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ORIGINAL ARTICLE

Postoperative excessive gain in visceral adipose tissue as well as body mass index are associated with adverse outcomes of an ileal pouch

Ganglei Liu[1,2], Xianrui Wu[1], Yi Li[1], Yuanyi Rui[1], Luca Stocchi[1], Feza H. Remzi[1], Bo Shen[1,3]*

[1]Department of Colorectal Surgery, the Cleveland Clinic Foundation, Cleveland, OH, USA, [2]Department of Geriatric Surgery, the Second Xiangya Hospital, Central South University, Changsha, China and [3]Department of Gastroenterology/Hepatology, the Cleveland Clinic Foundation, Cleveland, OH, USA

*Corresponding author. The Center for Inflammatory Bowel Diseases Digestive Disease and Surgery Institute-A31, Cleveland Clinic Foundation, 9500 Euclid Ave., Cleveland, Ohio 44195, USA. Tel: +1-216-444-9252, Fax: +1-216-444-6305, E-mail: shenb@ccf.org
Author contribution Ganglei Liu: Data gathering and entry, imaging measurement and manuscript preparation; Xianrui Wu: Data monitoring and statistical analysis; Yi Li: imaging measurement and data monitoring; Yuanyi Rui: data entry and manuscript review; Luca Stocchi: critical review of manuscript and supervision of trainees; Feza H. Remzi: critical review and supervision of the project; Bo Shen: concept, general supervision and manuscript revisions

Abstract

Background: There are no published studies on the impact of visceral adipose tissue (VAT) change on outcomes of restorative proctocolectomy and ileal pouch-anal anastomosis (IPAA). The aim of this historic cohort study was to evaluate the impact of excessive VAT gain on the outcomes of inflammatory bowel disease (IBD) patients with IPAA.
Methods: We evaluated all eligible patients with at least two sequential CT scans after pouch construction from our prospectively maintained Pouchitis Registry between 2002 and 2014. The visceral fat area (VFA) was measured on CT images. The study group comprised patients with a significant VAT gain (> 15%), and the control group was those without. The adverse outcomes of the pouch were defined as the new development of chronic pouch inflammation (chronic pouchitis, chronic cuffitis or Crohn's disease of the pouch), anastomotic sinus and the combination of above (the composite adverse outcome) or pouch failure, after the inception CT.
Results: Of 1564 patients in the Registry, 59 (3.8%) with at least 2 CT scans after pouch surgery were included. Twenty-nine patients (49.2%) were in the study group, and 30 (50.8%) were in the control group. The median duration from the inception to the latest CT was 552 (range: 31–2598) days for the entire cohort. We compared the frequency of new chronic pouch inflammation (13.8% *vs* 3.3%, $P = 0.195$), new pouch sinus (10.3% *vs* 0%, $P = 0.112$), composite adverse pouch outcome (24.1% *vs* 3.3%, $P = 0.026$) or pouch failure (10.3% *vs* 6.7%, $P = 0.671$) between the two groups. Kaplan-Meier plot for time-to-pouch failure between the pouch patients with or without excessive body mass index (BMI) gain (> 10%) showed statistical difference ($P = 0.011$). Limited stepwise multivariate analysis showed that excessive VAT gain (odds ratio = 12.608, 95% confidence interval: 1.190–133.538, $P = 0.035$) was an independent risk factor for the adverse pouch comes.
Conclusions: In this cohort of ileal pouch patients, excessive VAT gain as well as gain in BMI after pouch construction was found to be associated with poor long-term outcomes.

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Key words: inflammatory bowel disease; ileal pouch; pouch failure; visceral adipose tissue; visceral fat area

Introduction

, Restorative proctocolectomy with ileal pouch anal anastomosis (IPAA) has become the surgical treatment of choice for patients with ulcerative colitis (UC) or familial adenomatous , polyposis who required colectomy. Although IPAA has been consistently shown to improve patients' health-related quality , of life, a spectrum of adverse sequelae can occur including bowel obstruction, pouch sinus, fistula, chronic pouchitis, Crohn's disease (CD) of the pouch, irritable pouch syndrome and pouch failure with pouch excision, revision or permanent diversion [1].

Reported factors for chronic pouchitis include nucleotide-binding oligomerisation domain 2/ caspase recruitment domains 15 (NOD2/CARD15) gene mutations, perinuclear anti-neutrophil cytoplasmic antibody (p-ANCA), nonsmoking status, backwash ileitis, arthralgia, arthropathy and primary sclerosing cholangitis. Reported risk factors for CD of the pouch include NOD2/CARD15 gene mutation, the presence of family history of CD, and current smoking. The purported risk factors for chronic cuffitis are preoperative toxic megacolon and stapled J-pouch without mucosectomy. Finally, the main causes for pouch failure are surgical complications, chronic pouchitis, CD of the pouch, cuffitis, pouch vaginal fistula and chronic pouch sinus.

Obesity at the time of surgery has been found to be associated with postoperative complications and outcomes in non-pouch CD patients [2–4] as well as poor outcomes in pouch patients [5]. We recently reported that excessive weight gain (> 15%) in IPAA patients was associated with a 69% increased risk for pouch failure [6]. However, obesity or general weight gain is a widespread increase of adipose-tissue hypertrophy. The measurement of weight gain in general or in body mass index (BMI) does not specify the compartment of fat accumulation, particular in visceral adipose tissue (VAT).

