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Hernia

History of surgical site infection increases the odds for a new infection after open incisional hernia repair

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ABSTRACT

Background: It is unclear whether a history of surgical site infection is associated with developing a new infection after subsequent operations. We aim to investigate the impact of an earlier abdominal wall surgical site infection on future 30-day infectious wound complications after open incisional hernia repair with mesh.

Methods: Patients undergoing elective, clean open incisional hernia repair were identified within the Americas Hernia Society Quality Collaborative and were divided into those with and without a history of a surgical site infection. Predictors of a surgical site infection and a surgical site infection requiring a procedural intervention were investigated using logistic regression and propensity-matched analysis. A subgroup analysis was done to investigate whether an earlier methicillin-resistant *Staphylococcus aureus* surgical site infection specifically increases odds for infectious complications.

Results: Of 3,168 identified patients, 589 had a history of a surgical site infection and experienced higher rates of postoperative surgical site infection (6.5% vs 2.9%, $P < .001$) and surgical site infections requiring procedural intervention (5.3% vs 1.9%, $P < .001$). After adjusting for identified confounders, a previous surgical site infection was independently associated with developing another surgical site infection (odds ratio 2.04, 95% confidence interval 1.32–3.10, $P < .001$) and a surgical site infection requiring procedural intervention (odds ratio 2.2, 95% confidence interval 1.35–3.55, $P = .001$). Propensity-matched analysis controlling for additional confounders confirmed the association of an earlier surgical site infection with the outcomes of interest (odds ratio 2.1 and 2.8, respectively). A subgroup analysis found that an earlier methicillin-resistant *Staphylococcus aureus* infection specifically did not incur higher rates of surgical site infection when compared with non–methicillin-resistant *Staphylococcus aureus* pathogens.

Conclusion: History of a surgical site infection increases the odds for new infectious complications after open incisional hernia repair in a clean wound. Investigations on perioperative interventions to ameliorate the negative impact of such association are necessary.

Introduction

Surgical site infections (SSIs) remain a frequent complication after open incisional hernia repair (OIHR) with mesh and are associated with increased health care costs, decreased quality of life, and extended hospital stays.[1] Ultimately SSIs contribute to a vicious cycle of wound complications, hernia recurrence, and another—likely more complex—repair.[2] Innumerable determinants can influence the occurrence of a postoperative wound infection, which includes patient comorbidities, hernia characteristics, wound contamination, and surgical technique.[3,4] Although some of these factors are amenable to control or modification (ie, smoking, obesity, and surgical technique), others are not (ie, presence of contamination, hernia characteristics).[5] That said, identifying "high-risk" patients can allow for targeted interventions to potentially mitigate risk.

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Whether a history of skin and wound infections specifically are risk factors for a future SSI is controversial. Some investigators have found no association of an earlier SSI—including methicillin-resistant *Staphylococcus aureus* (MRSA) SSIs—with subsequent wound infection after ventral hernia repair (VHR).[6–8] Alternatively, some have found that a history of a wound infection is associated with a substantial increase in future SSI rates for patients undergoing VHR, with added risk in those with a history of MRSA-specific wound morbidity.[9,10] Others have found that *any* history of skin infection is an independent risk factor for a new SSI, potentially reflecting an increased susceptibility even among immunocompetent individuals.[11] Finally, even if a previous SSI does carry added risk, the degree of that risk is also disputed. For example, the Ventral Hernia Working Group classified those patients with an earlier SSI alongside hernias with active contamination (Grade 3),[12] and subsequent validation found that these patients carried a similar risk as those with medical comorbidities (Grade 2) but no contamination.[4]

Given the tremendous discrepancy in the available literature, we sought to investigate the association between an earlier abdominal wall SSI and future 30-day wound complications after OIHR, with a particular interest in patients developing another SSI or SSI requiring procedural intervention. In addition, we aimed to investigate whether a MRSA SSI confers additional impact on rates of wound complications when compared with other pathogens.

