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ORIGINAL SCIENTIFIC REPORT

Post-operative Glycaemic Control Using an Insulin Infusion is Associated with Reduced Surgical Site Infections in Colorectal Surgery

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Abstract

Background The incidence of surgical site infection (SSI) in colorectal surgery (CRS) is higher than other forms of general surgery. Post-operative hyperglycaemia causes increased SSI in CRS. Post-operative hyperglycaemia control in cardiac surgery reduces SSI. The aim was to evaluate using a cohort comparison the effect of post-operative glycaemic control using an insulin infusion on SSI in CRS.

Methods Collection of data for the ACS-NSQIP was commenced in 2015. The CRS unit added post-operative glycaemic control to the SSI bundle in late 2016. The intervention was an insulin infusion to titrate blood glucose between 135 and 180 mg/Dl (7.5 and 10 mmol/l). The effect of glycaemic control on SSI was assessed comparing ACS-NSQIP raw data prior and after the intervention was commenced.

Results The NSQIP data from July 2015 to June 2016 revealed the incidence of SSI were 25%. From January 2017 to December 2017, there was a significant reduction in SSI to 6.1% (OR = 517 Cl = 1.92–16.08, $p < 0.001$). The incidence of organ/space SSI fell significantly from 13% to 1.0% (OR = 11.35, Cl = 1.62–488.7, $p < 0.001$). There was non-significant reduction in superficial SSI from 11 to 4.0% (OR = 2.93, Cl = 0.68–13.03, $p = 0.06$). There was no significant difference in other factors associated with SSI in CRS.

Conclusion Post-operative glycaemic control in CRS reduces the rate of SSI. Post-operative glycaemic control should be included in SSI bundles for CRS and may be of benefit in other surgical specialties.

Introduction

Surgical site infections (SSI) are frequent in colorectal surgery (CRS) [1, 2] with a higher incidence than other forms of general surgery [3, 4]. SSI is associated with increased length of stay (LOS), readmission rates, re-operation rates, costs [5–7] and increased impairment of physical and mental well-being [8].

Factors associated with increased risk of SSI in CRS are: emergency CRS [9], obesity [2, 5, 9, 10], advanced age [10], male gender [1], rectal surgery [4, 5, 9, 11, 12], post-operative hyperglycaemia (diabetics and non-diabetics) [5, 11–16], operative duration [1, 5, 9, 10], high American Society of Anaesthetists (ASA) score [10], transfusion [1] and open surgery [9]. Other than post-operative hyperglycaemia, none can be easily modified. Glycaemic control using an insulin infusion following cardiac surgery and other surgeries reduces SSI [11, 15, 17–20].

The American College of Surgeons National Surgical Quality Improvement Program (ACS-NSQIP) is a validated program providing risk-adjusted, operative outcomes data using standardised, prospective, high-quality clinical data [21–23]. In 2015, Nepean and three other New South Wales (NSW) Hospitals formed the NSW NSQIP Collaborative [24]. Early reports identified SSI in CRS were high

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[24]. ACS-NSQIP data have been used to assess SSI bundles in CRS [25–29]. The Nepean bundle included: prophylactic antibiotics [30], chlorhexidine skin prep [31], normothermia [32], second dosing intra-operative antibiotics [33], wound protection drapes [34], change of gown and gloves and separate closure tray [29]. In late 2016, the CRS unit introduced insulin infusion for glycaemic control in all patients (diabetic and non-diabetic) with post-operative hyperglycaemia. No study has examined the effect of glycaemic control as the only additional intervention to reduce SSI in CRS. The aim of this study was to assess the effect of an insulin infusion for glycaemic control on SSI in CRS.

