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Wound Disruption Following Colorectal Operations

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Abstract

Objectives Postoperative wound disruption is associated with high morbidity and mortality. We sought to identify the risk factors and outcomes of wound disruption following colorectal resection.

Methods The American College of Surgeons National Surgical Quality Improvement Program (NSQIP) database was used to examine the clinical data of patients who underwent colorectal resection from 2005 to 2013. Multivariate regression analysis was performed to identify risk factors of wound disruption.

Results We sampled a total of 164,297 patients who underwent colorectal resection. Of these, 2073 (1.3 %) had wound disruption. Patients with wound disruption had significantly higher mortality (5.1 vs. 1.9 %, AOR: 1.46, P = 0.01). The highest risk of wound disruption was seen in patients with wound infection (4.8 vs. 0.9 %, AOR: 4.11, P\0.01). A number of factors are associated with wound disruption such as chronic steroid use (AOR: 1.71, P\0.01), smoking (AOR: 1.60, P\0.01), obesity (AOR: 1.57, P\0.01), operation length more than 3 h (AOR: 1.56, P\0.01), severe Chronic Obstructive Pulmonary Disease (COPD) (AOR: 1.36, P\0.01), urgent/emergent admission (AOR: 1.31, P = 0.01), and serum Albumin Level\3 g/dL (AOR: 1.27, P\0.01). Laparoscopic surgery had significantly lower risk of wound disruption compared to open surgery (AOR: 0.61, P\0.01).

Conclusion Wound disruption occurs in 1.3 % of colorectal resections, and it correlates with mortality of patients. Wound infection is the strongest predictor of wound disruption. Chronic steroid use, obesity, severe COPD, prolonged operation, non-elective admission, and serum albumin level are strongly associated with wound disruption. Utilization of the laparoscopic approach may decrease the risk of wound disruption when possible.

Introduction

Postoperative wound disruption or separation of the layers of a surgical wound with disruption of the fascia is a serious complication which is associated with increased morbidity, mortality, length of hospitalization, and hospital costs [1, 2]. It is estimated that 1-3 % of surgical patients develop wound disruption [3]. Despite improvements in contemporary perioperative care, data do not show any significant decrease in rate of wound disruption, and wound disruption still remains a major cause of morbidity in surgical patients [3].

Table 1	Definitions	of	some	study	variables
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Variable	Definition ^a	Variable	Definition ^a				
Wound disruption	Separation of the layers of a surgical wound, which may be partial or complete, with disruption of the fascia within 30 days of the operation	Ascites	Presence of fluid accumulation in the peritoneal cavity noted on physical examination, abdominal ultrasound, or abdominal CT/MRI within 30 days prior to the operation				
Chronic steroid	Administration of oral or parenteral corticosteroid	Chemotherapy	Within 30 days of operation				
use	medications in the 30 days prior to surgery	Dyspnea	Dyspnea at rest or moderate exertion within 30 days of operation				
Congestive heart failure	Within 30 days of operation	Radiotherapy	Within 90 days of operation				
Severe COPD	Chronic obstructive pulmonary disease resulting in any one or more of the following: Functional disability from COPD, Hospitalization in the past for treatment of COPD, Requires chronic	Dependency before surgery	Dependency in patient's abilities to perform activities of daily living in the 30 days prior to surgery such as bathing, feeding, dressing, toileting, and mobility				
	bronchodilator therapy with oral or inhaled agents, An FEV1 of <75 % of predicted on pulmonary function testing	Smoking	Smoking cigarettes in the year prior to admission for surgery. Patients who smoke cigars or pipes or use chewing tobacco are not included				
Dialysis	Acute or chronic renal failure requiring treatment with dialysis within 2 weeks prior to surgery	Preoperative sepsis	Preoperative sepsis, septic shock, and Systemic Inflammatory Response Syndrome				
Weight loss	Greater than 10 % decrease in body weight in the 6 month interval immediately preceding surgery. Patients who have intentionally lost weight as part of a weight reduction program do not qualify	Bleeding disorders	Any condition that places the patient at risk for excessive bleeding due to a deficiency of blood clotting elements. Patients who are on chronic aspirin therapy are not included				
Wound infection	An infection that occurs within 30 days after the opera- tissues of the incision and at least one of the follow		ction involves skin or subcutaneous tissue or deep sof				
	• Purulent drainage, with or without laboratory confi	irmation, from th	e superficial incision				
	• Organisms isolated from an aseptically obtained cu	ulture of fluid or	tissue from the incision				
	• Diagnosis of wound infection by the surgeon or attending physician						
	 An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopathologic or radiologic examination 						
	 At least one of the following signs or symptoms of incision is deliberately opened by the surgeon, unliberately opened by the surgeon. 						