The impact of VAT gain on the outcome of IPAA among patients with underlying inflammatory bowel disease (IBD) has not been studied. The current study with the accurate measurement of VAT with CT imaging is the natural extension of our previous studies [6,7]. Clinically, we frequently noticed that patients with a functioning pouch for a long time, then gradually developed chronic pouchitis or even an anastomotic sinus corresponding to an increased waist girth. Therefore we hypothesized that the VAT gain is associated with adverse pouch outcomes. The aim of this historic cohort study was to evaluate the impact of excessive gain in VAT as well as BMI on the outcomes of the IPAA patients with inflammatory bowel diseases (IBD).

Patients and Methods

Patients

Inclusion criteria were pouch patients with (i) underlying IBD; (ii) regular follow-up at our Pouchitis Clinic and (iii) at least two CT scans from the pouch construction to either pouch failure or to date if pouch has survived. If a patient had more than two CT scans after pouch surgery, the CT images of the first and latest were analyzed.

Exclusion criteria were patients with (i) less than two CT scans from pouch construction to either pouch failure or present if pouch has survived, (ii) the duration between the inception and latest CT less than month, (iii) pouch surgery for colon cancer with radiation or chemotherapy; or (iv) underlying familial adenomatous polyposis.

The Cleveland Clinic Institutional Review Board approved the prospectively maintained Pouchitis Registry. Of the 1564 patients in the Registry from 2002 to 2014, 59 (3.8%) met the inclusion criteria and were grouped with those having significant visceral fat area (VFA) gain (> 15% from the inception CT to the latest CT) (the study group) and those without (the control group).

Fat tissue measurement

Among various techniques for body fat measurement, the computed tomography (CT)-based measurement has emerged as having excellent accuracy and precision [8–14]. In this study, details of the CT imaging were obtained from the electronic patient records. Visceral fat area (VFA) and subcutaneous fat area (SFA) were measured retrospectively on the first and latest CT scans performed from pouch construction to either pouch failure or the latest present if the pouch had survived at the level of L3 (**Figure 1**). Briefly, we measured pixels with densities in the −190 HU to −30 HU range in order to delineate the subcutaneous and visceral compartments and to compute the cross-sectional area of each in square centimeters [15–17]. The same technique was used in our previous study in body fat and postoperative CD [7]. These measurements were performed by a researcher blinded to patient information (G. L.).

Demographic and clinical variables

Demographic data included age at pouch construction, sex, ethnicity, smoking history and family history of IBD. Clinical data included the following: "indeterminate colitis (IC)" (i.e. a histopathological diagnosis on proctocolectomy specimens that defied a clear distinction between CD and UC), "significant comorbidities" (i.e. congestive heart failure, coronary bypass surgery, chronic obstructive pulmonary diseases, renal insufficiency, non-gastrointestinal cancer, stroke and liver failure), "chronic pouch inflammation" (i.e. chronic pouchitis, CD of the pouch or chronic cuffitis), "surgical complications" (i.e. conditions that were believed to be caused by surgical techniques), "pouch failure" (i.e. dysfunctional pouch requiring in pouch excision, revision or a permanent diversion and "pouch survival" (i.e. the time from pouch construction to either pouch failure or presence if pouch has survived). The BMI at the inception or latest CT was defined as the nearest available BMI around the inception or latest CT in the database. Toxic megacolon, extraintestinal manifestations, primary sclerosing cholangitis, preoperative anti-tumor necrosis factor (TNF) biological therapy and postoperative use of immumodulators or the biological agents were also included. The definitions of all variables were consistent with our previously published criteria [6,18–25].

Outcome measurement

The adverse outcomes of the pouch were defined as the presence of newly developed chronic pouch inflammation (chronic pouchitis, chronic cuffitis or CD of the pouch), new anastomotic sinus, combination of above (i.e. the composite adverse outcome) or pouch failure at the time of or after the latest CT.