Materials and methods

A cohort comparison was performed using NSQIP raw data for the 12 months prior and 12 months after, the intervention was introduced. All resections (elective or emergency) in the CRS unit utilised the SSI bundle (Table 1). Post-operative glycaemic control with an insulin infusion was added in late 2016. All patients having CRS had a BSL performed intra-operatively (60 to 90 min after the commencement), on admission to the recovery ward and 60 min later. Hyperglycaemia was a blood sugar level (BSL) $>$ 180 mg/Dl ($>$10 mmol/L). Non-diabetics with hyperglycaemia were considered to have stress-induced hyperglycaemia (SIH) [35, 36]. Patients with a BSL $>$ 180 mg/Dl ($>$ 10 mmol/L) commenced an insulin infusion that was titrated to maintain the BSL between 135-180 mg/Dl (7.5 and 10 mmol/L) for 48 h. Beyond 48 h, diabetics were returned to their usual insulin regime or oral medications. Patients with SIH [35, 36] frequently remained euglycaemic 48 h after CRS. Hyperglycaemic patients able to eat commenced a low sugar diet.

The trained Surgical Clinical Reviewer (K.S.) collected NSQIP data over an 8-day cycle for selected procedures. Data include demographics, comorbidities, diagnosis, laboratory variables, operative data and 30-day complications [21]. The ACS-NSQIP raw data are prospectively collected and stored on a security-protected computer. The primary outcomes assessed were superficial wound, deep wound and organ/space SSI. Secondary outcomes included other complications, LOS, re-operation and readmission. Data were analysed for two 12-month periods: the pre-intervention group (July 2015 to June 2016) and the intervention group (January 2017 to December 2017). During the pre-intervention period, only general and thoracic surgery cases were collected for NSQIP. During the intervention period urology, head and neck and neurosurgery were included in the NSQIP.

Patient demographic and clinical characteristics were reported as mean and standard deviation, or confidence intervals for numerical scaled features and percentages for discrete characteristics. Risk factors for SSI were analysed using unconditional logistic regression analysis. All p-values calculated were two-tailed; the alpha level of significance was 0.05. The Western Sydney Local Health District Ethics Committee provided ethics approval for the utilisation of ACS-NSQIP. The manuscript has been prepared using STROBE guidelines (Appendix)

Table 1 Evidence-based components of the Nepean Hospital SSI bundle for CRS

Component
Antibiotic prep prophylaxis (Cephazolin 2 g, IV 1 h prior to incision).
Skin prep with 2% chlorhexidine
Skin hair clipping
Wound protection drape
Second dosing antibiotics after 3 h
Maintenance of normothermia (Temp $>$ 36.5°)
Change of gloves and separate closing instrument set
Wound lavage with saline

Results

There were 100 patients in the pre-intervention group (90.2% of CRS cases) and 99 in the intervention group (68.5% of CRS cases). In the pre-intervention group 19 (19%), patients were diabetic. Only two (10.5%) had post-operative insulin infusions. The remainder had long acting subcutaneous insulin injections. The number of patients with SIH is unknown as routine BSL in non-diabetics was not performed. The intervention group had 18 (18.2%) diabetic patients and 14 (14.1%) with SIH. Overall, 26 (26.3%) patients in the glycaemic control group had an insulin infusion, 12 diabetics and 14 with SIH.

The incidence of SSI in the pre-intervention group (25%) fell significantly in the intervention group (6.1%) (OR = 5.17, Cl = 1.92–16.08, $p < 0.001$). Superficial wound SSI reduced from 11 to 4.0%, although this was not significant. The incidence of deep SSI was low and similar in both groups. Organ/space SSI fell significantly from 13 to 1.0% (OR = 11.35, Cl = 1.62–488.75, $p < 0.001$) (Table 2).

There were no difference in the incidence of most SSI risk factors assessed in CRS including advanced age, male gender, diabetes, cigarette smoking, body mass index (BMI), steroid usage, emergency surgery, open surgery, ASA score (2 to 4), operation duration and transfusion (Table 3). The incidence of rectal surgery was significantly

higher in the intervention group (47.5% vs. 33%) (Table 3). An ASA score of 1 was more frequent in the intervention group (Table 3), although only 63% of cases had an ASA score in the pre-intervention group compared to 96.1% in the intervention group. SSI bundle compliance for CRS was greater than 95% for each group.

