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Beyond the Bowel

Addressing Obesity as a Modifiable Driver of IBD Outcomes



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All identified conflicts of interest have been mitigated.

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LEARNING OBJECTIVES

1. Identify the immunometabolic mechanisms by which visceral and mesenteric adipose tissue contribute to IBD disease activity, therapeutic attenuation, and surgical risk in people with IBD and obesity.

2. Apply appropriate, validated methods to systematically identify and characterize adiposity in people with IBD during routine gastroenterology encounters, including recognition of people at highest risk based on adiposity distribution.

3. Develop an individualized treatment plan for a patient with IBD and obesity that integrates weight management strategies and addresses the safety and appropriate use of GLP-1 RAs in this population.

Obesity and IBD: Prevalence and Clinical Impact

Among adults with IBD:

15% to 40%	25% to 40%
have obesity	**have overweight**

How Adiposity May Contribute

Visceral and mesenteric adipose tissue produce pro-inflammatory cytokines

↓

Promotes chronic low-grade inflammation

↓

Impaired intestinal barrier and gut dysbiosis in IBD allows for bacterial translocation

Population-based cohorts found that **46.7% of new CD diagnoses** and **55.4% of new UC diagnoses** are **among people with overweight or obesity**

Clinical Implications

Disease course	• Mixed evidence • Higher visceral fat linked to earlier flare, endoscopic recurrence, and stricturing CD
Medical therapy	• Obesity may reduce therapeutic effectiveness, particularly immunomodulators and anti-TNF agents
Surgery	• Obesity is associated with longer operative times, increased blood loss, higher rates of postoperative wound complications, and more difficult abdominal surgeries
Quality of life	• Obesity concomitantly reduces QoL in individuals with IBD

IBD = inflammatory bowel disease; CD = Crohn's disease; UC = ulcerative colitis; TNF = tumor necrosis factor; QoL = quality of life.

Sun J, et al. *Inflamm Bowel Dis.* 2025;31(9):2547-2560. Johnson AM, et al. *J Clin Gastroenterol*. 2024;58:176-182. Johnson AM, et al. *J Crohns Colitis.* 2021;15:1816-1823.

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Evidence Gaps in Obesity and IBD

1	**Mechanism and causality**	• Is obesity, or metabolic syndrome, a potential cause of IBD? • What inflammatory pathways do the two conditions share?
2	**Adiposity measurement and risk**	• To what extent does obesity alter disease course? • How do BMI, IA-VAT, and creeping fat best predict outcomes?
3	**Weight management and GLP-1 RA use**	• What is the long-term safety and efficacy of GLP-1 RAs in individuals with IBD? • Do GLP-1 RAs have anti-inflammatory effects in IBD?

BMI = body mass index; GLP-1 RA = glucagon-like peptide-1 receptor agonist; IA-VAT = intra-abdominal visceral adipose tissue.

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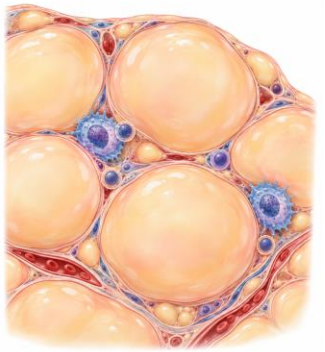
Adipose Tissue Can Shape IBD Outcomes

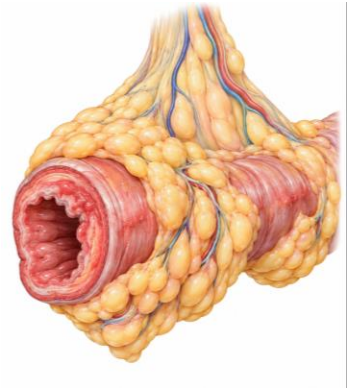
Adipose tissue releases immune and endocrine signals, including leptin and adiponectin.

Visceral obesity



Hypertrophied adipocytes with altered immune signaling/increased pro-inflammatory mediators

Crohn's creeping fat



Smaller hyperplastic adipocytes with pro- *and* anti-inflammatory mediators that wrap inflamed bowel

CONSTELLATION

Higher intra-abdominal visceral adipose tissue was independently associated with lower rates of corticosteroid-free deep remission at week 14/16* ($p < 0.001$), and lower rates of endoscopic remission at weeks 30-46 ($p = 0.02$)

*Week 14/16 = each drug's standard post-induction assessment (week 14 for infliximab/vedolizumab, week 16 for ustekinumab).

Yarur AJ, et al. *Gastroenterology.* 2023;165(4):963-975.e5. Kredel LI, et al. *Front Immunol.* 2014;5:462.

