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ABOUT THE AUTHOR

Joëlle St-Pierre, MD, PhD

Dr. Joëlle St-Pierre, MD, PhD is a Clinical Assistant Professor at the University of Calgary with a clinical and research focus on IBD. She completed her gastroenterology fellowship at the University of Calgary and an Advanced IBD Fellowship at the University of Chicago. Dr. St-Pierre's main research focus includes examining the role of visceral adiposity and metabolic dysregulation in IBD pathogenesis and treatment response, and the application of point-of-care intestinal ultrasound to monitor inflammation and clinical outcomes. She is an active member of the science committee of the Intestinal Ultrasound Group of the United States and Canada (IUSCAN) and seeking to advance ultrasound-based monitoring and education in IBD.

Affiliations: Inflammatory Bowel Disease Unit, University of Calgary, Calgary, AB, Canada

Obesity in Inflammatory Bowel Disease (IBD): Recognizing a Critical Modifier In Modern Disease Management

Joëlle St-Pierre, MD, PhD

Introduction: A Shifting Landscape in IBD And Obesity

The notion of obesity as a disease remains controversial. A recent consensus from the *Lancet Diabetes & Endocrinology Commission* reframes obesity by distinguishing between "preclinical obesity," defined as a state of excess adiposity with preserved organ function, and "clinical obesity," defined as a chronic, systemic illness caused by excess adiposity and characterized by measurable dysfunction in organ systems or limitations in daily living activities.[1] This distinction provides a medically meaningful basis to identify when obesity constitutes a disease in its own right.

Historically, inflammatory bowel disease (IBD) was associated with undernutrition and weight loss, a reflection of both disease activity and malabsorption. However, with shifting demographics, improved therapeutic options, and global lifestyle changes, obesity has emerged as an increasingly relevant coexisting condition in patients with IBD. While the current prevalence of overweight and obesity among Canadians with IBD remains unknown, population-level data from Statistics Canada show that 35.8% of adults in urban centers are classified as overweight, and 29.0% as obese.[2]

This epidemiologic shift has important clinical ramifications. Obesity contributes to systemic inflammation and is associated with increased healthcare utilization and reduced quality of life (QoL), which are burdens already faced by patients with IBD. The intersection of these two chronic conditions introduces complex challenges for disease management,

health outcomes, and healthcare systems. This review explores the clinical impact of obesity in patients with IBD, including its influence on disease phenotype, treatment response, surgical outcomes, and QoL.

Measuring Obesity in IBD: Moving Beyond BMI

While body mass index (BMI) remains the most common clinical tool for classifying overweight (BMI ≥25 kg/m^2) and obesity (BMI ≥30 kg/m^2), it fails to distinguish between lean and fat mass or account for fat distribution and adipose tissue function. In a meta-analysis comparing anthropometric tools to imaging standards, BMI showed low sensitivity (51.4% in women, 49.6% in men) albeit high specificity (95.4% and 97.3%, respectively) for detecting obesity.[3] Waist circumference and waist-to-height ratio had similar limitations, with modest sensitivity and variable specificity.

In IBD, visceral adipose tissue (VAT), and specifically mesenteric adipose tissue (MAT), is emerging as a critical player in disease biology. Mesenteric fat is known to expand in inflammation, a phenomenon known as "creeping fat." This "creeping fat," first described by Crohn himself,[4] has been associated with stricturing phenotypes and may predict postoperative recurrence.[5-7] MAT lies adjacent to inflamed bowel, making it more than a systemic marker of adiposity; it may actively drive local inflammation by producing cytokines such as interleukin (IL)-6, tumour necrosis factor (TNF)-α, and leptin that amplify intestinal inflammation.[7] Yet MAT and VAT remain largely invisible when relying on BMI.

Imaging tools such as dual-energy X-ray absorptiometry (DXA), magnetic resonance imaging (MRI), computed tomography (CT) and point-of-care ultrasound offer more granular insight into adipose tissue distribution and composition. Though not yet standard in routine care, they are increasingly used in research. Future studies should prioritize validating these direct measures of adiposity as clinical biomarkers to improve prognostication and guide personalized management in IBD. Many of these assessments could be incorporated into clinical practice using imaging-based tools such as point-of-care ultrasound, which is increasingly utilized for bedside evaluation in IBD.