The incidence of post-operative sepsis in the pre-intervention group was low (4%) with the NSQIP risk-adjusted odds ratio of 0.79. The reduction in the intervention group (1.0%) was not significant (Table 2). There were non-significant reductions in wound dehiscence, pneumonia and urinary tract infection in the intervention group (Table 2).

Re-operation rate fell from 12 to 7.8%, which was not significant (OR = 0.65, Cl = 0.03 –12.99, $p = 0.30$) (Table 2). The indications for re-operation were similar (Table 4) except the intervention group had no pelvic abscess or anastomotic leak.

There was a non-significant reduction in LOS from 11.8 ± 12.2 Days to 9.2 ± 10.0 days ($p = 0.10$) (Table 2). The readmission rate fell from 13 to 10.4%, which was not significant (Table 2), with many readmissions not related to SSI (Table 4).

The post-operative mortality was similar for the two groups (Table 2). All were following emergency CRS in elderly, high risk (ASA 4) patients (Table 5).

Discussion

Hyperglycaemia is a risk factor for SSI in CRS [3, 5, 16, 37]. These results demonstrate that post-operative glycaemic control significantly reduces SSI in CRS. Similar findings were reported for cardiac surgery [19, 20] and non-cardiac surgery [15, 17, 18]. Our results support the recent inclusion of post-operative glycaemic control in the ACS SSI guidelines [38].

Poor pre-operative glycaemic control in diabetics is associated with increased SSI risk in general, orthopaedic, cardiac and vascular surgery [5, 12, 13, 39, 40]. It is assumed that improving pre-operative glycaemic control for several months prior to surgery reduces the incidence of SSI. Poor patient compliance and limited time prior to surgery often prevent this occurring. The present study demonstrates that post-operative glycaemic control reduces SSI in CRS which may be more reliable.

Up to 46% of patients having general or cardiac surgery have SIH [36] which is associated with increased SSI rates [13–15]. Indeed, SSI is higher in patients with SIH than in diabetics [35]. The mechanisms for hyperglycaemia increasing SSI may be a reduction in macrophage and neutrophil function [41] and changes in cell-mediated immunity such as chemotaxis, opsonisation and phagocytosis [42]. Hyperglycaemia is associated with increased oxidative stress that may alter tissue perfusion and cellular immunity [43]. Insulin infusions may reduce the incidence of SSI through improved neutrophil function [41] and the anabolic, anti-inflammatory and anti-apoptotic effects of insulin [44].

The expected effect of glycaemic control was a reduction in superficial wound SSI, which was over 60%, but not statistically significant. This is due to a Type 2 statistical error. Power calculations determined that 120 patients are required to demonstrate a significant reduction in superficial SSI. Due to the methodology used, this was not possible and is a weakness of this study.

Table 2 Comparison for the various outcomes reported for the pre-intervention group (July 2015 to June 2016) and the intervention group (January 2017 to December 2017)

Criteria	July 2015–June 2016	Jan 2017–Dec 2017	OR	Cl	*p* value
Superficial wound SSI	11/100 (11%)	4/99 (4.0%)	2.93	0.83–13.03	0.06
Deep wound SSI	1/100 (1.0%)	1/99 (1.0%)	0.99	0.01–78.48	0.99
Organ/Space SSI	13/100 (13%)	1/99 (1.30%)	11.35	1.62–488.75	<0.001
Total SSI	25/100(25%)	6/99 (6.1%)	5.17	1.92–16.08	<0.001
Sepsis	4/100 (4%)	1/99 (1.0%)	4.08	0.39–203.07	0.18
Wound dehiscence	5/100 (5%)	2/99 (2.0%)	2.55	0.40–27.30	0.25
Pneumonia	5/100 (5%)	0/99 (0%)	5.15	0.55–246.35	0.10
UTI	9/100 (9%)	4/99 (4.0%)	2.35	0.62–10.76	0.16
Re-operation	12/100(12%)	6/99 (6.1%)	2.11	0.69–7.14	0.14
LOS (Days)	11.8 ± 12.2	9.2 ± 10.0			0.10
Readmission rate	13/100 (13%)	8/99 (8.1%)	1.70	0.61–4.96	0.26
Mortality	2/100 (2%)	3/99 (3.0%)	0.65	0.05-5.84	0.64