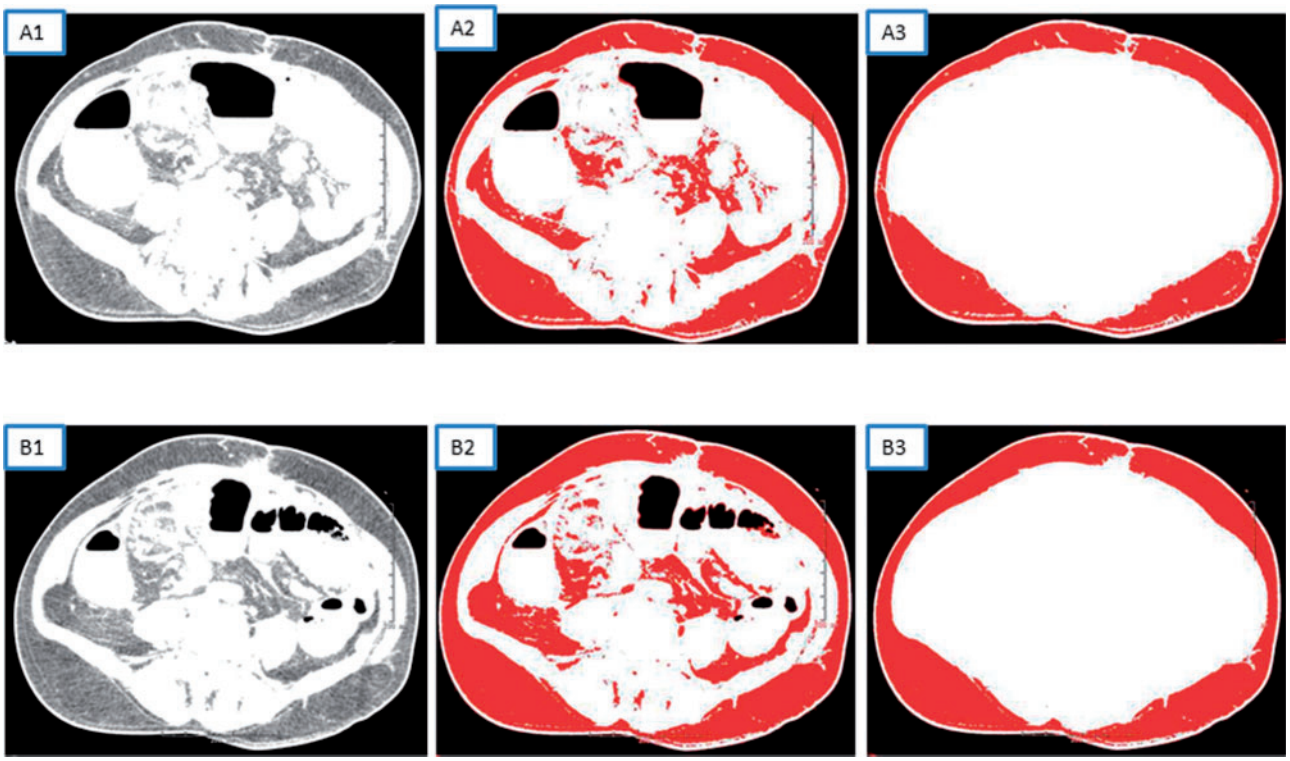

Figure 1. CT measurement of visceral fat area (VFA) and subcutaneous fat area (SFA). Original transverse CT images at the level of L3 (the first CT, **A1**; the latest CT, **B1**). Total fat tissue was extracted from the original image (the first CT, **A2**; the latest CT, **B2**). SFA was extracted from the original image (the first CT, **A3**; the latest CT, **B3**).

Statistical analysis

The data were analyzed with SPSS software, version 16.0 (SPSS Inc., Chicago, IL). Descriptive statistics (median and range, percentages) were computed for all variables in the study. An independent sample *t* test or Wilcoxon rank sum test was used for continuous variables as appropriate. Fisher exact or Chi-square test was used for categorical variables as appropriate. The impact of excessive gain in VAT or BMI on pouch failure or the new onset of composite adverse outcome was depicted with Kaplan–Meier curves with the log-rank test. Bivariate correlation model was used to assess the relationship between variables. A binary logistic regression model was applied to assess the risk factors for adverse pouch outcomes. Due to the small sample size, only three possible risk factors were included in the multivariate analysis. $P < 0.05$ was considered statistically significant.

Results

Of 1564 patients in the registry, 59 (3.8%) with more than one CT scan after pouch surgery were included, with 29 (49.2%) being in the study group and 30 (50.8%) in the control group. The median duration from the pouch construction to the inception CT was 1384 (range: 2– 10 753) days, and the median duration between the inception and the latest CT was 552 (range: 31–2598) days for the whole cohort.

Comparison of demographic and clinical features

Demographic and clinical features were compared between the study and control groups. The median VFA at the inception CT was 50.5 (range: 6.5–223.1) cm^2 in the study group and 57.6 (range: 10.1–202.9) cm^2 in the control group ($P = 0.534$). The median BMI at the inception CT was 24.3 (range: 17.2–39.0) in the study group and 24.9 (range: 16.3–32.8) in the control group ($P = 0.565$). The age at the pouch construction was older in the study group than in the control group (47 [range: 15–71] *vs* 33 [range: 18–55] years, $P = 0.010$). The 1- or 2- stage pouch construction were performed more in the study group than that in the control group (62.1% *vs* 26.7%, $P = 0.009$). Other demographic and clinical variables were comparable between the two groups (Table 1). In bivariate correlation analysis, the BMI change seemed to be correlated with SFA change ($P = 0.003$) but not with the change in VFA ($P = 0.085$) (Table 2).

Comparison of adverse pouch outcomes

The development of new chronic pouch inflammation (13.8% *vs* 3.3%, $P = 0.195$), new pouch sinus (10.3% *vs* 0%, $P = 0.112$), composite adverse pouch outcomes (24.1% *vs* 3.3%, $P = 0.026$) or pouch failure (10.3% *vs* 6.7%, $P = 0.671$) was comparable between the two groups (Table 1). However, Kaplan-Meier plot for the time-to-pouch failure showed statistical difference between the pouch patients with or without excessive BMI gain (> 10%) ($P = 0.002$) (Figure 2). Neither excessive SFA change (>15%) nor excessive VFA change (> 15%) showed a significant impact on the pouch survival (Figure 3 and 4).