Clinical Consequences of Obesity in IBD

Impact on Disease Phenotype

Obesity appears to influence disease phenotype. In ulcerative colitis (UC), obesity has been linked to more extensive disease, particularly pancolitis.[8] In Crohn's disease (CD), paradoxically, higher BMI has been linked to a lower risk of penetrating or fistulizing complications in some cohorts.[9] However, increased MAT in CD has been associated with transmural inflammation, stricturing phenotypes, and postoperative recurrence.[6] This further supports the hypothesis that body composition rather than body size may better define disease phenotype, highlighting the need for imaging-based adiposity measures.

Obesity and IBD-related Complications

The relationship between obesity and IBD-related complications is complex. Several large cohort studies have found no association between obesity and increased steroid use, adverse events, emergency visits, hospitalization, or IBD-related surgery.[10-14] In UC, patients with obesity were found to have a lower proportion of years with chronic active disease, were less likely to be prescribed anti-TNF therapy, and had lower rates of hospitalization or surgery.[8,15]

Conversely, other studies have shown that higher BMI is associated with persistent disease activity, relapse, and a higher risk of colectomy.[16] In patients with IBD and Clostridioides difficile infection, obesity is associated with longer hospital stays, increased colectomy rates, and increased healthcare costs.[17]

Obesity and Response to Therapy

Despite theoretical concerns that obesity may attenuate response to therapy through altered pharmacokinetics and a pro-inflammatory cytokine milieu, current evidence remains mixed. In a large multicenter cohort of over 3,000 biologic-treated patients with IBD, obesity was not associated with an increased risk of hospitalization, surgery, or serious infections within one year of biologic initiation (including TNF antagonists, vedolizumab, and ustekinumab).[13] Similarly, a pooled individual

participant data analysis from randomized clinical trials of infliximab (ACCENT-I/II, SONIC, ACT-1/2) found no association between obesity and rates of clinical or endoscopic outcomes in either CD or UC.[10]

In contrast, a study of infliximab-treated patients with CD has shown that higher VAT was independently associated with a reduced likelihood of mucosal healing after induction therapy.[18] A large real-world study using the TriNetX database has demonstrated that obesity was significantly associated with higher risks of therapy failure across multiple advanced therapies in UC, including TNF antagonists, vedolizumab, ustekinumab, and Janus kinase (JAK) inhibitors.[19] These patients had higher rates of corticosteroid use, therapy switching, and colectomy within two years compared to propensity-matched non-obese controls (adjusted hazard ratios [HR]s ranged from 1.26 to 1.38 depending on therapy).[19]

Evidence for small molecule therapies remains limited. In a post hoc analysis of OCTAVE, BMI did not affect treatment efficacy or safety in patients with UC, with similar remission and response rates across BMI categories.[20] Further studies are needed to disentangle the roles of pharmacokinetic variability and obesity-related pathophysiology. Future work should integrate clinical outcomes with mechanistic measures such as VAT distribution, adipokine profiles, and drug levels to optimize therapy in this population.

Surgical Risk and Outcomes in Patients with IBD and Obesity

Obesity is increasingly recognized as a contributor to adverse surgical outcomes in patients with IBD. A meta-analysis of over 12,000 patients has shown that obesity was associated with increased risks of overall postoperative complications (odds ratio [OR] 1.45, 95% confidence interval [CI] 1.15–1.84), infectious complications (OR 1.48, 95% CI 1.17–1.88), and conversion to laparotomy (OR 1.90, 95% CI 1.32–2.72).[21] Beyond BMI, body fat distribution also appears to influence outcomes. A high subcutaneous-to-visceral fat ratio was independently associated with postoperative infectious complications in CD (OR 2.01, 95% CI 1.20–3.19).[22] In addition, patients with excessive visceral fat area had more than twice the risk of endoscopic recurrence at 18 months following surgery (relative risk [RR] 2.1, 95% CI 1.5–3.0).[23]