SSI Surgical Site Infection, *UTI* Urinary tract Infection, *LOS* Length of Stay, *OR* Odds Ratio, *CI* Confidence Interval

Table 3 Demographic data and the incidence of factors associated with increased SSI in CRS for the pre-intervention group (July 2015 to June 2016) and the intervention group (January 2017 to December 2017)

Criteria	July 2015–June 2016	Jan 2017–Dec 2017	OR	Cl	*p* value
Number	100	99			
Age (Years)	66.6 ± 14.9	66.3 ± 16.5			0.89
Male gender	49/100 (49%)	50/99 (50.5%)	0.94	0.52–1.70	0.83
BMI	28.35 ± 6.2	28.9 ± 7.3			0.57
Diabetic	19/100 (19%)	18/99 (18.2%)	1.06	0.48–2.30	0.88
Smoker	20/100 (20%)	13/99 (13.1%)	1.65	0.72–3.86	0.19
Steroids	11/100 (11%)	10/99 (10.1%)	1.10	0.40–3.04	0.84
Pre-operative sepsis	9/100 (9%)	14/99 (14.1%)	0.60	0.22–1.58	0.26
Open surgery	50/100(50%)	48/99 (498.5%)	1.06	0.59–1.92	0.83
Laparoscopic	50/100 (50%)	45/99 (51.5%)	1.20	0.66–2.17	0.52
Rectal resection	33/100 (33%)	47/99 (47.5%)	0.47	0.26–0.89	<0.001
Right hemicolectomy	27/100 (27%)	25/99 (25.3%)	1.09	0.55–2.17	0.78
Hartmann's	7/100 (7%)	7/99 (7.1%)	0.77	0.23–2.43	0.62
Elective surgery	73/100 (73%)	55/99 (55.6%)	2.16	1.15–4.09	0.01
Emergency	27/100 (27%)	44/99 (44.4%)	0.46	0.24–0.87	0.01
OR duration (min)	213.5 ± 132.4	207 ± 81.3			0.68
Blood transfusion	6/100 (6%)	2/99 (2.0%)	3.09	0.53–31.94	0.15
ASA 1	3/63 (4.8%)	15/99 (15.2%)	0.28	0.05–0.99	0.04
ASA 2	31/63 (49.2%)	40/99 (40.4%)	1.43	0.72–2.83	0.27
ASA 3	21/63 (33.3%)	36/99 (36.4%)	0.87	0.42–1.79	0.69
ASA 4	8/63 (12.7%)	8/99 (8.1%)	1.65	0.50–5.36	0.34

BMI Body Mass Index, *OR* Operating Room, *ASA* American Society of Anaesthetists, *OR* Odds Ratio, *CI* Confidence Interval

The unexpected result in this study was the significant reduction in the organ/space SSI. Most general surgeons would consider organ/space SSI in CRS is due to anastomotic leakage which would not be altered by glycaemic control. Nonetheless, there was a reduction in organ/space SSI and no re-operations for anastomotic leak or pelvic abscess in the intervention group. The reduction in organ/space SSI may be related to improved anastomotic healing due to improved tissue perfusion and cell-mediated immunity [42, 43] as well as the anabolic, anti-inflammatory and anti-apoptotic effects of insulin [41, 44]. In addition to further studies to confirm our findings, further studies into the mechanisms glycaemic control which reduces SSI are required.