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GI Tolerability of GLP-1 RAs in IBD Cohorts

BMI Misses Fat Distribution

- Visceral adiposity can carry risk that BMI alone does not show
- MODIFY-IBD offers an IBD-specific consensus framework for obesity and cardiometabolic comorbidities

Where the GLP-1 RA Evidence Actually Stands

- Emerging IBD data are reassuring on safety, while a disease-modifying effect remains unproven
- Nausea, vomiting, diarrhea, and constipation may resemble IBD symptoms during treatment initiation or dose escalation

GI Tolerability in Separate IBD cohorts

13% Any adverse events

Sehgal et al, 2025 — n = 244, at 3 months
Nausea/vomiting 8%, diarrhea 2%

40% Any adverse events

Clarke et al, 2025 — n = 272, over up to 12 months
Nausea/vomiting 22%, diarrhea 6%, constipation 11%

12% Discontinued for intolerance

Clarke et al, 2025 — same n = 272 cohort
Most who had an adverse event continued therapy

Sehgal P, et al. *Clin Gastroenterol Hepatol.* 2025;23:1453-1454.e3. Clarke L, et al. *Dig Dis Sci.* 2025;70:2426-2432.

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Indications and Risk Stratification for GLP-1 RAs in IBD

A practical screen for benefits, precautions, and stop signals

PROCEED

When indicated for the agent

- Obesity/weight management
- Type 2 diabetes
- Established CVD*
- Noncirrhotic MASH*
- Obstructive sleep apnea*

PROCEED WITH CAUTION

Assess risk and monitor

- Gallbladder disease
- Reduced colonic motility
- Sarcopenia risk (up to 25% of GLP-1 RA weight loss is lean muscle mass)
- Acute kidney injury
- Mood changes

CONTRAINDICATED

Consider an alternative

- History of MTC (self/family) or MEN2
- Unintentional weight loss
- Active eating disorder
- Chronic nausea or gastroparesis (> 4 hrs)
- Pregnancy

STOP THERAPY

and assess

Acute pancreatitis

Non-arteritic anterior ischemic optic neuropathy

Anaphylaxis

*People with overweight/obesity may qualify if also having these comorbidities.
CVD = cardiovascular disease; MASH = metabolic dysfunction-associated steatohepatitis; MEN2 = multiple endocrine neoplasia type 2; MTC = medullary thyroid carcinoma.
Adapted from Beniwal-Patel P, et al. *Inflamm Bowel Dis*. 2026:izag151.

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Clinician Perspectives: Navigating Complexity in Obesity and IBD

HCP Reality in Obesity-IBD Management

- **Only ~24% always/often** see obesity as an active inflammatory contributor
- **51%** view it as **separately managed,** not an active driver
- **14.3%** report **no one consistently manages** obesity

Where the Barriers Actually Are

- Comfort isn't the issue: **65% comfortable discussing AOMs**
- **Barriers are diffuse:** time (25.5%), evidence (23.5%), tools (21.6%), interpretation (19.6%)
- **Ownership unclear:** only 18.4% of GIs see this as their own role

Clinical Challenges Clinicians Face

- **Distinguishing AOM GI effects from active IBD: ~33%** (top challenge)
- Persistent symptoms despite optimized therapy in obesity: ~29%
- Coordinating obesity management across specialties: ~25%

What This Means For Care

- **Top AOM concern is GI adverse effects (38.8%),** ahead of cost (24.5%)
- Beyond BMI tools underused: imaging ~14%, sarcopenic obesity assessment ~14%
- Multidisciplinary, **SDM-based models needed to bridge ownership gaps**

HCP CMEO survey responses: 49-51 clinicians, primarily gastroenterologists (72.5%) and gastroenterology NPs/PAs (17.6%), practicing in academic (35.3%), private/small-group (29.4%), and community (15.7%) settings.

Key takeaway – clinicians aren't uncomfortable talking about obesity or AOMs. They are unclear:

1) who owns the conversation
2) how to assess adiposity beyond BMI, and
3) how to interpret AOM-related symptoms against disease activity

AOM = anti-obesity medication; GI = gastrointestinal; SDM = shared decision-making.
CME Outfitters Internal Needs Assessment Survey. 2026. Not peer-reviewed.

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Patient Cases: What Would You Do?