These findings have led to growing interest in the mesentery as a surgical target in CD. Extended mesenteric resection, which involves the removal of affected mesenteric fat along with the bowel segment, has been proposed as a strategy to reduce disease recurrence. A recent meta-analysis of 4,358 patients has found that extended mesenteric resection significantly reduced surgical recurrence compared to mesenteric preservation (OR 4.94, 95% CI 2.22–10.97; I^2 = 0%) without increasing postoperative morbidity or length of hospital stay.[24] Together, these data support incorporating visceral adiposity assessment into preoperative planning and suggest that targeting mesenteric disease may help reduce postoperative complications and recurrence in IBD surgery.

Obesity and Metabolic Comorbidities in IBD

Obesity, while not the sole defining feature of metabolic syndrome (MetS), is a central component. Its rising prevalence in the global population has prompted growing interest in associated metabolic comorbidities in patients with IBD, including MetS, type 2 diabetes mellitus (T2DM), and metabolic dysfunction-associated steatotic liver disease (MASLD).

A recent meta-analysis estimated the pooled prevalence of MetS in patients with IBD to be 19.4% (95% CI 15.1–23.8%), with significantly higher rates in UC compared to CD (38.2% vs. 13.6%).[25] In a large prospective cohort, the prevalence of T2DM among patients with IBD was approximately 5%, and its presence was associated with greater systemic inflammation, worse clinical disease activity, lower QoL, and increased healthcare utilization.[26] In a meta-analysis including over 14,000 patients with IBD, the global pooled prevalence of MASLD in patients with IBD was 30.7%, nearly twice the odds compared to healthy controls (OR 1.96, 95% CI 1.13–3.41).[27] Moreover, 13.6% of patients with IBD and MASLD had advanced liver fibrosis. Higher BMI was significantly associated with increased risk of MASLD in patients with IBD, with a pooled adjusted odds ratio of 1.27 (95% CI 1.22–1.32), reinforcing the contribution of obesity to hepatic comorbidity in this population.

Given the prevalence of these conditions in IBD and their strong association with obesity, routine screening for metabolic comorbidities should be considered in patients with IBD and elevated adiposity to identify high-risk

Domain	Key Findings	Clinical Implications	Research Gaps
Assessment of Obesity	BMI poorly reflects fat distribution and adipose function. VAT and MAT are more strongly linked to IBD outcomes.	Clinicians should consider tools that reflect adiposity, such as imaging-based tools, rather than rely on BMI alone.	Validation of VAT/MAT measures as biomarkers. Need for definition of clinical thresholds for risk stratification.
Disease Phenotype	Obesity is linked to more extensive UC. MAT is associated with stricturing CD.	VAT/MAT imaging may help refine IBD phenotyping and prognosis.	Need for prospective studies linking fat distribution to disease behaviour and histology.
IBD-Related Complications	Findings are mixed: some show no effect; others report higher relapse or colectomy risk.	Consider individual body composition and comorbidities when evaluating prognosis.	Harmonize definitions and stratify by fat distribution and metabolic profiles in future studies.
Response to Therapy	Obesity may attenuate the response to biologics, but JAK inhibitors appear to be weight-neutral.	Account for adiposity when selecting or optimizing therapies.	Study pharmacokinetic mechanisms; integrate adiposity and drug levels in treatment-response models.
Surgical Outcomes	Obesity and VAT increase the risk of complications and post-op recurrence.	Use VAT assessment in pre-op planning; consider mesenteric resection in select CD cases.	Prospective trials evaluating mesenteric resection vs. preservation and its long-term outcomes.
Metabolic Comorbidities	MetS, T2DM, and MASLD are more prevalent in patients with IBD and obesity.	Screen for metabolic diseases in patients with elevated adiposity.	Determine the impact of metabolic disease control on IBD outcomes.
Quality of Life	Obesity and VAT are associated with worse patient-centered outcomes; lifestyle modification is beneficial.	Address body composition and lifestyle in routine care.	Longitudinal studies on the QoL impact of weight loss and body composition changes.