The Kaplan-Meier plot for duration from the inception CT to the new onset of composite adverse outcome showed no statistical difference ($P = 0.124$) between patients with or without excessive VFA gain (>15%) (Figure 5).

Limited stepwise multivariable analysis was performed due to the small sample size. In the multivariable analysis model, VFA gain (> 15%) and age at pouch construction and stage of pouch construction were included. The latter two were shown to have significant differences in univariable analysis between

Table 1. Demographic and clinical data

Characteristics	Gain in visceral fat area (N = 29)	No gain in visceral fat area (N = 30)	*p* value
Male	20 (69.0)	17 (56.7)	0.422
Age at pouch construction, years	47 (15–71)	33 (18–55)	0.010
Caucasian	26 (89.7)	28 (93.3)	0.671
IBD duration before pouch construction, years	6 (2–30)	6 (2–41)	0.847
Visceral fat area at the inception CT, cm^2	50.5 (6.5–223.1)	57.6 (10.1–202.9)	0.534
BMI on the inception CT	24.3 (17.2–39.0)	24.9 (16.3–32.8)	0.565
BMI increase > 10% from the inception to latest CT	3 (10.3)	3 (10.0)	1.000
Ever smoked	5 (17.2)	7 (23.3)	0.748
Family history of IBD	4 (13.8)	4 (13.3)	1.000
Concurrent autoimmune disorders	8 (27.6)	4 (13.3)	0.209
Extensive colitis	29 (100.0)	27 (93.3)	0.492
Toxic megacolon	2 (6.9)	2 (6.7)	1.000
Primary sclerosing cholangitis	3 (10.3)	4 (13.3)	1.000
Extraintestinal manifestations other than liver	8 (28.3)	13 (43.3)	0.284
Liver transplantation	1 (3.4)	3 (10.0)	0.612
Significant comorbidities	5 (17.2)	4 (13.3)	0.731
Precolectomy diagnosis			0.271
Ulcerative colitis	28 (96.6)	25 (83.3)	
Indeterminate colitis	1 (3.4)	4 (13.3)	
Crohn's colitis	0 (0)	1 (3.3)	
J pouch	26 (89.7)	27 (90.0)	1.000
Stage of the pouch			0.009
1 or 2	18 (62.1)	8 (26.7)	
3 or redo	11 (37.9)	22 (73.3)	
Preoperative biological therapy	4 (13.8)	9 (30.0)	0.209
Postoperative use of immunomodulators	4 (13.8)	5 (16.7)	1.000
Postoperative use of biological therapy	2 (6.9)	2 (6.7)	1.000
Chronic NSAID use, n (%)	2 (6.9)	0 (0)	0.237
Duration from pouch construction to the first CT, days	1841 (5–8280)	908 (2–10753)	0.162
Duration from the inception CT to the latest CT, days	742 (42–2598)	523 (31–2292)	0.454
Duration from pouch construction to the latest visit, years	9 (1–28)	6 (0 – 34)	0.097
Pouch disease at the first/inception CT			
Irritable pouch syndrome	1 (3.4)	1 (3.3)	1.000
Acute pouchitis	4 (13.8)	1 (3.3)	0.195
Chronic pouchitis	0 (0)	2 (6.7)	0.492
Crohn's disease of the pouch	1 (3.4)	2 (6.7)	1.000
Chronic cuffitis	0 (0)	1 (3.3)	1.000
Surgical complications	17 (58.6)	13 (43.3)	0.301
Pouch disease at the latest CT			
Irritable pouch syndrome	1 (3.4)	2 (6.7)	1.000
Acute pouchitis	4 (13.8)	4 (13.3)	1.000
Chronic pouchitis	0 (0)	2 (6.7)	0.492
Crohn's disease of the pouch	2 (6.9)	3 (10.0)	1.000
Chronic cuffitis	3 (10.3)	2 (6.7)	0.671
Surgical complications	17 (58.6)	14 (46.7)	0.438
*New chronic pouch inflammation	4 (13.8)	1 (3.3)	0.195
New pouch sinus	3 (10.3)	0 (0)	0.112
*New chronic pouch inflammation, or new pouch sinus (composite adverse outcome)	7 (24.1)	1 (3.3)	0.026
Pouch failure	3 (10.3)	2 (6.7)	0.671

Value presented as median (range) for continuous variables and cases (%) for categorical variables.
NSAID: non-steroidal anti-inflammatory drugs
*New development of chronic pouchitis, chronic cuffitis or Crohn's disease of the pouch

Table 2. Pearson correlation coefficients analysis between changes in body mass index and visceral and subcutaneous fat

Characteristic	Change in body mass index (%)	
	Correlation coefficients	*p* value
Change in visceral fat area (VFA) (%)	0.226	0.085
Change in subcutaneous fat area (SFA) (%)	0.378	0.003