^a Details of definitions of NSQIP collected data points are available online in the NSQIP user guide [8]

Although the development of wound disruption is closely related to the surgical technique of wound closure, local and systemic factors have significant influence on surgical wound healing [4]. Furthermore, factors such as obesity, jaundice, malignancy, diabetes mellitus, and wound infection have been reported as predictors of wound disruption [4, 5]. Recent published articles report on the benefits of prophylactic retention sutures and rectus sheath relaxing incisions in high-risk patients [2, 4, 6]. However, there are no specific guidelines of preventive strategies of wound disruption for high-risk patients. Using a nationwide database, this study aims to report the trends and the contemporary status of wound disruption following colorectal surgery in the United States (US), characterize reducible risk factors, and identify high-risk patients for wound disruption in colorectal surgery.

Materials and methods

Data for this study were collected within the American College of Surgeons National Surgical Quality Improvement Program (ACS NSQIP) Participant User Files (PUF) for 2005 through 2013. ACS NSQIP is the leading nationally validated, risk-adjusted, outcomes-based program to measure and improve the quality of surgical care in the United States [7]. This study evaluated preoperative to 30-day postoperative outcomes of patients who underwent colorectal resections using the appropriate procedure codes as specified by the current procedural terminology (CPT) codes. These codes include 44140–44160, 44204–44212, 44227, 44625, 45110-45114, 45119, 45120, 45123, 45126, 45135, 45397, 45402, 45550, and 45395. Patients who underwent colorectal surgery without colorectal resection, patients younger than 18 years, patients who had infected or open wounds during admission, and patients who had surgical abdominal wound other than wound class II (clean contaminated) were excluded from this study. Patient diagnoses were defined based on the International Classification of Diseases, 9th Revision, clinical modifications (ICD-9-CM) codes. Wound disruption was defined as separation of the layers of a surgical wound, which may be partial or complete, with disruption of the fascia within 30 days of the operation according to the standard definition of American College of Surgeons [7].

Median, year 63 64 Sex Male 77,542 (47.9 %) 1255 Race 77,542 (47.9 %) 1255 White 126,619 (85.5 %) 1573 Black or African American 14,927 (10.1 %) 235 Asian 4544 (3.1 %) 28 (Other 1546 (1 %) 32 (Comorbidity 1 1187 Diabetes mellitus 23,695 (14.6 %) 344 Chronic obstructive pulmonary disease 8224 (5.1 %) 235 Chronic steroid use 9781 (6 %) 201 Congestive heart failure 1465 (0.9 %) 39 (Disseminated cancer 8236 (5.1 %) 155 Dyspnea 14,561 (9 %) 346 Ascites 1392 (0.9 %) 41 (Anemia 13,241 (8.6 %) 268	± 15 5 (60.7 %) 3 (84.2 %) (12.6 %) (1.5 %) (1.7 %) 7 (57.3 %) (16.6 %) (11.3 %)	0.08 - <0.01 0.47 0.31 0.11 0.20 <0.01 0.06
Median, year 63 64 Sex Male 77,542 (47.9 %) 1255 Race 126,619 (85.5 %) 1573 Black or African American 14,927 (10.1 %) 235 Asian 4544 (3.1 %) 28 (Other 1546 (1 %) 32 (Comorbidity 1187 Diabetes mellitus 23,695 (14.6 %) 344 Chronic obstructive pulmonary disease 8224 (5.1 %) 235 Chronic steroid use 9781 (6 %) 201 Congestive heart failure 1465 (0.9 %) 39 (Disseminated cancer 8236 (5.1 %) 155 Dyspnea 14,561 (9 %) 346 Ascites 1392 (0.9 %) 41 (Anemia 13,241 (8.6 %) 268	5 (60.7 %) 3 (84.2 %) (12.6 %) (1.5 %) (1.7 %) 7 (57.3 %) (16.6 %)	- <0.01 0.47 0.31 0.11 0.20 <0.01
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Ascites 1392 (0.9 %) 41 (0.0 %) Anemia 13,241 (8.6 %) 268	(7.5 %)	0.23
Anemia 13,241 (8.6 %) 268	(16.7 %)	< 0.01
	(2 %)	0.70
Dialysis 1121 (0.7 %) 28 ((13.5 %)	0.08
	(1.4 %)	0.26
Weight loss 7156 (4.4 %) 127	(6.1 %)	0.32
Bleeding disorders 6563 (4 %) 143	(6.9 %)	0.96
ASA score ^b		
I 4987 (3.1 %) 23 ((1.1 %)	0.03
II 77,754 (48 %) 669	(32.3 %)	< 0.01
III 70,829 (43.7 %) 1147	7 (55.4 %)	0.01
VI 8508 (5.2 %) 233	(11.2 %)	0.12
Other factors		
Dependency before surgery ^c 6952 (4.3 %) 190	(9.2 %)	0.05
Smoking 27,181 (16.8 %) 532	(25.7 %)	< 0.01