Methods

Patient identification

After obtaining Institutional Review Board approval, adult patients who have undergone an elective OIHR in a clean wound—wound class I according to the Centers for Disease and Control (CDC) Classification[13]—with 30-day follow-up were identified within the Americas Hernia Society Quality Collaborative (AHSQC) database. Those patients undergoing repairs of primary ventral hernias (umbilical, epigastric, or lumbar), sutured repairs of incisional hernias (without mesh placement), emergent incisional hernia repairs, or repairs performed in the setting of contamination (CDC II, III, or IV) were excluded from analysis. Also, patients were not included for analysis if information about a history of SSI was missing in the database.

Data source

Information was abstracted from the AHSQC database in a deidentified manner. The AHSQC is a hernia-specific nationwide registry with the objective of continuous quality improvement, accomplished through patient-centered data collection, ongoing performance feedback to clinicians, and improvement based on analysis of collected data and collaborative learning. At the time of this study, the AHSQC had data available from 312 surgeons practicing in a variety of clinical settings, including academic, community, and academic-affiliated hospitals. Data on the AHSQC are collected prospectively and entered by the surgeon on a real-time basis during patient care. The AHSQC collects patient demographics information, hernia-specific variables, operative details, patient-reported outcomes, and postoperative follow-up information at each patient contact. Other details regarding the AHSQC and registry structure, governance, and data assurance process have been reported elsewhere.[14]

Outcomes of interest

Primary outcomes of interest of this study were 30-day SSI and the subset of SSIs requiring procedural intervention (wound opening, wound debridement, percutaneous drainage, and partial or complete mesh removal). SSIs were reported and classified according to the CDC classification as superficial, deep incisional, or organ-space infections.[13] Secondary outcomes of interest included surgical site occurrences (SSOs) and surgical site occurrences requiring procedural intervention (SSOPIs). SSOs include any SSI and wound cellulitis, nonhealing incisional wound, fascial disruption, skin or soft-tissue ischemia, skin or soft-tissue necrosis, wound serous or purulent drainage, stitch abscess, seroma, hematoma, infected or exposed mesh, or development of an enterocutaneous fistula. SSOPIs include any SSO that requires a procedural intervention for treatment (as detailed elsewhere).[15]

Statistical analysis

Data were described using median and interquartile ranges (IQRs) for continuous variables and counts with percentages for categorical variables as appropriate. For analysis, patients were divided into two groups: (A) no previous history of abdominal wall SSI and (B) previous history of abdominal wall SSI. Group B included patients with a previous history of SSI, independent of the pathogen identified in the earlier infection (non-MRSA or MRSA). Two separate analyses were performed. First, multivariate logistic regression models were built with SSI and SSIs requiring a procedural intervention as the outcomes of interest, adjusting for identified confounders. Selection of covariates included in the model was based on clinical consensus and respecting the limit of 1 covariate per every 10 events of the outcome of interest. As such, in addition to the variable of interest (previous history of abdominal wall SSI), the following were included for adjustment: age, obesity, diabetes, hernia width, myofascial release, mesh type, and mesh position.

Next, to balance the variability of demographic, hernia characteristic, and operative variables between the groups, a propensity-matching analysis was performed. A 2:1 matching ratio was used to compare the groups (no previous history of abdominal wall SSI versus previous history of abdominal wall SSI). Finally, a subgroup analysis with a 1:1 matching ratio compared those patients with a previous history of an MRSA infection to those with a previous history of non-MRSA infection. The penalized maximum likelihood estimation method was used in a logistic regression model because of the rarity of outcomes of interest in the study population. Correlation between case and control patients owing to matching was also considered in the logistic regression model. Statistical significance was achieved by examining the 95% confidence intervals (CIs) for odds ratios (ORs). R 3.3.1 (2016-06-21) was used for all analyses.