Audience, Community/Rural Clinician, KOL, AI, and Faculty Responses to Three Patients with IBD



PATIENT CASE

Case 1 — Denise

- 38-year-old woman with left-sided UC, diagnosed 5 years ago
- On infliximab for the past 6 months, now with worsening symptoms
  - 5-6 stools per day with intermittent blood
  - Increased urgency
  - Fatigue increasingly interfering with her work as a teacher
- Attributes some fatigue to poor sleep but has never been evaluated for sleep apnea
- On contraception; not presently interested in pregnancy
- BMI has risen from 27 to 34 over the past two years
  - Has not previously connected her weight gain to IBD or her response to treatment
  - No prior clinical discussion of this change
- Has seen ads for weight loss medications and asks whether one might help
  - Unsure whether that is something a GI provider would prescribe
- Reports new bilateral knee stiffness over the past month, which she assumes is unrelated



PATIENT CASE

Case 1 — Denise

Last endoscopy: 6 months ago, prior to infliximab initiation
Flexible sigmoidoscopy with Mayo endoscopic subscore: 2 (continuous moderate inflammation, rectum through sigmoid)
Current therapy: Infliximab x 6 months + mesalamine enema

	Result	Reference Range
CRP	11 mg/L	< 10 mg/L: normal/low
Fecal calprotectin	540 µg/g (310 µg/g 3 months ago; ~800 µg/g at treatment initiation)	< 50 µg/g: normal (low inflammation)
Infliximab trough level	6.2 µg/mL	> 7.0 µg/mL
Anti-infliximab abs	None detected	—
Hemoglobin	12.1 g/dL	12.0–15.5 g/dL: normal range for women
Albumin	3.6 g/dL	3.5 g/dL to 5.0 g/dL: normal
BMI	34 (up from 27 two years ago)	18.5–24.9 kg/m²: normal weight; ≥ 30: obesity
Waist circumference	41 in	< 35 in: below increased cardiometabolic risk threshold for women (NIH/ATP III)

abs = antibodies; CRP = C-reactive protein; NIH/ATP III = National Institutes of Health/Adult Treatment Panel III.







AUDIENCE POLLING – Case 1, Denise

What would you do next?

A. Optimize infliximab (increase dose or shorten interval)

B. Switch to an IL-23p19 inhibitor

C. Switch to a JAK inhibitor

D. Pursue structured adiposity workup before adjusting the biologic

E. Optimize infliximab and evaluate for pharmacologic weight management concurrently

F. Refer for evaluation of pharmacologic weight management (e.g., tirzepatide, semaglutide, orforglipron), deferring biologic changes

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IL-23p19 = interleukin-23, p19 subunit; JAK = janus kinase.

Beniwal-Patel P, et al. *Inflamm Bowel Dis*. 2026:izag151.



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Audio Recording – Case 1, Denise









E Optimize infliximab and evaluate for pharmacologic weight management concurrently

- **Initial infliximab response was real** (calprotectin dropped after loading) – low trough points to a dosing issue, not mechanistic failure
- **Emerging data suggest GLP-1 RAs may have a direct anti-inflammatory effect in IBD,** not just weight-mediated benefit
- Optimizing infliximab and addressing weight concurrently treats both problems at once

Rule outs

- **Switch mechanism:** not supported; response pattern points to a fixable trough, not failure
- **Optimize infliximab alone:** leaves a relevant comorbidity unaddressed

RI = Rhode Island.



PRIVATE GASTROENTEROLOGIST, CO

Audio Recording – Case 1, Denise









E Optimize infliximab and evaluate for pharmacologic weight management concurrently

- **Infliximab level below goal:** no antibodies detected; room to increase the dose now
- Managing her weight pharmacologically (e.g., a GLP-1 RA) may improve her infliximab response
- **Flags a possible S1P modulator down the line,** depending on response to the increased dose

Rule outs

- **None stated directly**

CO = Colorado.



Additional Community Gastroenterologist Responses

Case 1, Denise









RESPONDENT 3	E	Optimize infliximab and evaluate for pharmacologic weight management concurrently
RESPONDENT 4	E	Optimize infliximab and evaluate for pharmacologic weight management concurrently

KOL: Millie D. Long, MD, MPH

Video Recording – Case 1, Denise

E **Optimize infliximab and evaluate for pharmacologic weight management concurrently**



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KOL: Amanda M. Johnson, MD

Video Recording – Case 1, Denise

E **Optimize infliximab and evaluate for pharmacologic weight management concurrently**





EXPERT FACULTY

Case Discussion – Case 1, Denise



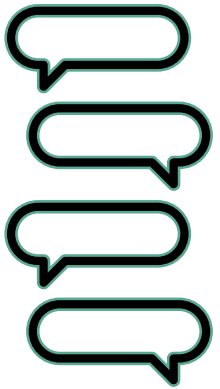






Decision Point

Patient Case: Denise



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AI: ChatGPT 5.6 Luna, OpenAI

User-Fed Response – Case 1, Denise

New chat → Here's the case's subjective, objective, and plan/multiple choice question. Based on the information provided, which answer choice would you choose and what is your brief rationale for your chosen answer and unchosen options?