Table 2 Demographic and comorbid factors of	patients who underwent colorectal resection
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^a Standard deviation

^b The American society of anesthesiologists score

^c Partial or total dependency before operation

Potential variables used in the analysis included baseline demographics (age, sex, and race), comorbidities, surgical approach (open vs. laparoscopic), operation length, admission type (emergent vs. non-emergent), and the most recent preoperative blood chemical values of serum albumin level, serum and white blood cell count. Definitions for NSQIP collected data points according to American College of surgeons (ACS) definition are available online in the NSQIP user guide and also are listed in Table 1 [8]. Also, anemia was defined as hematocrit less than ten. Primary end points investigated were preoperative and operative factors which had significant associations with postoperative wound dehiscence. Secondary endpoints investigated were associations between postoperative wound infection and wound disruption.

Statistical analysis

Statistical analyses were performed using the SPSS® software, Version 22 (SPSS Inc., Chicago, IL). The main analysis of the study was multivariable analysis. Logistic regression analysis was used to estimate the association between categorical variables and postoperative wound disruption, and multivariate analysis using linear regression was used for the continuous variables. For each correlation, the adjusted odds ratio (AOR) with a 95 % confidence interval was calculated and reported. P values less than 0.05 were considered statistically significant. Adjustments were made for all variables of the study. In order to report performance measures of the regression analysis model, Cox & Snell R2 of the regression analysis model was reported, which was 0.013.

Results

Overall, 164,297 patients who underwent colorectal resection were identified. The median age of patients was 63 years old; the majority of the patients were Caucasian (85.7 %) and female (52 %). Patients with and without wound disruption were compared for demographic data and comorbidities factors which are reported in Table 2.

The overall rate of postoperative wound disruption was 1.3 %. There was a steady decrease in rate of wound disruption during the study period from 1.6 % in 2005 to 1.1 % in 2013. Overall, 52.9 % of patients with wound disruption underwent reoperation in 30 days after surgery. The median day of diagnosis of wound disruption was 10 days after operation and 43.8 % of wound disruption events happened after discharge from hospital (Fig. 1). Further, wound infection existed in 36.7 % of the patients with wound disruption. Among patients with wound disruption and wound infection, 86.3 % had wound infection prior to wound disruption and 13.7 % developed wound infection after wound disruption. Also, 5.6 % of patients with wound infection had wound disruption (761/13,691).

The risk-adjusted analysis for factors associated with wound disruption is reported in Table 3. The strongest predictor of wound disruption was wound infection (AOR:4.11, P < 0.01). Among comorbid conditions, chronic steroid use had the strongest association with wound disruption. Also the ASA score was associated with wound disruption. Also, patients with a serum albumin level lower than three g/dL had a significantly higher risk of wound disruption compared to patients with a serum albumin level more than 3.5 g/dL (Fig. 2; Table 2). With regard to types of procedures, patients who underwent pelvic exenteration (3.5 %) had the highest rate of wound disruption. Following adjustment, pelvic exenteration was associated with a substantial risk of wound disruption compared to other procedures (AOR: 2.22, P = 0.03).

