wound integrity and not infection risk alone.

More evidence for a causal effect is available in the literature, but the optimal therapeutic target is far from settled. Umpierrez et al. performed the RABBIT 2 Surgery randomized study and found that basal-bolus insulin increased glycemic control and decreased postoperative complications compared to sliding-scale insulin in general surgical patients with type 2 diabetes [14]. Furnary et al. showed that constant intravenous insulin reduced deep sternal wound infection after cardiac surgery [15] and Ogawa et al. So later on, fewer deep sternal wound infections and reduced glucose variability were observed with continuous postoperative insulin infusion [16]. Meta-analyses by de Vries et al., Wang et al. and Lai et al. showed that low perioperative glucose targets were associated with SSI or postoperative infection but hypoglycemia was higher with more intensive interventions [8–10].

The counterpoint is just as important; the Cochrane review also found very intensive perioperative glucose control to be uncertain and warned of increased hypoglycemia [17]. The NICE-SUGAR trial in critically ill adults showed that targeting near-normal glucose resulted in severe hypoglycemia and 90-day mortality compared to a standard target of glucose up to 180 mg/dL [20]. Current advice is therefore for prevention of

clinically significant hyperglycemia rather than aggressive normalization. CDC recommendations have intraoperative glucose below 200 mg/dL [5], while recent ADA and Endocrine Society guidance focuses on monitoring and avoidance of sustained hyperglycemia and iatrogenic hypoglycemia [6, 7].

Preoperative HbA1c may indicate chronic metabolic risk, but is not a substitute for perioperative monitoring. Wong et al. found higher HbA1c in the cases of wound infection and major complications in abdominal surgery [18]. HbA1c $\geq 8\%$ had a higher point estimate but lost statistical significance after perioperative glucose was added, while glucose ≥ 180 mg/dL was separately predictive. The results show that chronic glycemic exposure and acute perioperative dysglycemia are indeed complementary, and immediate glucose levels may reflect the biological environment that may be most relevant during tissue injury, bacterial inoculation, and early repair.

The study had flaws and we could not determine causality using the observational method. Nutritional status, perfusion, anemia, antibiotic timing, surgical complexity, and surgeon factors could have continued to be confounding factors even after many adjustments. A cutoff of 180 mg/dL for classification simplified monitoring of the wound and intermittent tests may have missed short-term excursions. Finally, we were not able to infer treatment-specificity from the heterogeneous abdominal surgical population. In future multicenter studies, standardized wound assessment with continuous or high-frequency glucose monitoring, glycemic variability, and comparison of moderate treatment should be included. Such work could help to determine whether decreasing the duration and variability of clinically significant hyperglycemia facilitates wound healing without increasing hypoglycemia.

Conclusion

Perioperative hyperglycemia was associated with significantly worse surgical wound outcomes, such as more surgical-site infection, delayed healing, wound dehiscence, longer time to satisfactory wound recovery, and long hospitalization.

After controlling for metabolic and operative factors, glucose ≥ 180 mg/dL was independently associated with wound complications, and diabetes diagnosis alone did not predict perioperative glucose. These results suggest that glucose surveillance should be ongoing for patients with and without diabetes. Safe prevention and timely correction of serious perioperative hyperglycemia could be a fundamental and practical component of surgical wound protection efforts.