E **Optimize infliximab and evaluate for pharmacologic weight management concurrently**

Rationale:

- Active UC despite subtherapeutic infliximab trough (6.2 vs. goal 7.5–10) with no antibodies → correctable pharmacokinetic issue, not mechanistic failure
- **14-point BMI increase*** warrants parallel obesity workup; neither should wait on the other

Rule outs

Optimize infliximab alone: treats drug exposure, ignores a treatable comorbidity
Switch to IL-23p19 inhibitor: premature; no proven mechanistic failure yet
Switch to JAK inhibitor: same issue; optimization should come first
Adiposity workup before biologic change: worthwhile, but shouldn't delay active inflammation
Weight management, defer biologic changes: reverses priorities; active UC cannot wait

*It is worth noting that ChatGPT mistakenly stated a 14-point increase in BMI when it was only a 7-point increase in BMI.











Case Perspective Summary

Case 1, Denise

	Community/Rural	KOLs	AI
Optimize infliximab (increase dose or shorten interval)	—	—	—
Switch to an IL-23p19 inhibitor	—	—	—
Switch to a JAK inhibitor	—	—	—
Pursue structured adiposity workup before adjusting the biologic	—	—	—
Optimize infliximab and evaluate for pharmacologic weight management concurrently	**4**	**2**	**1**
Refer for evaluation of pharmacologic weight management (e.g., tirzepatide, semaglutide, orforglipron), deferring biologic changes)	—	—	—

Pearls Learned from Denise's Case

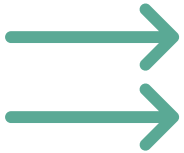
UC, infliximab optimization, visceral adiposity, mechanism selection, comorbidity screening



A subtherapeutic trough may reflect biology, not just dosing

Visceral adiposity can accelerate biologic clearance; a borderline trough with no antidrug antibodies argues for optimization, not an assumption of mechanistic failure

ACG guidance supports optimizing index therapy before switching mechanisms when trough levels fall below target



Obesity and active disease deserve parallel management

A 7-point BMI increase over two years is not incidental; structured obesity assessment should proceed alongside biologic optimization, not after it



Mechanism choice should account for treatment burden, not just efficacy

Injection frequency, monitoring requirements, and route of administration all shape whether a patient can sustain therapy, **factors that ACG guidance names explicitly alongside efficacy in mechanism selection**

ACG = American College of Gastroenterology.

Yarur AJ, et al. *Am J Gastroenterol*. 2023;118(11):2005-2013. Yarur AJ, et al. *Gastroenterology*. 2023;165(4):963-975.e5. Singh S, et al. *Gastroenterology*. 2024;167(7):1307-1343. Rubin DT, et al. *Am J Gastroenterol*. 2025;120(6):1187-1224.

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AUDIENCE POLLING REVISITED – Case 1, Denise

Now, what would you do next?

A. Optimize infliximab (increase dose or shorten interval)

B. Switch to an IL-23p19 inhibitor

C. Switch to a JAK inhibitor

D. Pursue structured adiposity workup before adjusting the biologic

E. Optimize infliximab and evaluate for pharmacologic weight management concurrently

F. Refer for evaluation of pharmacologic weight management (e.g., tirzepatide, semaglutide, orforglipron), deferring biologic changes



PATIENT PERSPECTIVE

Sarah Griffin — Patient Advocate



Looking back, with your own clinical background, did anyone discuss your weight or metabolic health before your surgery, and what do you wish had been part of that conversation?









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PATIENT CASE

Case 2 — Robert

- 54-year-old man with ileocecal Crohn's disease, diagnosed 8 years ago
- Has failed three advanced therapies:
  - Adalimimab, vedolizumab, and ustekinumab
- Now being evaluated for ileocecal resection
  - Montreal classification B2 (stricturing disease)
- Works in construction; physically active daily
- Weight has been stable for several years despite fluctuating GI symptoms
- At a visit last year, his PCP told him his BMI "looked fine" and that weight was not something he needed to worry about

PCP = primary care practitioner.