Table 1. Summary of Key Findings, Clinical Implications, and Research Gaps Related to Obesity in IBD; *courtesy of Joëlle St-Pierre, MD, PhD.*

Abbreviations: IBD: Inflammatory bowel disease, **UC:** ulcerative Colitis, **CD:** Crohn's disease, **BMI:** body mass index, **VAT:** visceral adipose tissue, **MAT:** mesenteric adipose tissue, **MetS:** metabolic syndrome, **MASLD:** metabolic dysfunction-associated steatotic Liver Disease, **T2DM:** type 2 diabetes mellitus, **QoL:** quality of life

individuals and optimize both IBD and metabolic clinical outcomes.

Obesity and QoL: Patient-Centred Outcomes

Emerging evidence suggests that both obesity and related metabolic comorbidities negatively impact patient-reported outcomes. In a prospective cohort, patients with IBD and T2DM had significantly lower QoL scores based on the Short Inflammatory Bowel Disease Questionnaire (SIBDQ); (49.3 vs. 54.8; P <0.001), higher disease activity and increased healthcare use.[26] A large longitudinal cohort study from the IBD Partners cohort found that obesity was independently associated with inferior patient-reported outcomes across multiple domains, including anxiety, depression, fatigue, pain interference, and social function. These effects were evident in both UC and CD, with exposure-response relationships and longitudinal worsening observed in patients with class II/III obesity.[16]

In a prospective cohort of patients with CD, a higher visceral-to-subcutaneous fat ratio, but not BMI, was independently associated with lower SIBDQ scores over 24 months, particularly in patients with ileal disease.[5] In a cross-sectional

study of 688 patients with IBD, those with an active or healthy lifestyle (Mediterranean diet plus physical activity) had significantly higher IBDQ-9 scores.[28] Inactivity and poor dietary adherence were also independently associated with obesity, MASLD, MetS, and T2DM. Finally, Guardado et al. reported that surgical resection led to significant SIBDQ improvements across all BMI groups, with no pre- or postoperative QoL differences by BMI, suggesting obesity does not preclude postoperative QoL improvement.[29] Overall, these findings underscore the need for future studies that move beyond traditional clinical endpoints to understand better how obesity, visceral adiposity, and lifestyle factors affect QoL, which is an increasingly recognized and key target in the holistic management of IBD.

Clinical Practice Implications and Research Priorities

Obesity is an increasingly common coexisting condition in patients with IBD, influencing disease phenotype, treatment response, surgical risk, metabolic health, and QoL. These domains, summarized in **Table 1**, reflect a growing body of evidence highlighting the need for a more nuanced and proactive approach to care. For example, providers should move beyond BMI, incorporating image-based assessments of visceral adiposity to better stratify risk and guide management, though broader implementation in practice remains a future goal.

Therapeutic decisions should consider how obesity modifies drug response. Obesity may reduce the effectiveness of biologic therapies, while JAK inhibitors appear to maintain efficacy across weight categories. Routine screening for cardiovascular disease, diabetes, and MASLD should be incorporated into the standard of care for patients with IBD with excess adiposity. Multidisciplinary care, through collaboration with dietitians, psychologists, endocrinologists, and hepatologists, may help address the complex needs of this population and optimize both gastrointestinal and metabolic outcomes.

Despite progress, critical gaps remain. Future research should clarify the mechanistic links between adiposity and intestinal inflammation. Longitudinal studies are needed to assess the impact of obesity and its management on IBD-specific outcomes. Comparative effectiveness studies evaluating medical (e.g., glucagon-like peptide-1 receptor agonists), surgical, and lifestyle interventions across diverse IBD populations are needed. Finally, as weight management therapies become more widely used, consensus guidelines are urgently needed to support their safe and effective integration into IBD care.

Correspondence

Joëlle St-Pierre, MD, PhD
Email: jmlstpie@ucalgary.ca

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