Results

A total of 3,168 patients met the inclusion criteria. Of those, 589 (18.6%) had a history of abdominal wall SSI and the rest did not. Univariate analysis (Table I) revealed several statistically significant differences between the groups in demographics, hernia characteristics, and operative details, with a clear tendency for greater case complexity in the group with a history of an SSI. For example, patients with a history of an SSI were more frequently obese (64.3% vs 59.3%, $P = .028$), had larger hernias (12 cm vs 8 cm, $P < .001$) that were more frequently recurrent (53.3% vs 38.8%, $P < .001$), and were more likely to require a myofascial release (84% vs 61.3 %, $P < .001$). In addition to patients with earlier SSI having lengthier operations (operative time >2 hours 87.6% vs 63.9%, $P < .001$), repairs were

Table I
Univariate comparisons: Unmatched population

$N = 3,168$	Previous history of abdominal wall surgical site infection		
Characteristic, N (%)	No $n = 2,579$	Yes $n = 589$	P value
Age (years), median [IQR]	59 [49–67]	56 [47–65]	.001
BMI (kg/m^2), median [IQR]	31.3 [27.5–35.9]	32.1 [28–36.4]	.015
Obesity (BMI > 30 kg/m^2)	1530 (59.3)	379 (64.3)	.028
ASA class			< .001
1	108 (4.2)	7 (1.2)	
2	1040 (40.3)	194 (32.9)	
3	1350 (52.3)	374 (63.5)	
4	81 (3.1)	14 (2.4)	
Recurrent hernia	1000 (38.8)	314 (53.3)	< .001
Previous component separation	67 (2.6)	57 (9.7)	< .001
History of open abdomen	292 (11.3)	120 (20.4)	< .001
Prophylactic antibiotics administered	2566 (99.5)	585 (99.3)	.539
Preoperative chlorhexidine scrub at home	1556 (60.3)	432 (73.3)	< .001
Hernia width (cm), median [IQR]	8 [4–12]	12 [8–15]	< .001
Myofascial release	1580 (61.3)	495 (84)	< .001
TAR	806 (31.3)	305 (51.8)	< .001
Mesh type			.021
Permanent synthetic	2399 (93)	533 (90.5)	
Absorbable synthetic	105 (4.1)	27 (4.6)	
Biologic	48 (1.9)	24 (4.1)	
Biologic/synthetic hybrid	14 (0.5)	4 (0.7)	
Uncoated permanent synthetic mesh	1669 (64.7)	438 (74.4)	< .001
Mesh location			< .001
Onlay	296 (11.5)	37 (6.3)	
Sublay	1876 (72.7)	478 (81.2)	
Intraperitoneal	318 (12.3)	52 (8.8)	
Inlay	89 (3.4)	22 (3.7)	
Operative time > 2 hours	1649 (63.9)	513 (87.6)	< .001
Drains used	1841 (71.4)	537 (91.2)	< .001
Length of stay (days), median [IQR]	4 [1–5]	5 [4–6]	< .001
30-day SSI	75 (2.9)	38 (6.5)	< .001
SSIs requiring a procedural intervention	50 (1.9)	31 (5.3)	< .001
30-day SSO	296 (11.5)	94 (16)	.004
30-day SSOPI	139 (5.4)	54 (9.2)	< .001
Unplanned readmissions	118 (4.6)	53 (9)	< .001

Note: The following did not present statistically significant differences: sex, diabetes, immunosuppressants, chronic obstructive pulmonary disease, liver failure, dialysis, ascites, history of inflammatory bowel disease, smoking, subcutaneous flaps raised, fascial closure, SSI classification (superficial, deep, organ space), unplanned reoperations.
ASA class, American Society of Anesthesiologists Classification; *BMI*, body mass index; *IQR*, interquartile range; *TAR*, transversus abdominis release.

Table II
Multivariate logistic regression results: unmatched population ($N = 3,168$)