Following risk adjustment, wound disruption had a significant effect on mortality of patients (5.1 vs. 1.9 %, AOR: 1.46, P = 0.01). Also, patients who had wound disruption had 1 week longer postoperative hospitalization compared to patients without the complication (adjusted mean difference: 7 days, 7.22–7.96, P < 0.01). Complications of sepsis, septic shock, intra-abdominal infections, hospitalization more than 1 month, return to operation room, and deep vein thrombosis were significantly higher in patients with wound disruption (Table 4).



Fig. 1 Timing of postoperative wound after operation in colorectal surgery

Table 3 Risk-adjusted analysis of predictors of wound disruption in colorectal surgery

Variables	Adjusted odds ratio	95 % Confidence interval	P value	
Age				
Age	1	0.99-1	0.08	
Sex				
Male	1.65	1.47-1.85	< 0.01	
Comorbidity				
Chronic steroid use	1.71	1.40-2.09	< 0.01	
Severe chronic obstructive pulmonary disease	1.36	1.12-1.66	< 0.01	
Dyspnea	1.30	1.10-1.52	< 0.01	
Hypertension	1.20	1.05-1.37	< 0.01	
Diabetes mellitus	0.69	0.59-1	0.06	
Anemia	1.17	0.97-1.41	0.08	
Congestive heart failure	0.93	0.60-1.43	0.75	
Disseminated cancer	1.15	0.91-1.45	0.23	
Ascites	1.08	0.71-1.64	0.70	
Dialysis	1.30	0.81-2.07	0.26	
Bleeding disorders	1	0.79-1.26	0.96	
Weight loss	1.12	0.89-1.42	0.32	
Body mass index				
$18.5 \le BMI < 25$	Reference	Reference	Reference	
18.5 < BMI	0.97	0.67-1.39	0.88	
25 < BMI < 30	1.10	0.93-1.29	0.24	
$30 \leq BMI$	1.57	1.34-1.84	< 0.01	
ASA score ^a				
I	Reference	Reference	Referenc	
II	1.73	0.93-3.19	0.07	
III	2.42	1.30-4.50	< 0.01	
VI	3.82	1.75-8.34	< 0.01	
Serum albumin				
Serum albumin level ≥3.5 g/dL	Reference	Reference	Referenc	
$3 \leq$ Serum albumin level < 3.5 g/dL	1.14	0.95-1.38	0.15	
Serum albumin level <3 g/dL	1.27	1.02-1.58	0.02	
Surgical approach				
Open	Reference	Reference	Reference	
Laparoscopic	0.61	0.52-0.70	< 0.01	
Procedure				
Partial colectomy	Reference	Reference	Reference	
Total colectomy	1.25	1.02-1.54	0.03	
Proctectomy	1.36	1.11-1.66	< 0.01	
Pelvic exenteration	2,22	1.04-4.72	0.03	
Type of operation				
Elective	Reference	Reference	Reference	
Urgent/emergent	1.31	1.06-1.62	0.01	
Operation length				
Less than 2 h	Reference	Reference	Reference	
2–3 h	1.17	1-1.37	0.05	
More than 3 h	1.56	1.33-1.84	< 0.01	
Surgeon				
Attending	Reference	Reference	Reference	

