Canadian IBD Today

2026 Clinical Updates in Gastroenterology Symposium

Event Summary

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In This Report

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Acronyms

aCRN	ADVANCED COLORECTAL NEOPLASIA
ACG	AMERICAN COLLEGE OF GASTROENTEROLOGY
AGA	AMERICAN GASTROENTEROLOGICAL ASSOCIATION
anti-TNF	ANTI–TUMOUR NECROSIS FACTOR THERAPY
BMI	BODY MASS INDEX
CRC	COLORECTAL CANCER
CRP	C-REACTIVE PROTEIN
CRSwNP	CHRONIC RHINOSINUSITIS WITH NASAL POLYPS
CTLA-4	CYTOTOXIC T-LYMPHOCYTE–ASSOCIATED PROTEIN 4
CVID	COMMON VARIABLE IMMUNODEFICIENCY
DDW	DIGESTIVE DISEASE WEEK
EoE	EOSINOPHILIC ESOPHAGITIS
ELF	ENHANCED LIVER FIBROSIS
EREFS	EOSINOPHILIC ESOPHAGITIS ENDOSCOPIC REFERENCE SCORE
FDA	U.S. FOOD AND DRUG ADMINISTRATION
FIB-4	FIBROSIS-4 INDEX
FMT	FECAL MICROBIOTA TRANSPLANTATION
FODMAP	FERMENTABLE OLIGOSACCHARIDES, DISACCHARIDES, MONOSACCHARIDES AND POLYOLS
GIP	GLUCOSE-DEPENDENT INSULINOTROPIC POLYPEPTIDE
GLP-1	GLUCAGON-LIKE PEPTIDE-1
HLA	HUMAN LEUKOCYTE ANTIGEN
IBD	INFLAMMATORY BOWEL DISEASE
IBS	IRRITABLE BOWEL SYNDROME
ICI	IMMUNE CHECKPOINT INHIBITOR
ICU	INTENSIVE CARE UNIT
IgA	IMMUNOGLOBULIN A
IgE	IMMUNOGLOBULIN E
IL-23	INTERLEUKIN-23

Acronyms Continued

LSM	LIVER STIFFNESS MEASUREMENT
IMC	IMMUNE-MEDIATED COLITIS
IV	INTRAVENOUS
JAK	JANUS KINASE
MASH	METABOLIC DYSFUNCTION-ASSOCIATED STEATOHEPATITIS
MASLD	METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE
NAFLD	NON-ALCOHOLIC FATTY LIVER DISEASE
NICE	NATIONAL INSTITUTE FOR HEALTH AND CARE EXCELLENCE
NSAIDS	NON-STEROIDAL ANTI-INFLAMMATORY DRUGS
PPI	PROTON PUMP INHIBITOR
SES-CD	SIMPLE ENDOSCOPIC SCORE FOR CROHN'S DISEASE
SNRI	SEROTONIN-NOREPINEPHRINE REUPTAKE INHIBITOR
SSRI	SELECTIVE SEROTONIN REUPTAKE INHIBITOR
STRIDE-II	SELECTING THERAPEUTIC TARGETS IN INFLAMMATORY BOWEL DISEASE II
TL1A	TUMOUR NECROSIS FACTOR-LIKE LIGAND 1A
TNF	TUMOUR NECROSIS FACTOR
tTG-IgA	TISSUE TRANSGLUTAMINASE IMMUNOGLOBULIN A ANTIBODY
UC	ULCERATIVE COLITIS
UC-CaRE	ULCERATIVE COLITIS-CANCER RISK ESTIMATOR
ULN	UPPER LIMIT OF NORMAL

Update on the Treatment of Crohn's Disease in 2026

VIPUL JAIRATH, MBChB, DPhil, FRCP, FRCPC

Dr. Jairath reviewed the evolving treatment landscape in IBD, highlighting that Health Canada has now authorized several IL-23 agents for both ulcerative colitis and Crohn's disease, including risankizumab, guselkumab, and mirikizumab. In trials, advanced therapies can achieve clinical remission in approximately 50% of patients with IBD, a result limited by the reality that many patients enrolled in the trials failed multiple therapies before initiating a biologic and trial populations are heterogeneous, including patients with ileal and colonic strictures. Early-phase data in ulcerative colitis for the oral IL-23 inhibitor icotrokinra suggest that oral therapy may achieve efficacy comparable to injectable advanced therapies.