Outcome: 30-day SSI	Odds ratio	95% CI	P value
Previous history of abdominal wall SSI	2.04	1.32–3.10	< .001
Age	0.98	0.96–0.99	.008
Obesity	1.29	0.85–1.99	.241
Diabetes	1.66	1.07–2.52	.020
Hernia width	1.03	1.00–1.06	.061
Myofascial release	0.76	0.46–1.27	.291
Mesh type			
Permanent synthetic (reference)	1.0		
Absorbable synthetic	0.9	0.27–2.20	.832
Biologic	1.57	0.52–3.87	.372
Biologic/synthetic hybrid	1.25	0.07–6.81	.837
Mesh location			
Sublay (reference)	1.0		
Onlay	1.09	0.53–2.05	.802
Intraperitoneal	1.04	0.51–2.01	.907
Inlay	1.52	0.56–3.43	.359
Outcome: 30-day SSI requiring procedural intervention	**Odds ratio**	**95% CI**	***P* value**
Previous history of abdominal wall SSI	2.21	1.35–3.55	.001
Age	0.98	0.96–1.00	.026
Obesity	1.43	0.87–2.43	.173
Diabetes	1.11	0.64–1.87	.694
Hernia width	1.04	1.00–1.07	.030
Myofascial release	1.18	0.62–2.34	.628
Mesh type			
Permanent synthetic (reference)	1.0		
Absorbable synthetic	0.59	0.10–1.94	.472
Biologic	1.13	0.26–3.43	.850
Biologic/synthetic hybrid	1.60	0.08–8.91	.661
Mesh location			
Sublay (reference)	1.0		
Onlay	0.61	0.18–1.53	.350
Intraperitoneal	1.35	0.57–3.01	.477
Inlay	1.32	0.37–3.50	.621

more frequently performed with an uncoated piece of synthetic mesh (74.4% vs 64.7%, $P < .001$) placed as a sublay (81.2% vs 72.7%, $P < .001$). At 30-day follow-up, patients with a history of SSI subsequently had higher 30-day SSI rates (6.5 versus 2.9%, $P < .001$), which more frequently required a procedural intervention (5.3% vs 1.9%, $P < .001$). Higher rates of SSOPIs (9.2% vs 5.4%, $P < .001$) and unplanned readmissions (9% vs 4.6%, $P < .001$) were also observed in this group. Table II details the results of multivariate logistic regression models. After adjustment, a history of SSI was shown to be independently associated with increased odds for another SSI (OR 2.04, 95% CI 1.31–3.1, $P < .001$) and SSI requiring a procedural intervention (OR 2.21, 95% CI 1.35–3.55, $P = .001$).

Table III reveals the distribution of variables between groups after propensity-score matching. A total of 589 patients with a history of SSI were matched to 1,178 patients with no SSI history. All variables were well matched between the groups with the exception of age (58 vs 56 years, $P = .004$), hernia width (10 vs 12 cm, $P < .001$), history of component separation (9.7% vs 5.7%, $P = .003$), and history of open abdomen (20.4% vs 16.4%, $P = .032$). Although these differences are statistically significant, some residual differences were not clinically relevant. For patients with an SSI history, higher rates of subsequent 30-day SSI, SSIs requiring procedural intervention, SSOPI, and unplanned readmissions remained. After adjusting for those residual differences (age, hernia width, history of open abdomen, and history of component separation), an earlier SSI was again shown to be independently associated with increased odds for another SSI (OR 2.1, 95% CI 1.33–3.37, $P = .0021$) and SSI requiring procedural intervention (OR 2.77, 95% CI 1.55–4.97, $P < .001$). Table IV details the results of this additional analysis.

Finally, subgroup analysis after 1:1 matching of the group with a history of SSI (104 MRSA to 104 non-MRSA) is presented in Table V. There were no statistically significant differences between the groups on any of the investigated outcomes.

Discussion

This is the largest study of its kind investigating the influence of a history of SSI on the occurrence of subsequent wound complications after VHR, using data from a nationwide database that collects hernia-specific variables and outcomes. We found that even in clean cases of OIHR, a history of SSI is not only a marker of greater case complexity but is also independently associated with a two-fold increase in the odds of developing another SSI. In addition, those subsequent infections are more likely to require a procedural intervention for treatment. Although these findings are limited by their retrospective nature, these associations remained, despite several statistical analyses to adjust for confounding demographics, comorbidities, hernia variables, and operative details. Although the mechanism of this association remains unclear, these results should generate an important discussion on whether patients with