Obesity is a risk factor for SSI in CRS [2, 5, 9], and the BMI of the comparative groups were similar. Of note, patients with BMI greater than 40 received a 50% increase dose of antibiotic prophylaxis. Obesity is a risk factor for SIH due to insulin resistance. A possible mechanism for increased SSI associated with obesity may be SIH. Not knowing the incidence of post-operative hyperglycaemia in the pre-intervention cohort is a weakness in our study. As all factors, including BMI, were similar between the groups, the incidence of SIH is likely to be similar. Further studies are needed to examine the relationship between obesity, SIH and SSI in CRS.

Although combining the results for glycaemic control for diabetics and SIH has been reported in previous studies [11, 15, 17–20], this is a potential weakness of this study. There were inadequate cases for a subset analysis. Future studies on glycaemic control need to have much larger numbers in order to compare the outcomes of glycaemic control in diabetics against those with SIH.

A bias in NSQIP patient selection is a potential problem and weakness. This is a particular issue with the proportion of CRS cases selected during the intervention period due to the expansion of NSQIP to other surgical specialties. However, the selection process for patients entered into NSQIP reduces any selection bias [22]. A selection bias is unlikely as the incidence of factors associated with increased SSI risk in CRS including advanced age [10], male gender [1], obesity [5, 9, 10], operative duration [1, 5, 9, 10], ASA score 2 to 4 [10] and intra-operative blood transfusion [1] is the same. Due to the low incidence of factors such as smoking, steroid usage, transfusion and pre-operative sepsis, subtle differences in the incidence of these factors may not be apparent due to the low total numbers and a risk of a Type II statistical error.

Table 4 Indications and incidence for re-operation and readmissions during the pre-intervention group (July 2015 to June 2016) and the intervention group (January 2017 to December 2017)

Indication	July 2015–June 2016	Jan 2017–Dec 2017
Re-operation		
Anastomotic leak	2 (2%)	0
Pelvic Abscess	4 (4%)	0
Ileostomy complications	1 (1%)	1 (1.3%)
Small bowel obstruction/Ileus	0	1 (1.3%)
Wound dehiscence	1 (1%)	1 (1.3%)
Wound debridement/drainage	1 (1%)	1 (1.3%)
Post-operative haemorrhage	0	1 (1.3%)
Anastomotic haemorrahge	1 (1%)	0
Obstructive Inguinal Hernia	0	1 (1.3%)
Acute calculous cholecystitis	1 (1%)	0
Total Re-operations	12 (12%)	6 (7.8%)
Readmission		
Small bowel obstruction/Ileus	3 (3%)	1 (1.3%)
Non-specific abdominal pain	3 (3%)	2# (1.3%)
Stoma complications	2 (1.5%)	1 (1%)
Wound SSI	2* (1%)	3** (1.3%)
Organ/Space SSI	1 (1%)	0
Superior mesenteric vein thrombosis	1 (1%)	0
Acute cholecystitis	1 (1%)	0
Urinary tract infection with sepsis	0	1 (1.3%)
Total Readmissions	13 (13%)	8 (8.1%)

Table 5 Post-operative mortality in the pre-intervention and glycaemic control groups

Diagnosis	ASA score	Age	Surgery	Death days Post Op
Pre-intervention				
Perforated sigmoid	4	82	Hartmann's procedure	4
Malignant obstruction and perforated caecum	4	84	Subtotal colectomy	5
Glycaemic Control				
Malignant obstruction	4	90	Subtotal colectomy	4
Malignant obstruction and ischaemic caecum	4	79	Subtotal colectomy	11
Malignant obstruction and perforated caecum	4	87	Extended Right Hemicolectomy	11

Rectal surgery is a risk factor for SSI [3, 5, 9, 11] and had a higher incidence in the intervention group. This should be associated with an increase in SSI. The other less frequent types CRS performed included extended right hemicolectomy, subtotal and total colectomy and pan-proctocolectomy. There were no difference in the incidences of these forms of CRS between the comparison groups, although the low numbers may result in overlooking any real effect.