PATIENT CASE

Case 2 — Robert

Endoscopy and imaging:

- Colonoscopy: SES-CD score 14; severe ileal involvement, right-sided colonic involvement
- CT enterography: stricture in the distal terminal ileum with pre-stenotic dilation

Patient discontinued pharmacotherapy due to lack of symptom reduction

	Result	Reference Range
CRP	15 mg/L	< 10 mg/L: normal/low
Fecal calprotectin	780 µg/g	< 50 µg/g: normal (low inflammation)
Hemoglobin	12.8 g/dL	13.5–17.5 g/dL: normal range for men
Albumin	3.2 g/dL	3.5 g/dL to 5.0 g/dL: normal
BMI	27	18.5–24.9 kg/m²: normal weight; 25–29.9: overweight
Waist circumference	43 in	< 40 in: below increased cardiometabolic risk threshold for men (NIH/ATP III)
IA-VAT% (CT-derived)	3.8%	No generally accepted population reference range exists; his value (3.8%) exceeds the lower-quartile threshold (< 1.2%) seen in a comparable IBD cohort, and is disproportionate to what his BMI of 27 would predict
SES-CD (colonoscopy)	14	0–2: remission; 3–6: mild; 7–15: moderate; ≥ 16: severe

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CT = computed tomography; SES-CD = simple endoscopic score for Crohn's disease.

Yarur AJ, et al. *Am J Gastroenterol.* 2023;118(11):2005-2013.







AUDIENCE POLLING – Case 2, Robert

Which of these approaches most accurately characterizes this patient's adiposity-related surgical risk?

A. BMI of 27 confirms no elevated adiposity-related risk

B. Visceral adiposity findings alone indicate elevated surgical risk beyond what BMI shows

C. Refer to surgery given the stricture and significant inflammation; medications are most effective at preventing recurrence when used postoperatively

D. Low albumin points to malnutrition as the dominant risk driver; prioritize nutritional optimization first

E. Sarcopenic obesity phenotype; muscle loss and visceral fat should be addressed as a combined risk profile

F. Refer to bariatric surgery before proceeding

G. Repeat imaging in 6 months before deciding



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Audio Recording – Case 2, Robert









E Sarcopenic obesity phenotype; muscle loss and visceral fat should be addressed as a combined risk profile

- **Low albumin and waist circumference are concerning** even given his low BMI
- **Prestenotic dilation confirms obstructive disease;** surgery is indicated
- **Nutritional consultation should precede surgical evaluation;** post-op therapy likely needs optimization with an IL-23 agent

Rule outs

- **BMI confirms no risk:** implied, not stated directly; his concern held “even in light of his low BMI”

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Audio Recording – Case 2, Robert









C **Refer to surgery given the stricture and significant inflammation; medications are most effective at preventing recurrence when used postoperatively**

- **Active disease and a distal terminal ileum stricture, after failing multiple prior treatments** → refer to surgery
- Optimize with a different agent postoperatively, since his current regimen is not working
- His BMI is **unlikely to be contributing to the issue**

Rule outs

- **None stated directly**



Additional Community Gastroenterologist Responses

Case 2, Robert









RESPONDENT 3	E	Sarcopenic obesity phenotype; muscle loss and visceral fat should be addressed as a combined risk profile
RESPONDENT 4	B	Visceral adiposity findings alone indicate elevated surgical risk beyond what BMI shows

KOL: Millie D. Long, MD, MPH

Video Recording – Case 2, Robert

C **Refer to surgery given the stricture and significant inflammation; medications are most effective at preventing recurrence when used postoperatively**













KOL: Amanda M. Johnson, MD

Video Recording – Case 2, Robert

C/E

Refer to surgery given the stricture and significant inflammation; medications are most effective at preventing recurrence when used postoperatively/Sarcopenic obesity phenotype; muscle loss and visceral fat should be addressed as a combined risk profile



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EXPERT FACULTY

Case Discussion – Case 2, Robert



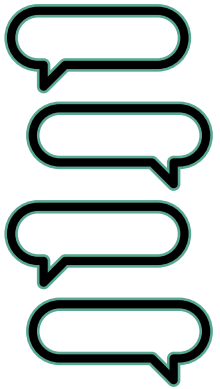






Decision Point

Patient Case: Robert



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AI: ChatGPT 5.6 Luna, OpenAI

User-Fed Response – Case 2, Robert

New chat → Here's the case's subjective, objective, and plan/multiple choice question. Based on the information provided, which answer choice would you choose and what is your brief rationale for your chosen answer and unchosen options?