Table III
2:1 Matched populations

$N = 1,767$	Previous history of abdominal wall surgical site infection		
Patient characteristics, *n* (%)	No $n = 1,178$	Yes $n = 589$	*P* value
Age (years), median [IQR]	58 [50–66]	56 [47–65]	.004
Sex			.087
Female	678 (57.6)	313 (53.1)	
Male	500 (42.4)	276 (46.6)	
BMI (kg/m^2) median [IQR]	32.4 [28.4–36.7]	32.1 [28–36.4]	.891
Obesity (BMI > 30 kg/m^2)	763 (64.8)	379 (64.3)	.902
Diabetes	270 (22.9)	135 (22.9)	1.00
Immunosuppression	60 (5.1)	31 (5.3)	.970
Smoking	106 (9)	58 (9.8)	.622
Chronic obstructive pulmonary disease	107 (9.1)	55 (9.3)	.930
Inflammatory bowel disease	39 (3.3)	26 (4.4)	.304
ASA class			.947
1	18 (1.5)	7 (1.2)	
2	392 (33.3)	194 (32.9)	
3	741 (62.9)	374 (63.5)	
4	27 (2.3)	14 (2.4)	
Recurrent hernia	586 (49.7)	314 (53.3)	.173
Previous component separation	67 (5.7)	57 (9.7)	.003
History of open abdomen	190 (16.4)	120 (20.4)	.032
Preoperative chlorexidine scrub	856 (72.7)	432 (73.3)	.806
Prophylactic antibiotics used	1173 (99.6)	585 (99.3)	.492
Hernia width (cm), median [IQR]	10 [8–15]	12 [8–15]	< .001
Myofascial release	975 (82.8)	495 (94)	.544
TAR	610 (51.8)	305 (51.8)	1.00
Mesh type			.492
Permanent synthetic	1070 (90.8)	533 (90.5)	
Absorbable synthetic	63 (5.35)	27 (4.6)	
Biologic	31 (2.6)	24 (4.1)	
Biologic/synthetic hybrid	11 (0.9)	4 (0.7)	
Unknown	3 (0.1)	1 (0.2)	
Uncoated permanent synthetic mesh	876 (74.4)	438 (74.4)	1.00
Mesh location			.614
Onlay	78 (6.6)	34 (6.3)	
Sublay	973 (82.6)	472 (81.2)	
Intraperitoneal	83 (7)	52 (8.9)	
Inlay	44 (3.7)	22 (3.7)	
Subcutaneous flaps raised	368 (31.2)	208 (35.3)	.095
Fascial closure	1112 (94.4)	556(94.4)	1.00
Operative time > 2 hours	1027 (87.2)	516 (87.6)	.860
Drains used	1072 (91)	537 (91.2)	.976
Length of stay (days), median [IQR]	5 [3–6]	5 [4–6]	.004
30-day SSI	36 (3)	38 (6.45)	.001
Superficial	29 (80.6)	27 (71.1)	
Deep	6 (16.7)	10 (26.3)	
Organ space	1 (2.8)	1 (2.7)	
SSIs requiring procedural intervention	27 (2.3)	31 (5.3)	.002
30-day SSO	135 (11.5)	94 (16)	.010
30-day SSOPI	65 (5.5)	54 (9.2)	.005
Unplanned readmissions	59 (5)	53 (9)	.002
Unplanned reoperations	18 (1.4)	16 (2.7)	.067

ASA class, American Society of Anesthesiologists Classification; *BMI*, body mass index; *IQR*, interquartile range; *TAR*, transversus abdominis release.

Table IV
Multivariate logistic regression results: Matched population ($N = 1,767$)

Outcome: 30-day SSI	Odds ratio	95% CI	*P* value
Previous history of abdominal wall SSI	2.11	1.33–3.37	.0021
Age	0.97	0.95–0.99	.006
Hernia width	1.03	0.99–1.07	.125
History of open abdomen	0.53	0.26–1.09	.070
History of component separation	1.14	0.50–2.63	.758
Outcome: 30-day SSI requiring procedural intervention	**Odds ratio**	**95% CI**	***P* value**
Previous history of abdominal wall SSI	2.77	1.55–4.97	< .001
Age	0.97	0.95–0.99	.005
Hernia width	1.01	1.00–1.08	.054
History of open abdomen	0.50	0.22–1.14	.078
History of component separation	1.21	0.49–2.99	.688

a history of an SSI should be considered “high risk” and warrant additional interventions to minimize the risk of additional infectious complications.