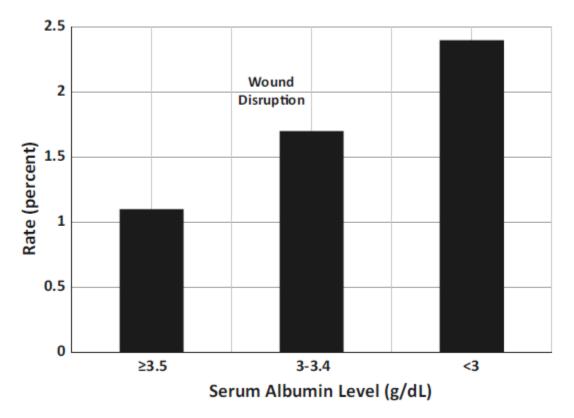
Table	3	continued

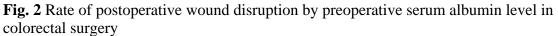
Variables	Adjusted odds ratio	95 % Confidence interval	P value	
Residents	0.87	0.76-1	0.05	
Fellows	0.75	0.61-1	0.06	
Intraoperative transfusion ^b				
No transfusion	Reference	Reference	Reference	
One or two units	1.31	1.02–1.67	0.02	
More than two units	1.51	1.06-2.16	0.02	
Other factors				
Wound infection	4.11	3.62-4.65	< 0.01	
Smoking	1.60	1.39–1.83	< 0.01	
White blood cell count	1.01	0.99-1.02	0.08	
Chemotherapy	0.78	0.54-1.14	0.20	
Radiotherapy	0.87	0.66-1.16	0.37	
Dependency before surgery	1.22	0.99-1.51	0.05	
Preoperative sepsis ^c	0.97	0.76-1.23	0.83	

^a The American society of anesthesiologists score

^b The number of packed or whole red blood cells given during the operative procedure

^c Preoperative sepsis, septic shock, and Systemic Inflammatory Response Syndrome





Discussion

Our analysis reinforces the serious effects of wound disruption on outcomes of surgical patients. Our study shows the mortality of patients with wound disruption to be twice that of patients without wound disruption. Also, we found an increased risk of sepsis, septic shock, prolonged hospitalization, deep vein thrombosis, intra-abdominal infection, and reoperation in patients with wound disruption. High mortality, morbidity, hospitalization length, and hospital cost of patients with wound disruption were reported previously [4, 5, 9, 10]. However, comparison of outcomes of patients with and without wound disruption should be judged with caution as these two groups of patients are not two homogeneous groups of patients and their comorbidities and severity of disease varied broadly. Although we adjusted our results with multiple factors, it is not possible to adjust for all covariants. Nevertheless, investigating high-risk patients and implementing risk factor modification prior to surgery may decrease the morbidity and mortality of patients.

Wound disruption is a preventable morbidity in colorectal surgery. This study introduces a number of perioperative factors which have associations with wound disruption. While previous reports noted low serum albumin level, chronic lung disease, wound infection, and obesity as risk factors of wound disruption in surgical patients [4, 5, 10, 11], we further identified 13 other factors which had associations with wound disruption. Considering

Complication	Patients with wound disruption (%)	Patients without wound disruption (%)	Adjusted odds ratio	95 % Confidence interval	P value
Mortality	5.1	1.9	1.46	1.09-1.95	0.01
Return to operation room	52.9	4.8	15.82	14.02–17.86	< 0.01
Hospitalization >30 days	14.9	1.8	5.72	4.78-6.85	<0.01
Intra-abdominal infections	16.4	3.4	4.61	3.92-5.41	<0.01
Septic shock	10.1	1.6	3.93	3.22-4.81	< 0.01
Sepsis	19.5	3.5	3.85	3.32-4.47	< 0.01
Deep vein thrombosis	4.2	1.2	2.28	1.72-3.03	< 0.01

Table 4 Risk-adjusted analysis of complications were associated with wound disruption

a number of risk factors are reducible, preventive strategies with the focus on reducing the risk factors should be planned. Overall, wound infection is the strongest predictor of wound disruption. We found the risk of wound disruption increases more than four times in presence of wound infection. Effect of wound infection on wound disruption has been cited multiple times in literature [11–13]. Among comorbid conditions, chronic use of steroids was the factor with the strongest association with wound disruption. The adverse effects of corticosteroids on wound healing have been cited previously [14, 15]. The adverse effects are explained by interactions of corticosteroids in the process of fibroblast proliferation, collagen synthesis, angiogenesis, wound contraction, and reepithelialization of surgical wound [15]. In addition, recent studies reported adverse effects of steroid use in colonic anastomosis [16, 17]. We suggest steroids should be tapered out or at least reduced before surgery whenever possible. Vitamin A can ameliorate the deleterious effects of steroids on wound healing when they cannot be reduced [18]. We further demonstrated that the serum albumin level can predict the risk of postoperative wound dehiscence (Fig. 2). Serum albumin level has been linked to the

patients' nutritional status and mortality and morbidity of colorectal patients [19–22]. This correlation is in line with previous reports of an association between malnutrition and wound disruption [4, 9]. Improving the nutritional status in patients with nutritional deficiencies seems logical. However, serum albumin level is not a reliable marker of nutritional status in presence of physiological stress and high CRP levels [23, 24]. Evaluating methods for nutritional status other than serum albumin level should be used for patients with high CRP.