E **Sarcopenic obesity phenotype; muscle loss and visceral fat should be addressed as a combined risk profile**

Rationale:

- BMI 27 masks a high-risk phenotype; CT shows markedly elevated visceral fat plus reduced muscle mass, alongside active severe CD and low albumin
- This combination predicts perioperative risk better than BMI alone

Rule outs

BMI confirms no risk: misses both the visceral fat and muscle findings

Visceral adiposity alone: incomplete; ignores the muscle depletion

Surgery now, meds post-op: doesn't address the adiposity risk question asked

Malnutrition as dominant driver: incomplete; misses the visceral fat component

Bariatric surgery first: wrong priority; CD surgery is the immediate need

Repeat imaging in 6 months: unnecessary delay given active disease and surgical indication











Case Perspective Summary

Case 2, Robert

	Community/Rural	KOLs	AI
BMI of 27 confirms no elevated adiposity-related risk	—	—	—
Visceral adiposity findings alone indicate elevated surgical risk beyond what BMI shows	1	—	—
Refer to surgery given the stricture and significant inflammation; medications are most effective at preventing recurrence when used postoperatively	1	2	—
Low albumin points to malnutrition as the dominant risk driver; prioritize nutritional optimization first	—	—	—
Sarcopenic obesity phenotype; muscle loss and visceral fat should be addressed as a combined risk profile	2	1	1
Refer to bariatric surgery before proceeding	—	—	—
Repeat imaging in 6 months before deciding	—	—	—

Pearls Learned from Robert's Case

CD, stricturing disease, sarcopenic obesity, preoperative risk, body composition



BMI can mask a high-risk body composition phenotype

A BMI of 27 looked unremarkable, but CT showed markedly elevated visceral adiposity alongside reduced skeletal muscle mass; a sarcopenic obesity phenotype invisible to BMI alone



Visceral fat burden predicts postoperative recurrence, independent of BMI

The **POCER study** found visceral adipose tissue above a defined threshold (VAT/height² > 51 cm²/m²) more than doubled the risk of endoscopic recurrence at 18 months, regardless of postoperative treatment



Sarcopenia independently predicts complications and recurrence

Nearly three-quarters of CD surgical patients had preoperative sarcopenia; it independently predicted postoperative complications; persistent sarcopenia after surgery was linked to lower 5-year recurrence-free survival

Holt DQ, et al. *Aliment Pharmacol Ther*. 2017;45(9):1255-1264. Yi GH, et al. *Medicine*. 2026;105(21):e48930. Lichtenstein GR, et al. *Am J Gastroenterol*. 2025;120(6):1225-1264.

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AUDIENCE POLLING – Case 2, Robert

Now, which of these approaches most accurately characterizes this patient's adiposity-related surgical risk?

A. BMI of 27 confirms no elevated adiposity-related risk

B. Visceral adiposity findings alone indicate elevated surgical risk beyond what BMI shows

C. Refer to surgery given the stricture and significant inflammation; medications are most effective at preventing recurrence when used postoperatively

D. Low albumin points to malnutrition as the dominant risk driver; prioritize nutritional optimization first

E. Sarcopenic obesity phenotype; muscle loss and visceral fat should be addressed as a combined risk profile

F. Refer to bariatric surgery before proceeding

G. Repeat imaging in 6 months before deciding



PATIENT CASE

Case 3 — Angela

- 45-year-old woman with UC, diagnosed 11 years ago
- On mirikizumab for ~1 year after inadequate response to an anti-TNF agent
  - Before treatment, urgency dominated her life; mapped bathrooms on her commute, missed work and family events
  - Within months, urgency resolved almost entirely; calls it the change that gave her her life back
- Surveillance endoscopy 4 months ago: Mayo endoscopic subscore 1, despite clinical quiescence
- Started semaglutide 3 months ago via telehealth for a BMI of 33 without telling her GI team
  - Insurance denied coverage; now paying out of pocket
  - Titrated 0.5 mg → 1 mg weekly four weeks ago
- Since that increase: stool frequency up from 1–2/day to 4–5/day, with new cramping and nausea (no blood)
  - Lost 18 lbs; reduced appetite
- Frightened: worried her IBD team will stop the GLP-1, and that her urgency is returning



PATIENT CASE

Case 3 — Angela

Partial Mayo score: 2 (0–1: remission; ≥ 2: active disease of some degree)
Mirikizumab maintenance dosing on schedule; no missed doses; no clinical signs of loss of response

PRO	Pre-Mirikizumab Baseline	Maintenance (4 mo ago)	Today
Urgency NRS (0–10)	8	1	2
Stool frequency (per day)	8–10	1–2	4–5
Shortest stool deferral time	< 1 min	> 15 min	~10 min
Rectal bleeding subscore	2	0	0