Admittedly, SSI history is not a common factor involved in the surgical decision-making of our practice for OIHR patients, especially for those who present without clinical or radiologic evidence of active infection. Inconsistent findings within the literature have potentially undermined the association. For instance, Blatnik et al[6] investigated the association between previous wound infections and the risk of developing a new SSI specifically in the 146 OIHR patients and did not find an association.[6] Although the groups were comparable regarding demographics, hernia variables, and operative details, the limited numbers of patients with a history of SSI ($n = 22$) likely indicates the study was underpowered to demonstrate such a difference. Likewise, our follow-up analysis of 10 patients with an earlier MRSA infection with no comparison group was also insufficient to make any meaningful conclusions.[7] More commonly, studies like the one by Berger et al[3] of 888 OIHR find a larger difference in SSI-history rates between those with and without subsequent wound morbidity (24% vs 8%, $P < .01$), but the multivariate analysis does not recognize SSI history as an independent risk factor.[3] As such, surgeons tend to view an SSI history as a marker of complexity, but not an independent variable associated with future SSI risk like diabetes, obesity, wound class, skin flaps etc.[3,16]

Other publications have supported our conclusion that SSI history incurs added risk for a new SSI. As early as 1989, Houck et al[9] found that OIHR patients with a history of SSI subsequently had a 41% SSI rate after subsequent repair, compared with 12% in those without an SSI history ($P < .05$). Although their conclusion was similar to ours—that patients with an SSI history are at risk for a future SSI—they did not perform propensity matching or multivariate analysis to exclude the possibility of confounding factors. Faraday et al,[11] however, set out to adjust for additional confounders in their attempt to identify an independent association between any previous skin infections and risk of new SSIs. The authors prospectively investigated the association between previous skin infections and the development of deep or organ-space SSI during a 6-month period after elective, clean, cardiac, vascular, and neurosurgical procedures. After propensity-score adjustment of all potential confounders, they reported an increase in the risk of SSI associated with a history of skin infection (HR = 3.41; 95% CI, 1.36–8.59; $P = .009$). The authors hypothesized that these findings might be related to individual differences in immunologic susceptibility to infections and that earlier skin infections could be a marker of enhanced predisposition. Alternatively, other authors have postulated that indolent bacteria from earlier infection may remain viable in tissues and predispose to new infections.[17,18] Although these studies provide a significant contribution to the literature, there is limited ability to generalize those findings to the hernia population. Our study supports the concept that a history of SSI is not just a marker of hernia and patient complexity, but independently associated with future infectious wound morbidity and wound complications that will require procedural intervention.

Other studies have investigated the role of remote MRSA-specific infections in wound complications after VHR. Ousley et al[10] investigated the association between earlier MRSA infections at any body site and 30-day SSI after VHR. By looking at 768 patients with 54 previous MRSA infections, the authors found that any prior MRSA infection resulted in a 2.3-fold increase in the

Table V
Subgroup analysis of the group with a previous abdominal wall SSI (MRSA versus non-MRSA) after 1:1 matching ($N = 208$)