Obesity increases risk of wound dehiscence. We found the lowest rates of wound disruption occur in patients with a normal BMI (18.5–24.9) and the highest rates of wound disruption occur in patients with BMI greater than forty. The association between obesity and wound healing has long been established [25]. Obese patients may benefit from nutritional support with A, B complex, and C vitamins prior to operation [25].

In line with literature, our results show that a laparoscopic approach is associated with a decrease in the risk of wound disruption [26]. Although the inherent selection bias of laparoscopic compared to open surgery makes comparison difficult, we found that laparoscopic surgery is associated with a decrease in risk of wound disruption by 39 % compared to open operation. However, emergently admitted patients more often have wound disruption and it is difficult to establish the feasibility and benefits of a laparoscopic approach for these patients. Utilization of laparoscopic surgery in abdominal emergencies needs more investigation.

Among colorectal procedures, pelvic exenteration has the highest risk of postoperative wound disruption. We found a two-time higher risk of wound disruption after pelvic exenteration compared to a partial colectomy. It can be related to the higher rate of wound infection and disruption in perineal wound compared to abdominal wound [27, 28]. Wound dehiscence was previously reported as a one of the common complications of pelvic exenteration by Wydra [27]. Preventive strategies such as the vacuum wound closure system in high-risk patients undergoing pelvic exenteration warrant consideration. Advantages of vacuum assisted closure on healing of perineal surgical wound have been reported in literature [29, 30]. Although vacuum wound closure does not reduce the bacterial load of surgical wound, a significant reduction of wound surface area using vacuum assisted closure may have positive effects on wound healing [31].

Our study results show that 43.8 % of wound disruption events occur after hospital discharge in colorectal surgery. Also, we found that postoperative day eight was the time with the highest incidence of wound disruption (Fig. 1) which is in line with the previously reported postoperative period of 6–8 day as the most common day of wound disruption by Begum [5]. Considering a significant percentage of wound disruption events occur following hospital discharge, it seems logical to take all necessary steps to prevent this complication after discharge. Currently there is no specific postoperative strategy for prevention of wound disruption.

The treatment of wound disruption is associated with unsatisfactory results [11, 32–34]. The decision is usually made according to the size of the fascia defect, presence or absence of evisceration, general condition of patients, and presence of infection [11]. However, the postoperative incidence rate of incisional hernia has been reported 19–45 % in literature [11, 32–34]. The available options include primary closure of fascia and skin, temporary packing, use of prosthetic mesh, or skin closure only [11,

32]. In about half of cases, as our data also revealed, the patient will receive surgical closure immediately [11, 32, 35]. However, suture repair in patients with obvious tissue necrosis, infection, and loss of the abdominal wall is associated with a 50 % rate of failure [36]. In such patients, we would suggest closure of the wound only after debridement of the necrotic tissue and control of any infection until granulation tissue appears [11, 32]. Nonoperative management with only skin closure, accepting an inevitable hernia, is possible for patients with small defects [11, 32]. Also, if the general status of the patient does not support immediate surgery, non-operative management with temporary packing or skin closure is suggested [11, 32]. We found 52.9 % of patients with wound disruption underwent reoperation within 30 days after surgery. However, considering the retrospective nature of this study due to using patients' discharge data, the reported rate may be lower than the actual rate of need for reoperation following wound disruption. Also, our result shows 43.8 % of wound disruption events occurred after discharge, so some of the patients may have been re-operated in NSQIP nonparticipant hospitals and we could not track them. Finally, we have no data on rate of ultimate hernia formation or hernia repair on this group of patients.

Study limitations

The retrospective nature of this study makes any conclusions difficult. The surgical technique is one of the most important factors associated with wound disruption and the ACS NSQIP database lacks information regarding type and length of surgical incision, place of the surgical incision (abdominal vs. perineal) as well as the closing technique of the fascia and surgical wound [37]. We compared clinical factors of patients with and without wound disruption. However, these two groups of patients were not homogeneous regarding indication for surgery, stage of the disease, type of procedure, and surgical approach. Although we adjusted study results with the multiple factors, we could not adjust them for the stage of the disease which can affect the results. In addition, NSQIP does not provide any details regarding long term outcomes of closure of surgical wound in patients with wound disruption as incisional hernia is reported in 43 % of patients who suffer wound disruption [3]. NSQIP database also does not include some details such as the utilization of drains in surgery and dosage and type of the corticosteroid drugs, which may impact risk for wound disruption [1]. Despite these limitations, in our knowledge, this study is the first nationwide report on wound disruption in colorectal resection procedures using multivariate analysis.