The intervention group had a higher incidence of emergency CRS. This would be expected to increase SSI. The apparently high incidence of emergency CRS at Nepean Hospital is due to several local factors including the Acute Surgical Unit [45], which manages all emergency general surgery for the region and a separate private hospital where up to 50% of elective, but almost no emergency CRS occurs. The higher proportion of emergency surgery in the intervention group may also be due to an unintended selection bias in NSQIP patient selection.

The lower incidence of ASA grade 1 in the pre-intervention group may be due to the under reporting of ASA during this period. This improved after the review of the NSQIP data collection process in late 2016. It is possible that where the ASA was not recorded, it was a score of 1. Nonetheless, if an ASA score of 1 was more frequent in the intervention group, this would lower the SSI rate.

There was a non-significant reduction the incidence of post-operative sepsis in the glycaemic control group. However, the incidence of sepsis in the pre-intervention group was lower than average (OR = 0.79). In order to demonstrate a significant reduction in sepsis of 15% with 80% power, 300 patients are required in each group.

The re-operation rate had a non-significant reduction from 12 to 6.1%. Although there is a trend towards a reduction in re-operation rate, the indications were often factoring other than an SSI and as expected, the fall in re-operation rate is not as large as the fall in SSI incidence. There were two re-operations for anastomotic leak and four for pelvic abscesses in the pre-intervention group, but none in the intervention group suggests a possible reduction in the risk of anastomotic leak associated with the intervention group.

The NSQIP results for LOS for surgery in all NSW collaborative hospitals demonstrated they are all high outliers [24]. The higher LOS is due to differences in the health system between Australia and North America. In North America, many hospitals discharge the patient from the acute hospital to a step-down facility. Australia has no step-down facilities, with patients often discharged directly to home. However, despite a much lower SSI rate, the LOS was not reduced. As organ/space SSI is a serious problem resulting in longer LOS [5, 7], it would be expected that the LOS would be significantly reduced. The likely explanation is a type 2 statistical error. Power calculations determined that 315 cases required in each group to demonstrate a significant reduction by 2 to 3 days.

The readmission rate was not significantly different as only a quarter of readmissions were related to SSI in each group. The other reasons varied and were not related to SSI and therefore are unlikely to be affected by glycaemic control.

A further criticism of this study is that it is not a randomised controlled trial. However, given that post-operative hyperglycaemia is an independent factor in post-operative SSI [5, 11, 13] and post-operative glycaemic control is associated with reduced SSI [19, 20], there was a lack of equipoise.

Post-operative insulin infusion did not affect mortality. All deaths were in high-risk patients (ASA 4) with significant comorbidities having emergency CRS. The deaths were related to pre-existing sepsis at presentation and/or exacerbation of their comorbidities.

Previous studies demonstrating a reduction in SSI in CRS after the implementation of an SSI bundle [25–27, 29] are unable to determine the extent that each component contributes to the improvement. One strength of this study is that glycaemic control with an insulin infusion was the sole difference, providing good evidence that glycaemic control reduces SSI in CRS. Additional studies are required to confirm these results and assess if post-operative glycaemic control reduces SSI in other types of surgery.