Dr. Jairath then presented emerging TL1A therapies, including afimkibart, tulisokibart, and duvakitug, all of which are being investigated for effects on both inflammation and fibrosis. The ARTEMIS-UC trial phase 2 trial of tulisokibart suggests higher clinical remission rates among biomarker-positive patients than non-biomarker-positive patients (37.5% versus 21.2%), raising the possibility of matching selected patients to specific therapies through a buccal swab or similar test. The RELIEVE-UCCD trial of duvakitug demonstrated endoscopic response rates at week 14 of 48%, compared with 13% in the placebo arm, which Dr. Jairath noted is a promising result.

While some patients are primary nonresponders, other patients lose response to therapy after years of stability. New data suggest that combination therapy may have a key role after primary nonresponse or secondary loss of response. The VEGA trial assessed the combination of guselkumab and golimumab in ulcerative colitis, and demonstrated stronger endoscopic remission outcomes with the combination than with either medication alone, without major new safety signals. The DUET-CD trial enrolled patients with a mean SES-CD score of 13.7; 50% were refractory or intolerant to two or more systemic therapies. In the overall population, combination therapy didn't improve outcomes. However, in patients with Crohn's disease who had failed two or more systemic medications, the combination of guselkumab and golimumab led to clinical remission rates of 49.2%, compared to 27.3% with IL-23 inhibition alone. Endoscopic response was also improved with the combination, especially in patients who had failed two or more previous systemic therapies. Dr. Jairath suggested that in the future, combination therapy is likely to be recommended for patients who fail two therapies. In an exciting development, Spyre Therapeutics is testing a combination therapy with a prolonged half-life that could allow for two or three times yearly dosing.

Perianal Crohn's disease was another major focus. Dr. Jairath described it as one of the most challenging and burdensome presentations in clinic, often associated with poor quality of life and limited therapeutic options. Dr. Jairath highlighted the phase 3 FUZION trial showing that guselkumab improved combined clinical and radiologic fistula outcomes in a refractory population, including many patients who had already failed anti-TNF therapy. Combined fistula remission was approximately 28% in the guselkumab arms, versus approximately 10% in the placebo arm at week 24. He noted this is the first positive trial for perianal Crohn's disease since infliximab maintenance therapy was shown to be effective in 2004.

Finally, Dr. Jairath discussed treatment targets in Crohn's disease. Intestinal ultrasound is gaining traction in Canada, but it remains unclear whether treating bowel wall thickness as a formal target improves long-term outcomes. He showed early data from the VECTORS trial, which suggest that targeting robust remission, including ultrasound response, leads to better outcomes than targeting clinical and biomarker remission only. He concluded that IBD therapeutic development is very active, but delayed diagnosis and delayed initiation of effective advanced treatment remain major barriers to optimizing outcomes.

VIPUL JAIRATH, MBChB, DPhil, FRCP, FRCPC


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Update on the Treatment of Ulcerative Colitis in 2026