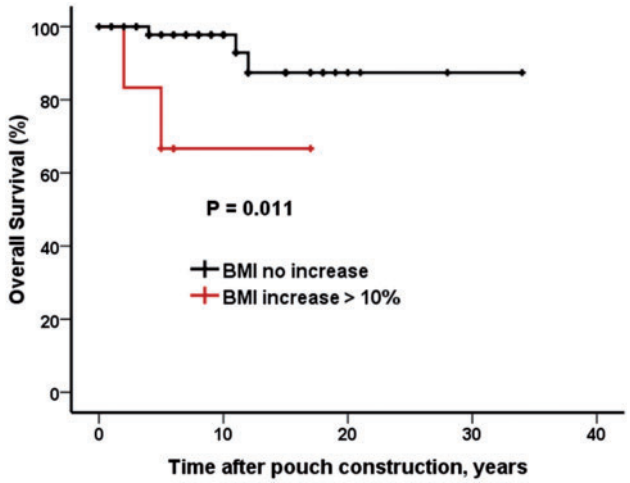

Figure 2. Kaplan-Meier plot for the time-to-pouch failure between patients with or without excessive body mass index (BMI) gain.

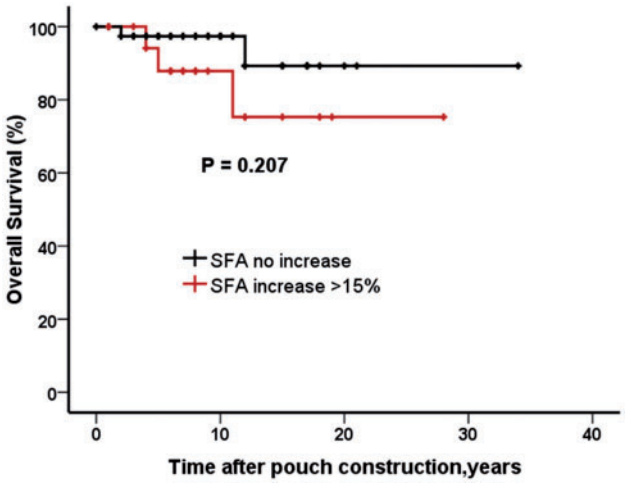

Figure 4. Kaplan-Meier plot for time-to-pouch failure between patients with or without excessive subcutaneous fat area (SFA) gain.

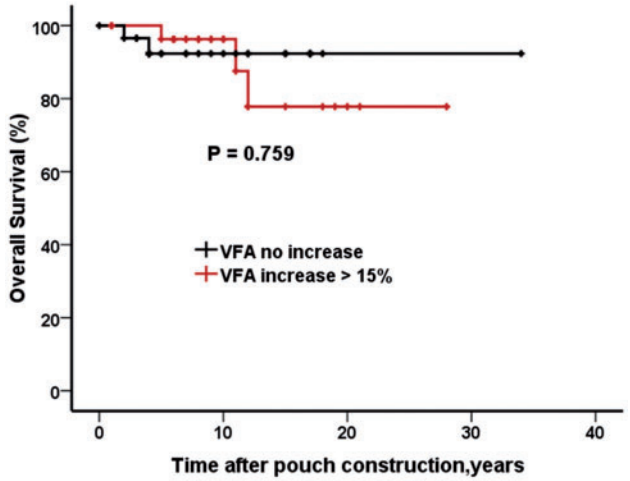

Figure 3. Kaplan-Meier plot for time-to-pouch failure between patients with or without excessive visceral fat area (VFA) gain.

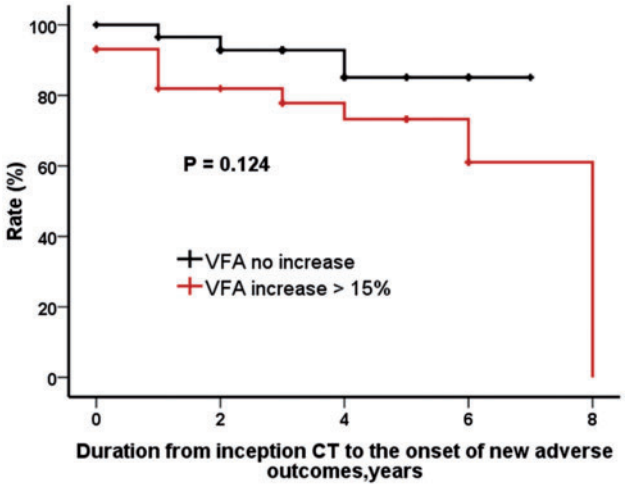

Figure 5. Kaplan-Meier plot for duration from the inception CT to the onset of new adverse outcome between patients with or without excessive visceral fat area (VFA) gain. The new adverse outcome was defined as new development of chronic pouchitis, chronic cuffitis, Crohn's disease of the pouch, new sinus or pouch failure.

the two groups. The stepwise multivariate analysis showed that an excessive VAT gain (odds ratio [OR] = 12.608, 95% confidence interval [CI]: 1.190–133.538, $P = 0.035$) was an independent risk factor for the composite adverse pouch outcomes. Age at the pouch construction (OR = 1.01, 95%CI: 0.955–1.067, $P = 0.737$) and stage of pouch construction (OR = 2.999, 95%CI: 0.546–16.479, $P = 0.206$) were not shown to be significant risk factors (Table 3).