	Result	Reference Range
CRP	4 mg/L	< 10 mg/L: normal/low
Fecal calprotectin	110 µg/g (140 µg/g 4 months ago; flare baseline ~900 µg/g)	< 50 µg/g: normal (low inflammation)
Hemoglobin	13.1 g/dL	12.0 g/dL–15.5 g/dL: normal range for women
Albumin	4.0 g/dL	3.5–5 g/dL: normal
Stool studies	*C. difficile* toxin/PCR negative; culture negative for enteric pathogens	Negative expected
BMI	33 (3-months prior to visit)	18.5–24.9 kg/m²: normal weight; ≥ 30: obesity

NRS = Numerical Rating Scale; PCR = polymerase chain reaction; PRO = patient-reported outcomes.
Yarur AJ, et al. *Am J Gastroenterol.* 2023;118(11):2005-2013.







AUDIENCE POLLING – Case 3, Angela

What would you do next?

A. Discontinue mirikizumab, assume loss of response, switch mechanism

B. Instruct her to stop semaglutide and reassess in 4 weeks

C. Continue both, attribute symptoms to titration, and reassess at her routine visit

D. Add topical mesalamine enemas for management of urgency

E. Proceed immediately to flexible sigmoidoscopy

F. Continue mirikizumab, coordinate on GLP-1 titration pace through dose reduction, and continue monitoring both symptoms and calprotectin



PRIVATE GASTROENTEROLOGIST, RI

Audio Recording – Case 3, Angela









F — Continue mirikizumab, coordinate on GLP-1 titration pace through dose reduction, and continue monitoring both symptoms and calprotectin

- **Frames the decision around SDM** to give Angela ownership over her care, not override her
- **Notes anxiety is playing a role and should be listened to,** not dismissed
- **Stay on the GLP-1 RA, but reduce the dose** and slow the pace
- **Follow fecal calprotectin** on mirikizumab; consider sigmoidoscopy as well

Rule outs

- **None stated directly**



PRIVATE GASTROENTEROLOGIST, CO

Audio Recording – Case 3, Angela









F — Continue mirikizumab, coordinate on GLP-1 titration pace through dose reduction, and continue monitoring both symptoms and calprotectin

- Acknowledges it is hard to tell what's causing her symptoms
- **Keep mirikizumab dosing unchanged;**
  - coordinate the GLP-1 titration
  - back down to the GLP-1 dose that was working
- Watch fecal calprotectin at the lower dose; titrate up more slowly next time
- Consider flexible sigmoidoscopy in ~6 months to assess response

Rule outs

- **None stated directly**



Additional Community Gastroenterologist Responses

Case 3, Angela









RESPONDENT 3	F	Continue mirikizumab; adjust GLP-1 titration pace and monitor closely
RESPONDENT 4	F	Continue mirikizumab; adjust GLP-1 titration pace and monitor closely







KOL: Millie D. Long, MD, MPH

Video Recording – Case 3, Angela

F

Continue mirikizumab, coordinate on GLP-1 titration pace through dose reduction, and continue monitoring both symptoms and calprotectin



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KOL: Amanda M. Johnson, MD

Video Recording – Case 3, Angela

F Continue mirikizumab, coordinate on GLP-1 titration pace through dose reduction, and continue monitoring both symptoms and calprotectin



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EXPERT FACULTY

Case Discussion – Case 3, Angela



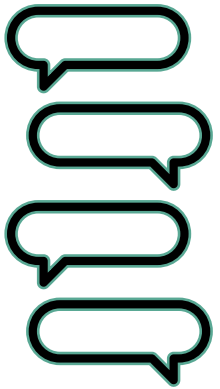






Decision Point

Patient Case: Angela



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AI: ChatGPT 5.6 Luna, OpenAI

User-Fed Response – Case 3, Angela

New chat → Here's the case's subjective, objective, and plan/multiple choice question. Based on the information provided, which answer choice would you choose and what is your brief rationale for your chosen answer and unchosen options?