Conclusion

Overall, 1.3 % of colorectal operations are complicated with wound disruption, and 43.8 % of these events happen after hospital discharge. Wound disruption increases mortality and hospitalization length of surgical patients significantly. Wound infection is the strongest predictor of wound disruption. Chronic steroid use, obesity, and serum albumin level are significantly associated with wound disruption. Considering a number of risk factors are reducible, preventive strategies should be designed for high-risk patients. Pelvic exenteration procedure has the highest procedure-specific risk of wound disruption. Strategies to decrease the rate of wound infection and correcting nutritional

deficiencies of patients as well as utilization of laparoscopic surgery may lead to a decreased rate of wound disruption.

References

- 1. Cöl C, Soran A, Cöl M (1998) Can postoperative abdominal wound dehiscence be predicted? Tokai J Exp Clin Med 23:123–127
- 2. Khorgami Z, Shoar S, Laghaie B, Aminian A, Hosseini Araghi N, Soroush A (2013) Prophylactic retention sutures in midline laparotomy in high-risk patients for wound dehiscence: a randomized controlled trial. J Surg Res 180:238–243
- 3. Hahler B (2006) Surgical wound dehiscence. Medsurg Nurs 15:296-300; quiz 301
- 4. Mäkelä JT, Kiviniemi H, Juvonen T, Laitinen S (1995) Factors influencing wound dehiscence after midline laparotomy. Am J Surg 170:387–390
- Begum B, Zaman R, Ahmed M, Ali S (2008) Burst abdomen-A preventable morbidity. Mymensingh Med J 17:63–66
- Marwah S, Marwah N, Singh M, Kapoor A, Karwasra RK (2005) Addition of rectus sheath relaxation incisions to emergency midline laparotomy for peritonitis to prevent fascial dehiscence. World J Surg 29:235–239
- 7. National surgical quality improvement program (2005). American College of Surgeons, Chicago. Cited 17 Jan 2012. www.acsnsqip.org
- 8. User guide for the 2011 participant use data file. American College of Surgeons national surgical quality improvement program (2012). <u>http://site.acsnsqip.org/wp-content/uploads/2012/03/2011-User-Guide_Final.pdf</u>. Cited April 2015
- Rodríguez-Hermosa JI, Codina-Cazador A, Ruiz B, Roig J, Gironè s J, Pujadas M et al (2005) Risk factors for acute abdominal wall dehiscence after laparotomy in adults. Cir Esp 77:280–286
- Shanmugam VK, Fernandez S, Evans KK, McNish S, Banerjee A, Couch K et al (2015) Postoperative wound dehiscence: predictors and associations. Wound Repair Regen 23:184–190
- 11. van Ramshorst GH, Eker HH, Harlaar JJ, Nijens KJ, Jeekel J, Lange JF (2010) Therapeutic alternatives for burst abdomen. Surg Technol Int 19:111–119
- 12. Gürleyik G (2001) Factors affecting disruption of surgical abdominal incisions in early postoperative period. Ulus Travma Derg 7:96–99
- Yahchouchy-Chouillard E, Aura T, Picone O, Etienne JC, Fingerhut A (2003) Incisional hernias. I. Related risk factors. Dig Surg 20:3–9
- 14. Wicke C, Halliday B, Allen D, Roche NS, Scheuenstuhl H, Spencer MM et al (2000) Effects of steroids and retinoids on wound healing. Arch Surg 135:1265–1270
- 15. Anstead GM (1998) Steroids, retinoids, and wound healing. Adv Wound Care 11:277–285
- 16. Baca B, Ozben V, Boler DE, Onur E, Hamzaoglu I, Karahasanoglu T et al (2010) Effect of corticosteroid dose and duration of administration on colonic anastomosis. Inflamm Bowel Dis 16:2162–2167
- Eubanks TR, Greenberg JJ, Dobrin PB, Harford FJ, Gamelli RL (1997) The effects of different corticosteroids on the healing colon anastomosis and cecum in a rat model. Am Surg 63:266–269