Discussion

In our current study of 59 eligible patients, 29 (49.2%) were in the study group and 30 (50.8%) in the control group. The median VFA at the inception CT was comparable in the study and control groups. The median BMI at the inception CT was also comparable between the study and control groups. However, the patient's age at the time pouch construction was older in the study group than that in the control group. More patients in the study group underwent one- or two- stage pouch construction. Interestingly, we did not find a significant correlation between the change in BMI and the change in VFA despite correlated change in BMI and SFA. Kaplan-Meier plots showed that excessive BMI gain was a risk factor for pouch failure. In addition, the stepwise multivariate analysis showed the VAT gain was an independent risk factor for the composite adverse pouch outcome.

Adipose tissue has been considered an active endocrine and metabolic organ, not just a simple reservoir of excess calories [26,27]. General adipose-tissue accumulation in obese patients was found to be associated with systemically increased pro-inflammatory mediators and humoral or cellular changes within the compartment, which led to the concept of obesity being a chronic inflammatory state. Obesity (BMI $\geq$30) has been reported to be associated with an increased risk of overall and pouch-related complications following IPAA. Several reports also suggested that high BMI was associated with poor clinical outcomes in non-pouch CD patients [2–4]. The fat accumulation in CD was found to be localized and was independent of body weight [28]. Even though CD patients may have a normal or low BMI, they often have creeping fat with hyperplasia of the mesenteric fat adjacent to the inflamed segments of the intestine—

Table 3. Limited multivariate analysis for possible risk factors associated with composite adverse pouch outcomes

Characteristic	Composite adverse pouch outcome*	
	Odds ratio (95% confidence interval)	*p* value
Visceral fat area gain (> 15%)	12.608 (1.190–133.538)	0.035
Age at pouch construction	1.010 (0.955–1.067)	0.737
Stage of pouch construction	2.999 (0.546–16.479)	0.206

*The development of newly developed chronic pouch inflammation (chronic pouchitis, chronic cuffitis or Crohn's disease of the pouch), or new anastomotic sinus, after the inception CT.

an almost pathognomonic feature of the disease. Excessive VAT has been shown to be associated with an increased risk for postoperative recurrence of CD [7]. Our current study showed that excessive visceral fat gain after the surgery is associated with adverse pouch outcomes.

The notion that "obesity is paired with being unhealthy" has limitations,[26] and the measurement of BMI *per se* does not reflect the true change in fat compartments, particularly in VAT [29]. Compared with the metabolically unhealthy obese, the metabolically healthy obese is often characterized by a more favorable inflammatory status, less visceral fat, less infiltration of macrophages into adipose tissue and smaller adipocyte cell size [26]. Furthermore, a previous study showed that VAT was more strongly correlated with waist circumference than with BMI [30]. We believe that visceral fat is a more accurate reflection of the metabolic condition than BMI in pouch or non-pouch IBD patients.

Subcutaneous adipose tissue (SAT) and VAT, two components of adipose tissue, have different metabolic impacts [27]. VAT secretes more pro-inflammatory cytokines and less adiponectin. VAT effuses more free fatty acids into the circulation than SAT [31]. VAT has been shown to be a determinant of colorectal neoplasia [32,33], along with insulin resistance [31,34]. The past decade has witnessed the macroscopic, histologic and molecular evidence of involvement of mesenteric adipose tissue in the pathogenesis of IBD [35]. CD patients with a normal or low BMI may still have creeping mesenteric fat adjacent to the inflamed segments of the intestine [28,36]. Corresponding to the fat accumulation and inflammatory activity, creeping fat was found to be associated with transmural inflammation, fibrosis, muscular hypertrophy and stricture formation [35,37,38].

To date, there are no published studies about the impact of VAT change on the outcome of ileal pouch patents with underlying IBD. In our previous study, excessive weight gain, regardless of BMI, was found to be associated with an increased risk of worse pouch outcomes [6]. The current study is a natural follow-up of our prior study. The results of the current study suggest that it is VAT, rather than cutaneous fat, which contributes to the poor pouch outcomes. The gain in VAT (> 15%) was found to be a risk factor for the adverse pouch outcomes, which may be associated with increased pro-inflammatory activity from visceral adipose tissue in those patients. According to the current literature, VAT from patients with UC and CD appears to be different in its morphology and molecular profile [39]. In this study, we did not find significant difference in the development of CD of the pouch between those with or without VFA gain.

In our current study, the change in BMI seemed to correlate with the change in SFA instead of VFA. Regardless the measurement, the change in SFA may contribute more to the change in BMI. However, a previous study has shown that diet and exercise caused preferential fat loss, more so from VAT than from SAT [28]. To address this controversy, we need more data to find the relationship between BMI change and VAT change among pouch patients. We found that excessive BMI gain (> 10%) was associated with a higher risk of pouch failure in this study.