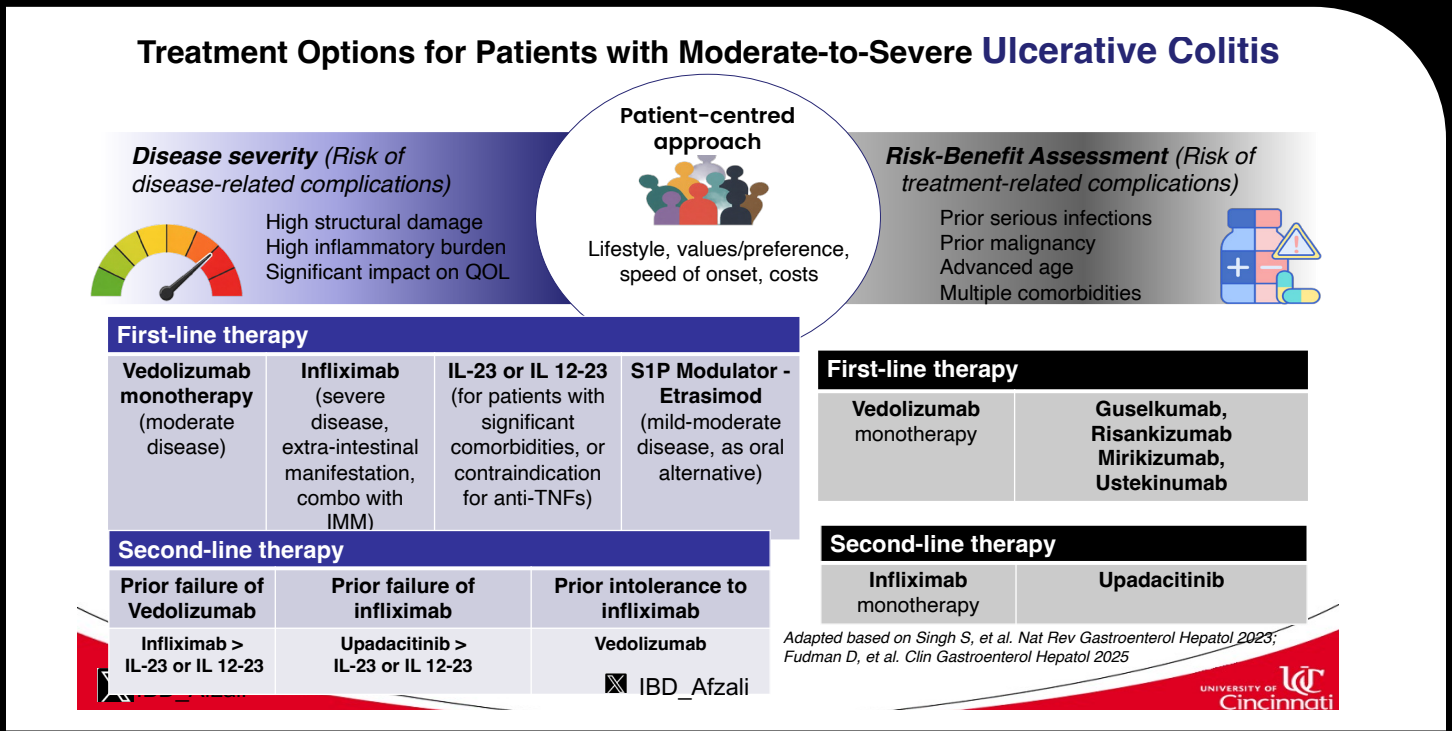
ANITA AFZALI, MD, MPH, MHCM, FACG, AGAF

Dr. Afzali began by outlining prognostic factors in UC that indicate a higher risk of disease progression, including younger age at onset, extensive or pancolonic disease, and a high inflammatory burden reflected in laboratory markers. She emphasized that, as in Crohn's disease, UC has a "window of opportunity" during which appropriate treatment may prevent progression and complications. Waiting too long to initiate or escalate therapy increases the risk of long-term consequences such as dysmotility, fibrosis/scarring, strictures, CRC, and colectomy.

Presenting the results of an international survey, Dr. Afzali described the gap between clinician and patient definitions of remission. Clinicians increasingly focus on objective targets such as fecal calprotectin levels and endoscopy results, while patients often define remission as symptom resolution. She also highlighted a mismatch in expectations around treatment durability, with clinicians often expecting a year of benefit from a therapy while 44% of patients expect therapies to remain effective for 3 years or longer. Dr. Afzali presented survey data showing that clinicians often prioritize efficacy when choosing therapies. However, U.S. claims data suggest that in UC, less than 10% of patients receive biologics or oral small molecules as first-line therapy. She lamented the continued overreliance on mesalamine and repeated courses of corticosteroids, arguing that educational efforts are needed to change practice.

She then described how treatment endpoints have evolved. Historically, symptom control was often considered sufficient, but newer approaches require more stringent targets. A longitudinal cohort trial published in 2016 demonstrated a more than six-fold reduction in risk of relapse after 1 year among patients with a Mayo endoscopic score of 0, compared to 1.