F **Continue mirikizumab; adjust GLP-1 titration pace and monitor closely**

Rationale:

- Symptoms began right after a semaglutide dose increase; a typical GLP-1 GI effect, not a flare pattern
- CRP and calprotectin remain low; no bleeding, urgency still far improved from baseline
- Don't mistake AOM side effects for biologic failure; continue monitoring since she hasn't reached full endoscopic remission

Rule outs

BMI confirms no risk: misses both the visceral fat and muscle findings

Visceral adiposity alone: incomplete; ignores the muscle depletion

Surgery now, meds post-op: doesn't address the adiposity risk question asked

Malnutrition as dominant driver: incomplete; misses the visceral fat component

Bariatric surgery first: wrong priority; CD surgery is the immediate need

Repeat imaging in 6 months: unnecessary delay given active disease and surgical indication



Case Perspective Summary

Case 3, Angela









	Community/Rural	KOLs	AI
Discontinue mirikizumab, assume loss of response, switch mechanism	—	—	—
Instruct her to stop semaglutide and reassess in 4 weeks	—	—	—
Continue both, attribute symptoms to titration, and reassess at her routine visit	—	—	—
Add topical mesalamine enemas for management of urgency	—	—	—
Proceed immediately to flexible sigmoidoscopy	—	—	—
Continue mirikizumab, coordinate on GLP-1 titration pace through dose reduction, and continue monitoring both symptoms and calprotectin	4	2	1

Pearls Learned from Angela's Case

UC, GLP-1 RA titration, PROs, real-world safety, SDM



GLP-1 RA dose escalation can mimic a flare

A 244-patient IBD cohort found early adverse events (nausea, diarrhea) are common during titration; preserved urgency, deferral trends, and the absence of rectal bleeding argue against a true flare



Real-world data are more reassuring than assumed

A Mayo Clinic case-control study found comparable weight loss and safety with AOMs in IBD vs non-IBD patients; a Mass General Brigham cohort found no change in flare rates after starting a GLP-1 RA (Clarke L, et al.)



Self-initiated AOM may be undisclosed

Individuals may not think to mention a weight loss medication to their IBD team; normalize the question at every visit

Sehgal P, et al. *Clin Gastroenterol Hepatol.* 2025;23:1453-1454.e3. Pham JT, et al. *Am J Gastroenterol.* 2024;119(6):1197-1200. Clarke L, et al. *Dig Dis Sci.* 2025;70:2426-2432.

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AUDIENCE POLLING REVISITED – Case 3, Angela

Now, what would you do next?

A. Discontinue mirikizumab, assume loss of response, switch mechanism

B. Instruct her to stop semaglutide and reassess in 4 weeks

C. Continue both, attribute symptoms to titration, and reassess at her routine visit

D. Add topical mesalamine enemas for management of urgency

E. Proceed immediately to flexible sigmoidoscopy

F. Continue mirikizumab, coordinate on GLP-1 titration pace through dose reduction, and continue monitoring both symptoms and calprotectin











PATIENT PERSPECTIVE

Renika Wood, MS, LCAS, CCS — Patient Advocate



Describe your experiences with GLP-1 RAs during your personal journey with IBD and overweight.

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Closing the Gap

Turning Recognition Into Action in IBD and Obesity Care

Treat obesity and active disease in parallel, not in sequence

Visceral adiposity can blunt drug response; ensure medication optimization and address weight concurrently

BMI alone doesn't tell the whole story

Visceral adiposity and sarcopenia both predict surgical and postoperative outcomes that a normal-looking BMI can hide

Don't mistake AOM side effects for a flare

Distinguishing GI adverse effects of weight loss medications from active IBD can be difficult given overlapping symptoms; evaluate with objective markers (calprotectin, CRP, endoscopy) before assuming loss of response

The “right” plan starts with the right conversation

Normalizing questions about weight and AOM use as well as listening when patients raise it themselves is how individualized, team-based care happens

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Team-Based Approach for the Management of IBD and Obesity/Overweight

- No single specialty owns obesity management in IBD; it works best as a shared responsibility
- Gastroenterology, primary care, endocrinology/obesity medicine, and dietetics each bring something the others can't
- Only 18% of GI clinicians in the CMEO survey saw obesity management as their role; coordination should be built in deliberately, not assumed
- The patient is part of the team, not just the subject of it: preferences and goals should shape the plan



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Put information into action!

Takeaways from this program can be implemented into your practice to improve patient care.

- **Improve clinician confidence by ≥ 20%** in recognizing visceral and mesenteric adiposity as an active, modifiable driver of IBD treatment response and outcomes, not just an associated comorbidity
- **Expand appropriate adiposity assessment beyond BMI by ≥ 25%,** incorporating waist circumference or imaging-based body composition at treatment escalation and preoperative decision points
- **Strengthen individualized treatment planning by ≥ 25%,** incorporating SDM on weight management and GLP-1 RA safety at therapy selection, and using biomarkers and PROs rather than symptoms alone to distinguish adverse effects from disease flare

QUESTIONS & ANSWERS

Additional Resources

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Beyond the Bowel

Addressing Obesity as a Modifiable Driver of IBD Outcomes



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