The findings of the current study have clinical implications. In our clinical practice, we have encountered patients with a long-functioning pouch who gradually developed chronic pouchitis, CD-like conditions of the pouch, and even late-onset pouch anastomotic sinus. We reviewed life events of those patients and found that one common phenomenon was excessive weight gain, especially in male patients with an enlarged waist girth. In this study, we confirmed that excessive gain in VFA or BMI was associated with worse pouch outcomes. Proper diet control, exercise and weight loss may help maintain the health status of an ileal pouch. Furthermore, the body weight and abdominal birth of all pouch patients should be monitored closely. However, monitoring VFA with CT scan can be challenging due to the radiation risk and cost. It is possible to develop less costly and less invasive modalities such as ultrasound. Nonetheless, the findings of our current study will further our knowledge in the impact of adipose tissue on various adverse pouch outcomes. Finally, the findings of the study support our speculation that chronic pouch inflammation and anastomotic sinus are very likely associated with tissue ischemia, hypoxia or fat accumulation; in some patients, dysbiosis with dominant anaerobes and Clostridium difficile infection, may be the secondary event leading to mesenteric tissue inflammation.

There are limitations to our study. First, not all patients in our Pouch Registry routinely had CT imaging after the pouch construction. Those with more than one CT scan might have a diseased condition of the pouch. This might have resulted in referral bias. Of 1564 patients in the registry, only 59 (3.8%) had more than two CT scans after pouch surgery, which might have resulted in selection bias. A longitudinal, consecutive study design with the patients being on their own control may help reduce the noise from the referral pattern. Secondly, there might be type II errors due to the small sample size. The small sample size precluded the inclusion of all possible risk factors such as the use of immunosuppressive agents. Similarly, the small sample size restricted the authors' choice of combining several adverse outcomes together for the purpose of statistical analysis. Finally, the causal relationship between fat or weight gain and the adverse pouch outcomes may be validated with a longitudinal, interventional study with weight reduction. Nonetheless, the findings of this study send a signal that mesenteric fat can be an important component in the disease process of the pouch.

In conclusion, excessive postoperative gain in VAT as well as BMI was found to be associated with adverse outcomes in this cohort of ileal pouch patients. We recommend that the body weight, especially waist girth, be carefully monitored in patients with IPAA. It is possible that weight control may help maintain the health status of the ileal pouch.

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Conflict of interest statement: none declared.