- Phillips JD, Kim CS, Fonkalsrud EW, Zeng H, Dindar H (1992) Effects of chronic corticosteroids and vitamin A on the healing of intestinal anastomoses. Am J Surg 163:71–77
- 19. Lohsiriwat V, Chinswangwatanakul V, Lohsiriwat S, Akaraviputh T, Boonnuch W, Methasade A et al (2007) Hypoalbuminemia is a predictor of delayed postoperative bowel function and poor surgical outcomes in right-sided colon cancer patients. Asia Pac J Clin Nutr 16:213–217
- 20. Lohsiriwat V, Lohsiriwat D, Boonnuch W, Chinswangwatanakul V, Akaraviputh T, Lert-Akayamanee N (2008) Pre-operative hypoalbuminemia is a major risk factor for postoperative complications following rectal cancer surgery. World J Gastroenterol 14:1248–1251
- 21. Gibbs J, Cull W, Henderson W, Daley J, Hur K, Khuri SF (1999) Preoperative serum albumin level as a predictor of operative mortality and morbidity: results from the National VA Surgical Risk Study. Arch Surg 134:36–42
- 22. Fuhrman MP, Charney P, Mueller CM (2004) Hepatic proteins and nutrition assessment. J Am Diet Assoc 104:1258–1264
- 23. Gabay C, Kushner I (1999) Acute-phase proteins and other systemic responses to inflammation. N Engl J Med 340:448–454
- 24. Banh L (2006) Serum proteins as markers of nutrition: what are we treating? Pract Gastroenterol 30:46–64
- 25. Pierpont YN, Dinh TP, Salas RE, Johnson EL, Wright TG, Robson MC et al (2014) Obesity and surgical wound healing: a current review. ISRN Obes 2014:638936
- 26. Moghadamyeghaneh Z, Masoomi H, Mills SD, Carmichael JC, Pigazzi A, Nguyen NT, et al (2014) Outcomes of conversion of laparoscopic colorectal surgery to open surgery. JSLS 18:e2014.00230. doi:10.4293/JSLS.2014.00230
- 27. Wydra D, Emerich J, Sawicki S, Ciach K, Marciniak A (2006) Major complications following exenteration in cases of pelvicmalignancy: a 10-year experience. World J Gastroenterol 12:1115–1119
- 28. Artioukh DY, Smith RA, Gokul K (2007) Risk factors for impaired healing of the perineal wound after abdominoperineal resection of rectum for carcinoma. Colorectal Dis 9:362–367
- 29. Cresti S, Ouaïssi M, Sielezneff I, Chaix JB, Pirro N, Berthet B et al (2008) Advantage of vacuum assisted closure on healing of wound associated with omentoplasty after abdominoperineal excision: a case report. World J Surg Oncol 6:136
- 30. Gümüs, N (2009) Vacuum-assisted closure of perineal war wound related to rectum. Eplasty 9:e55
- 31. Mouës CM, Vos MC, van den Bemd GJ, Stijnen T, Hovius SE (2004) Bacterial load in relation to vacuum-assisted closure wound therapy: a prospective randomized trial. Wound Repair Regen 12:11–17
- 32. Cliby WA (2002) Abdominal incision wound breakdown. Clin Obstet Gynecol 45:507–517
- 33. Grace RH, Cox SJ (1973) Incidence of incisional hernia following dehiscence of the abdominal wound. Proc R Soc Med 66:1091–1092
- 34. White H, Cook J, Ward M (1977) Abdominal wound dehiscence. A 10-year survey from a district general hospital. Ann R Coll Surg Engl 59:337–341
- 35. Fleischer GM, Rennert A, Ru⁻hmer M (2000) Infected abdominal wall and burst

abdomen. Chirurg 71:754–762

36. Carlson MA (1997) Acute wound failure. Surg Clin North Am 77:607–636

37. Israelsson LA, Millbourn D (2013) Prevention of incisional hernias: how to close a midline incision. Surg Clin North Am 93:1027–1040

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