Patient characteristics	Previous history of abdominal wall surgical site infection		
	Non-MRSA $n = 104$	MRSA $n = 104$	*P* value
Age (years), median [IQR]	55 [49–65]	57 [48–65]	.674
Sex			1.00
Female	53 (51)	54 (52)	
Male	51 (49)	50 (48)	
BMI (kg/m^2) mean, SD	33 [29–36]	32.3 [28.4–36.4]	.860
Obesity (BMI > 30 kg/m^2)	70 (67.3)	67 (64.4)	.770
Diabetes	29 (27.9)	29 (27.9)	1.00
Immunosuppression	6 (5.8)	7 (6.7)	1.00
Smoking	9 (8.6)	11 (10.6)	.814
Chronic obstructive pulmonary disease	11 (10.6)	11 (10.6)	1.00
Inflammatory bowel disease	4 (3.8)	4 (3.8)	1.00
ASA class			1.00
1	0 (0)	1 (0.97)	
2	32 (30.8)	32 (30.8)	
3	70 (67.3)	69 (66.6)	
4	2 (1.92)	2 (1.92)	
Recurrent hernia	73 (70.2)	73 (70.2)	1.00
Previous component separation	20 (19.2)	21 (20.2)	1.00
History of open abdomen	23 (22.1)	26 (25)	.744
Prior mesh infection	30 (33)	50 (51.5)	.015
Preoperative chlorexidine scrub	82 (78.8)	82 (78.8)	1.00
Prophylactic antibiotics used	103 (99)	104 (100)	1.00
Hernia width (cm), median [IQR]	13 [10–16]	13 [10–15]	.920
Myofascial release	89 (85.6)	89 (85.6)	1.00
TAR	58 (55.8)	58 (55.8)	1.00
Mesh type			.678
Permanent synthetic	93 (89.4)	91 (87.5)	
Absorbable synthetic	4 (3.8)	5 (4.8)	
Biologic	6 (5.8)	6 (5.8)	
Biologic/synthetic hybrid	0	2 (1.9)	
Mesh position			1.00
Onlay	7 (6.7)	6 (5.8)	
Sublay	85 (81.7)	86 (82.7)	
Intraperitoneal	9 (8.7)	9 (8.7)	
Inlay	3 (2.9)	3 (2.9)	
Subcutaneous flaps raised	172 (35.5)	36 (34.6)	.959
Fascial closure	96 (92.3)	96 (92.3)	1.00
Operative time >2 hours	96 (92.3)	96 (92.3)	1.00
Drains used	98 (94.2)	98 (94.2)	.782
Length of stay (days), median [IQR]	5 [4–6]	5 [4–7]	.125
30-day SSI	8 (7.7)	6 (5.8)	.782
Superficial	5 (65.5)	5 (83.3)	
Deep	3 (37.5)	1 (16.7)	
SSIs requiring procedural intervention	7 (6.8)	4 (3.8)	.536
30-day SSO	22 (21.2)	17 (16.3)	.477
30-day SSOPI	15 (14.4)	8 (7.7)	.185
Readmissions	8 (7.7)	10 (9.6)	.805
Reoperations	2 (1.9)	3 (2.9)	1.00

ASA class, American Society of Anesthesiologists Classification; *BMI*, body mass index; *IQR*, interquartile range; *TAR*, transversus abdominis release.

odds of 30-day SSI after adjusting for confounding factors. Fortunately, this association did not increase the odds of mesh infection for patients during a 2-year follow-up. Nevertheless, it is important to note that only 28% of the patients with a history of MRSA infectious were soft-tissue infections and they included CDC II/III wound classes, which limit the ability to compare such findings with ours. A more recent study by Baucom et al[8] analyzed the implications of an earlier MRSA infection on long-term SSOs after VHR, but only in clean cases. They found that an earlier MRSA infection was a marker of greater case complexity, but not independently associated with higher rates of SSO and SSOPI. Given the similarity of our analysis to that of Baucom et al,[8] which focused on clean cases, we believe that our data support their findings that MRSA history does not specifically add additional risk compared with other pathogens.

Our analysis differs from these studies in several ways. First, we have not limited our analysis to MRSA-specific infections. Inversely, we have analyzed all patients with a history of SSI first and then performed an MRSA subgroup analysis later. Second, many of the cited studies have included laparoscopic repairs, repairs performed in clean-contaminated and contaminated settings, and repairs without mesh. To make our study population more homogeneous, we intentionally focused our analysis on open repairs performed in clean wounds. As such, we have tried to isolate a population of patients where the repair was performed in the absence of any type of contamination (clean wounds), the incidence of wound complications is naturally higher (open repairs), and the consequences of SSIs are clinically more relevant (mesh repairs). Also, the fact that only repairs with mesh were included excludes small umbilical and epigastric hernias where the clinical relevance of a wound infection differs importantly from cases of large ventral hernias repaired with synthetic mesh. It is important to note that even with the attempt to create groups with more similarities, a history of SSI was an independent variable that contributes to several baseline differences between groups as has been outlined. Therefore, patient and hernia-specific covariates were adjusted for in multivariate analysis. Despite adjustment, this relationship remained significant. We further complemented our investigation with a propensity-matched analysis to address the fact that there were more differences between the groups that could be adjusted for in a logistic regression model (as the number of covariates allowed in the model depends on the number of events of interest). Similarly, after matching for a multitude of clinical and operative factors, the analysis yielded similar results and confirmed the association between a history of SSI and 30-day SSIs and the subset of SSIs requiring procedural intervention. As such, we can support that further studies are necessary to help elucidate the etiology of such an association. Furthermore, we can focus on investigating the types of perioperative interventions that can potentially be beneficial to ameliorate the negative impact of an SSI history. For example, future studies investigating different types and duration of antibiotic prophylaxis protocols, different methods for wound closure, routine removal of all meshes already in place, and scar tissue when feasible, providing to the new mesh a healthy and vascularized bed.