References

1. Shen B. Diagnosis and management of postoperative ileal pouch disorders. *Clin Colon Rectal Surg* 2010;**23**:259–68.

2. Causey MW, Johnson EK, Miller S, *et al*. The impact of obesity on outcomes following major surgery for Crohn's disease: an American College of Surgeons National Surgical Quality Improvement Program assessment. *Dis Colon Rectum* 2011;**54**:1488–95.
3. Hass DJ, Brensinger CM, Lewis JD, *et al*. The impact of increased body mass index on the clinical course of Crohn's disease. *Clin Gastroenterol Hepatol* 2006;**4**:482–8.
4. Blain A, Cattan S, Beaugerie L, *et al*. Crohn's disease clinical course and severity in obese patients. *Clin Nutr* 2002;**21**:51–7.
5. Klos CL, Safar B, Jamal N, *et al*. Obesity increases risk for pouch-related complications following restorative proctocolectomy with ileal pouch-anal anastomosis (IPAA). *J Gastrointest Surg* 2014;**18**:573–9.
6. Wu XR, Zhu H, Kiran RP, *et al*. Excessive weight gain is associated with an increased risk for pouch failure in patients with restorative proctocolectomy. *Inflamm Bowel Dis* 2013;**19**: 2173–81.
7. Li Y, Zhu W, Gong J, *et al*. Visceral fat area is associated with a high risk for early postoperative recurrence in Crohn's disease. *Colorectal Dis* 2015;**17**:225–34.
8. Walker GE, Marzullo P, Ricotti R, *et al*. The pathophysiology of abdominal adipose tissue depots in health and disease. *Horm Mol Biol Clin Investig* 2014;**19**:57–74.
9. Ellis KJ. Human body composition: in vivo methods. *Physiol Rev* 2000;**80**:649–80.
10. O'Leary D. Health care today and tomorrow. *Soc Work Health Care* 1991;**15**:19–30.
11. Shen W, Punyanitya M, Chen J, *et al*. Visceral adipose tissue: relationships between single slice areas at different locations and obesity-related health risks. *Int J Obes (Lond)* 2007;**31**:763–9.
12. Demerath EW, Shen W, Lee M, *et al*. Approximation of total visceral adipose tissue with a single magnetic resonance image. *Am J Clin Nutr* 2007;**85**:362–8.
13. Gradmark AM, Rydh A, Renstrom F, *et al*. Computed tomography-based validation of abdominal adiposity measurements from ultrasonography, dual-energy X-ray absorptiometry and anthropometry. *Br J Nutr* 2010;**104**:582–8.
14. Kullberg J, Brandberg J, Angelhed JE, *et al*. Whole-body adipose tissue analysis: comparison of MRI, CT and dual energy X-ray absorptiometry. *Br J Radiol* 2009;**82**:123–30.
15. Guiu B, Petit JM, Bonnetain F, *et al*. Visceral fat area is an independent predictive biomarker of outcome after first-line bevacizumab-based treatment in metastatic colorectal cancer. *Gut* 2010;**59**:341–7.
16. Malietzis G, Aziz O, Bagnall NM, *et al*. The role of body composition evaluation by computerized tomography in determining colorectal cancer treatment outcomes: A systematic review. *Eur J Surg Oncol* 2015;**41**:186–96.
17. Li Y, Zhu W, Gong J, *et al*. Visceral fat area is associated with a high risk for early postoperative recurrence in Crohn's disease. *Colorectal Dis* 2015;**17**:225–34.
18. Li Y, Zhu H and Shen B. Diagnosis and management of Crohn's disease of the ileal pouch. *Minerva Gastroenterol Dietol* 2012;**58**:123–35.
19. Shen B, Remzi FH, Lavery IC, *et al*. Administration of adalimumab in the treatment of Crohn's disease of the ileal pouch. *Aliment Pharmacol Ther* 2009;**29**:519–26.
20. Appau KA, Fazio VW, Shen B, *et al*. Use of infliximab within 3 months of ileocolonic resection is associated with adverse postoperative outcomes in Crohn's patients. *J Gastrointest Surg* 2008;**12**:1738–44.
21. Shen B, Fazio VW, Remzi FH, *et al*. Risk factors for diseases of ileal pouch-anal anastomosis after restorative proctocolectomy for ulcerative colitis. *Clin Gastroenterol Hepatol* 2006;**4**:81–9; quiz 2–3.
22. Shen B, Fazio VW, Remzi FH, *et al*. Risk factors for clinical phenotypes of Crohn's disease of the ileal pouch. *Am J Gastroenterol* 2006;**101**:2760–8.
23. Zhu H, Wu XR, Queener E, *et al*. Clinical value of surveillance pouchoscopy in asymptomatic ileal pouch patients with underlying inflammatory bowel disease. *Surg Endosc* 2013;**27**: 4325–32.
24. Wu B, Lian L, Li Y, *et al*. Clinical course of cuffitis in ulcerative colitis patients with restorative proctocolectomy and ileal pouch-anal anastomoses. *Inflamm Bowel Dis* 2013;**19**:404–10.
25. Shen B, Achkar JP, Lashner BA, *et al*. Irritable pouch syndrome: a new category of diagnosis for symptomatic patients with ileal pouch-anal anastomosis. *Am J Gastroenterol* 2002;**97**:972–7.
26. Navarro E, Funtikova AN, Fito M, *et al*. Can metabolically healthy obesity be explained by diet, genetics and inflammation? *Mol Nutr Food Res* 2015;**59**:75–93.
27. Gregor MF and Hotamisligil GS. Inflammatory mechanisms in obesity. *Annu Rev Immunol* 2011;**29**:415–45.
28. Kredel LI and Siegmund B. Adipose-tissue and intestinal inflammation - visceral obesity and creeping fat. *Front Immunol* 2014;**5**:462.
29. Beechy L, Galpern J, Petrone A, *et al*. Assessment tools in obesity - psychological measures, diet, activity, and body composition. *Physiol Behav* 2012;**107**:154–71.
30. Janssen I, Heymsfield SB, Allison DB, *et al*. Body mass index and waist circumference independently contribute to the prediction of nonabdominal, abdominal subcutaneous, and visceral fat. *Am J Clin Nutr* 2002;**75**:683–8.
31. Riondino S, Roselli M, Palmirotta R, *et al*. Obesity and colorectal cancer: role of adipokines in tumor initiation and progression. *World J Gastroenterol* 2014;**20**:5177–90.
32. Keum N, Lee DH, Kim R, *et al*. Visceral adiposity and colorectal adenomas: dose-response meta-analysis of observational studies. *Ann Oncol* 2015;**26**:1101–9.
33. Oh TH, Byeon JS, Myung SJ, *et al*. Visceral obesity as a risk factor for colorectal neoplasm. *J Gastroenterol Hepatol* 2008;**23**:411–7.
34. Donohoe CL, Doyle SL and Reynolds JV. Visceral adiposity, insulin resistance and cancer risk. *Diabetol Metab Syndr* 2011;**3**:12.
35. Fink C, Karagiannides I, Bakirtzi K, *et al*. Adipose tissue and inflammatory bowel disease pathogenesis. *Inflamm Bowel Dis* 2012;**18**:155–7.
36. Ungar B, Kopylov U, Goitein D, *et al*. Severe and morbid obesity in Crohn's disease patients: prevalence and disease associations. *Digestion* 2013;**88**:26–32.
37. Peyrin-Biroulet L, Chamaillard M, Gonzalez F, *et al*. Mesenteric fat in Crohn's disease: a pathogenetic hallmark or an innocent bystander? *Gut* 2007;**56**:577–83.
38. Sheehan AL, Warren BF, Gear MW, *et al*. Fat-wrapping in Crohn's disease: pathological basis and relevance to surgical practice. *Br J Surg* 1992;**79**:955–8.
39. Zulian A, Cancello R, Ruocco C, *et al*. Differences in visceral fat and fat bacterial colonization between ulcerative colitis and Crohn's disease. An in vivo and in vitro study. *PLoS One* 2013;**8**:e78495.