Appendix

STROBE checklist of items that should be included in reports of observational studies

Criteria	Item No.	Recommendation	Page no.	Comments
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	2	Added "Using a cohort comparison"
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2	Provided
Introduction				
Background/ rationale	2	Explain the scientific background and rationale for the investigation being reported	3 & 4	Brief background of the effect of hyperglycaemia causing SSI, the effect of glycaemic control in other surgeries and the use of NSQIP data to examine the hypothesis.
Objectives	3	State-specific objectives, including any prespecified hypotheses	4	
Methods				
Study design	4	Present key elements of study design early in the paper	5	Clearly stated in the first paragraph

Criteria	Item No.	Recommendation	Page no.	Comments
Setting	5	Describe the setting, locations and relevant dates, including periods of recruitment, exposure, follow-up and data collection	5 & 6	
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up	5, 9 & 10	Provided in the methods and discussed in the discussion section.
		Case–control study—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls		
		Cross-sectional study—Give the eligibility criteria, and the sources and methods of selection of participants		
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed		This is not a matched study. This has been discussed in the discussion section
		Case–control study—For matched studies, give matching criteria and the number of controls per case		
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders and effect modifiers. Give diagnostic criteria, if applicable	7 & 8 Table 3	Confounders discussed in the discussion section
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	*5*	*Used NSQIP data and references provided for the definitions of this data.*
Bias	9	Describe any efforts to address potential sources of bias	11 & 12	Bias was discussed in several paragraphs in the discussion section.
Study size	10	Explain how the study size was arrived at	5 & 6. 10	Numbers were dependant on the NSQIP data collected which is explained in the methods and results. Also discussed in the discussion section
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	7 & Table 3	The reasons are explained in the results and discussion section
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	6	
		(*b*) Describe any methods used to examine subgroups and interactions		Not applicable due to small numbers. Discussed in the discussion section.
		(*c*) Explain how missing data were addressed	7& 11	The only missing data was the ASA classification. This was explained, acknowledged and discussed.
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed		The NSQIP data are a 30-day follow-up and follow-up was complete for all patients.
		Case–control study—If applicable, explain how matching of cases and controls was addressed		
		Cross-sectional study—If applicable, describe analytical methods taking account of sampling strategy		
		(*e*) Describe any sensitivity analyses	Not relevant	

Criteria	Item No.	Recommendation	Page no.	Comments
Results				
Participants	13*	(a) Report numbers of individuals at each stage of study—e.g., numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	7	
		(b) Give reasons for non-participation at each stage		Non-participation in data collection was explained.
		(c) Consider use of a flow diagram		Considered but would not add to the understanding of the information.
Descriptive data	14*	(a) Give characteristics of study participants (e.g., demographic, clinical, social) and information on exposures and potential confounders	7, Table 3	The only missing data was the ASA classification. This was explained, acknowledged and discussed.
		(b) Indicate number of participants with missing data for each variable of interest	7, Table 3	
		(c) *Cohort study*—Summarise follow-up time (e.g., average and total amount)		Defined as 30 days in the methods and references to NSQIP
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time	7, Tables 2, 3, 4 and 5	
		Case–control study—Report numbers in each exposure category, or summary measures of exposure		
		Cross-sectional study—Report numbers of outcome events or summary measures		
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (e.g., 95% confidence interval). Make clear which confounders were adjusted for and why they were included		No unadjusted estimates were sued.
		(*b*) Report category boundaries when continuous variables were categorised	7, Tables 2 & 3	Reported
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period		Not relevant to this study
Other analyses	17	Report other analyses done—e.g., analyses of subgroups and interactions and sensitivity analyses		Subgroup analyses were not possible due to small numbers. This is acknowledged in the discussion section, Pages 10 & 11
Discussion				
Key results	18	Summarise key results with reference to study objectives	9	
Limitations	19	Discuss limitations of the study, considering sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	10, 11 & 12	Potential bias and limitations discussed were Page 10: Type 2 error for wound SSI, obesity as a confounder. Page 11: Stress-induced hyperglycaemia, NSQIP case selection bias and rectal surgery
				Page 12: Emergency surgery and ASA scores.
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies and other relevant evidence	9 & 14	
Generalisability	21	Discuss the generalisability (external validity) of the study results	9 & 14	

Criteria	Item No.	Recommendation	Page no.	Comments
Other information				
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based		There was no funding provided. This is acknowledged on the title page.

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