Although our analysis has significant strengths, we believe that the findings of our study should be interpreted in light of several limitations. First, although we could identify an independent association between a history of SSI and new infections, the etiology of such an association remains unclear, and it is important to note that our study cannot determine whether there is a causative relationship between a history of SSI and the new SSIs resulting from the hernia repair. Further, our study does not support that such patients should be treated differently than those without a history of SSI. As such, further research is necessary to confirm our findings, and we hope to stimulate other researchers to recreate our analysis with data from other hernia registries to investigate whether it yields similar results. Moreover, more studies are necessary to investigate the etiologic basis of such an association, and in the future, to test different interventions to ameliorate the negative impact of such an association in the outcomes of OIHR. Next, our data originated from a review of prospectively collected data that are entered by the surgeon. Consequently, there is a potential for underreporting before abdominal wall SSIs by the surgeon and for recall bias from the patients in reporting this factor to the surgeon during preoperative evaluation. Along the same line, there is a potential for misclassification of the pathogen type involved in the earlier infections because, in clinical practice,

detailed information is not always available about past medical events. We believe that additional unmeasured confounders that contribute to wound morbidity might also have an impact on our results. For example, the type of skin preparation is a variable that is not collected by the AHSQC and therefore was not analyzed in our study. Other unmeasured confounders include the use of prehabilitation or preoperative optimization protocols before hernia repair and the use of enhanced recovery after surgery pathways for perioperative care. Last, admittedly there is a significant heterogeneity related to the previous techniques used for hernia repair. Therefore, we acknowledge that other important unmeasured confounders might be present, such as more granular details about the patient before surgical history, including procedures resulting in disruption of the blood supply to the skin and subcutaneous tissue (related or not to an earlier hernia repair).

We do acknowledge that our study could not measure the differences between the MRSA and non-MRSA groups because of the limited number of patients and events in this subgroup. In addition, we cannot assess whether decolonization protocols recommended for MRSA carriers might be an unmeasured confounder because this information is not collected by the AHSQC. Another limitation is that the AHSQC database does not record the specific pathogen related to a wound infection. Therefore, we were unable to analyze whether a history of an MRSA SSI was associated with a new SSI caused by the same pathogen. Last, as we have attempted to make our study population homogeneous and only included open, elective, incisional hernia repairs with mesh, our findings cannot be extrapolated for other hernia populations, such as those undergoing a minimally invasive repair or a repair without mesh.

In conclusion, a history of SSI increases the odds for a new infectious complication after OIHR, even in repairs performed in a clean setting. However, a history of an MRSA SSI does not appear to incur added risk. Further studies are necessary to investigate the etiology of such infections. Moreover, investigations on perioperative interventions to ameliorate the negative impact of such association in the outcomes of OIHR are necessary.

Conflicts of interest

Luciano Tastaldi and Aldo Fafaj received a research grant from the Americas Hernia Society Quality Collaborative that is not related to the present work. David M. Krpata declares an educational grant from W.L GORE that is not related to the present work. Ajita S. Prabhu receives compensation for consulting for Medtronic and has an ongoing research grant from Intuitive Surgical Inc., and none of these conflicts of interest are related to the submitted work. Benjamin K. Poulose receives research funding from Bard/Davol and salary support from Americas Hernia Society Quality Collaborative for his leadership position. Michael J. Rosen receives salary support from the Americas Hernia Society Quality Collaborative for his leadership position, is a board member of Ariste Medical Inc., has ongoing research grants from Pacira Pharmaceuticals and Intuitive Inc. and has stocks from Ariste Medical. None of these conflicts of interest are related to the submitted work. Clayton Petro, Hemasat Alkhatib, Chao Tu and Steven Rosenblatt declares no conflicts of interest.

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