References

1. Shuford, R., & Miller-Ocuin, J. L. (2023). Hyperglycemia in the perioperative period. *Clinics in Colon and Rectal Surgery*, 36(3), 198–200. <https://doi.org/10.1055/s-0043-1761153>
2. Frisch, A., Chandra, P., Smiley, D., Peng, L., Rizzo, M., Gatliffe, C., Hudson, M., Mendoza, J., Johnson, R., Lin, E., & Umpierrez, G. E. (2010). Prevalence and clinical outcome of hyperglycemia in the perioperative period in noncardiac surgery. *Diabetes Care*, 33(8), 1783–1788. <https://doi.org/10.2337/dc10-0304>
3. Kwon, S., Thompson, R., Dellinger, P., Yanez, D., Farrohi, E., & Flum, D. (2013). Importance of perioperative glycemic control in general surgery: A report from the Surgical Care and Outcomes Assessment Program. *Annals of Surgery*, 257(1), 8–14. <https://doi.org/10.1097/SLA.0b013e31827b6bbc>
4. Ata, A., Lee, J., Bestle, S. L., Desemone, J., & Stain, S. C. (2010). Postoperative hyperglycemia and surgical site infection in general surgery patients. *Archives of Surgery*, 145(9), 858–864. <https://doi.org/10.1001/archsurg.2010.179>
5. Berríos-Torres, S. I., Umscheid, C. A., Bratzler, D. W., et al. (2017). Centers for Disease Control and Prevention guideline for the prevention of surgical site infection, 2017. *JAMA Surgery*, 152(8), 784–791. <https://doi.org/10.1001/jamasurg.2017.0904>
6. American Diabetes Association Professional Practice Committee for Diabetes. (2026). 16. Diabetes care in the hospital: Standards of Care in Diabetes—2026. *Diabetes Care*, 49(Supplement 1), S339–S355. <https://doi.org/10.2337/dc26-S016>
7. Korytkowski, M. T., Muniyappa, R., Antinori-Lent, K., et al. (2022). Management of hyperglycemia in hospitalized adult patients in non-critical care settings: An Endocrine Society clinical practice guideline. *Journal of Clinical Endocrinology & Metabolism*, 107(8), 2101–2128. <https://doi.org/10.1210/clinem/dgac278>
8. Lai, J., Li, Q., He, Y., Zou, S., Bai, X., & Rastogi, S. (2022). Glycemic control regimens in the prevention of surgical site infections: A meta-analysis of randomized clinical trials. *Frontiers in Surgery*, 9, 855409. <https://doi.org/10.3389/fsurg.2022.855409>
9. de Vries, F. E. E., Gans, S. L., Solomkin, J. S., Allegranzi, B., Egger, M., Dellinger, E. P., & Boerma, M. A. (2017). Meta-analysis of lower perioperative blood glucose target levels for reduction of surgical-site infection. *British Journal of Surgery*, 104(2), e95–e105. <https://doi.org/10.1002/bjs.10424>
10. Wang, Y. Y., Hu, S. F., Ying, H. M., Chen, L., Li, H. L., Tian, F., & Zhou, Z. F. (2018). Postoperative tight glycemic control significantly reduces postoperative infection rates in patients undergoing surgery: A meta-analysis. *BMC Endocrine Disorders*, 18(1), 42. <https://doi.org/10.1186/s12902-018-0268-9>
11. Bellusse, G. C., Ribeiro, J. C., de Freitas, I. C. M., & Galvão, C. M. (2020). Effect of perioperative hyperglycemia on surgical site infection in abdominal surgery: A prospective cohort study. *American Journal of Infection Control*, 48(7), 781–785. <https://doi.org/10.1016/j.ajic.2019.11.009>
12. McConnell, Y. J., Johnson, P. M., & Porter, G. A. (2009). Surgical site infections following colorectal surgery in patients with diabetes: Association with postoperative hyperglycemia. *Journal of Gastrointestinal Surgery*, 13(3), 508–515. <https://doi.org/10.1007/s11605-008-0734-1>
13. Kotagal, M., Symons, R. G., Hirsch, I. B., Umpierrez, G. E., Dellinger, E. P., Farrohi, E. T., & Flum, D. R. (2015). Perioperative hyperglycemia and risk of adverse events among patients with and without diabetes. *Annals of Surgery*, 261(1), 97–103. <https://doi.org/10.1097/SLA.0000000000000688>
14. Umpierrez, G. E., Smiley, D., Jacobs, S., et al. (2011). Randomized study of basal-bolus insulin therapy in the inpatient management of patients with type 2 diabetes undergoing general surgery (RABBIT 2 Surgery). *Diabetes Care*, 34(2), 256–261. <https://doi.org/10.2337/dc10-1407>
15. Furnary, A. P., Zerr, K. J., Grunkemeier, G. L., & Starr, A. (1999). Continuous intravenous insulin infusion reduces the incidence of deep sternal wound infection in diabetic patients after cardiac surgical procedures. *The Annals of Thoracic Surgery*, 67(2), 352–360. [https://doi.org/10.1016/S0003-4975\(99\)00014-4](https://doi.org/10.1016/S0003-4975(99)00014-4)
16. Ogawa, S., Okawa, Y., Sawada, K., et al. (2016). Continuous postoperative insulin infusion reduces deep sternal wound infection in patients with diabetes undergoing coronary artery bypass grafting using bilateral internal mammary artery grafts: A propensity-matched analysis. *European Journal of Cardio-Thoracic Surgery*, 49(2), 420–426. <https://doi.org/10.1093/ejcts/ezv106>
17. Bellon, F., Solà, I., Gimenez-Perez, G., Hernández, M., Metzendorf, M. I., Rubinat, E., & Mauricio, D. (2023). Perioperative glycaemic control for people with diabetes undergoing surgery. *Cochrane Database of Systematic Reviews*, 2023(8), CD007315. <https://doi.org/10.1002/14651858.CD007315.pub3>
18. Wong, J. K. L., Ke, Y., Ong, Y. J., Li, H., Wong, T. H., & Abdullah, H. R. (2022). The impact of preoperative glycated hemoglobin

- (HbA1c) on postoperative complications after elective major abdominal surgery: A meta-analysis. *Korean Journal of Anesthesiology*, 75(1), 47–60. <https://doi.org/10.4097/kja.21295>
19. Richards, J. E., Hutchinson, J., Mukherjee, K., Jahangir, A. A., Mir, H. R., Evans, J. M., Perdue, A. M., Obremskey, W. T., Sethi, M. K., & May, A. K. (2014). Stress hyperglycemia and surgical site infection in stable nondiabetic adults with orthopedic injuries. *Journal of Trauma and Acute Care Surgery*, 76(4), 1070–1075. <https://doi.org/10.1097/TA.0000000000000177>
20. NICE-SUGAR Study Investigators. (2009). Intensive versus conventional glucose control in critically ill patients. *The New England Journal of Medicine*, 360(13), 1283–1297. <https://doi.org/10.1056/NEJMoa0810625>