Dr. Afzali reviewed the STRIDE-II treatment-target framework, describing short-term, intermediate, and long-term goals. At approximately 3 months, clinicians should aim for clinical remission, C-reactive protein <ULN, and, especially in UC, fecal calprotectin levels of 100–250 µg/g. At 6 to 12 months, targets should include endoscopic



ANITA AFZALI, MD, MPH, MHCM, FACG, AGAF

healing, with histology likely to be included in future STRIDE recommendations. She emphasized proactive monitoring of the disease, not merely drug monitoring, and drew attention to the reality that the adoption of treat-to-target recommendations remains low in the U.S. She encouraged clinicians to use the updated ACG Ulcerative Colitis Disease Activity Index when assessing therapeutic response. The ACG recommends targeting endoscopic improvement (Mayo 0 whenever possible), steroid-free remission, and preventing hospitalization and surgery. The guidelines support all preferred advanced therapies for ulcerative colitis induction therapy, and recommend avoiding thiopurine or methotrexate monotherapy for induction. Steroids and methotrexate should be avoided in maintenance but thiopurines can be used as maintenance after steroid-induced remission. The ACG recommends vedolizumab more strongly than adalimumab in ulcerative colitis and also recommends reactive drug level monitoring for anti-TNF loss of response.

While the VARSITY trial demonstrated vedolizumab superiority over adalimumab in biologic-naive moderate-to-severe ulcerative colitis, real-world data suggest vedolizumab and infliximab may perform similarly as first-line biologics in IBD (Bressler et al, 2021). After anti-TNF failure, however, tofacitinib appeared more effective than vedolizumab in observational data (Buisson et al, 2023). Based on the VISIBLE and LIBERTY-UC study of vedolizumab and infliximab, Dr. Afzali recommended subcutaneous therapies based on patient preference and appropriateness.

She concluded by encouraging clinicians to assess both disease factors, such as inflammatory burden and progression risk, and patient factors, including comorbidities, extraintestinal manifestations, infection risk, and malignancy risk. Her central message was that ulcerative colitis treatment in 2026 should be individualized, target-driven, and timely.

Update on the Treatment of Celiac Disease

JOE MURRAY, MD

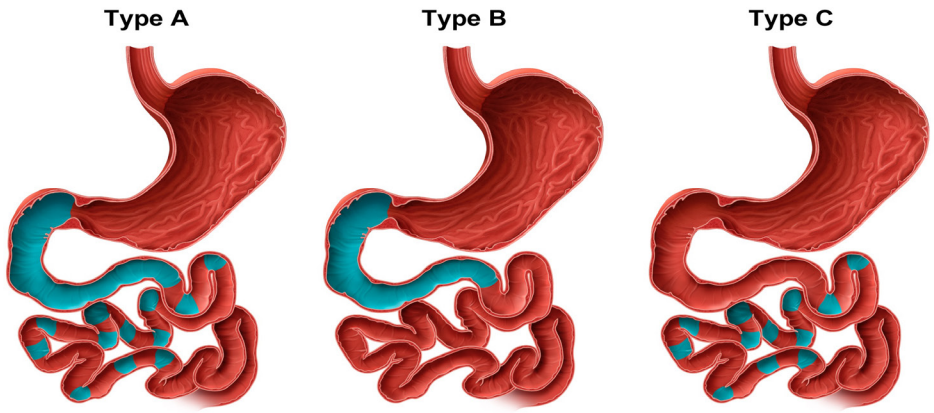
Celiac disease is a genetically determined, inflammatory disorder triggered by gluten. Although about one-third of the population carries a genetic risk for the disease, only a small minority (1%) develop it. Dr. Murray explained that celiac disease may present in a myriad of different ways, including with iron-deficiency anemia, bloating, abdominal pain, diarrhea, fatigue, premature osteoporosis, dermatitis herpetiformis, and even neuropsychiatric manifestations such as depression or recurrent anxiety. Dr. Murray emphasized that the presentation has changed in recent decades, with slightly below 50% of patients presenting with "classic" symptoms of diarrhea, abdominal pain, and malabsorption symptoms and slightly above 50% of patients presenting with less specific, more mild disease.

Celiac disease has become more common in recent decades, especially in northern latitudes, with the global incidence rising before plateauing around 2010. While traditionally considered a proximal small bowel disease, patients may have "ultra-short" celiac disease limited to the duodenal bulb. Dr. Murray highlighted recent recommendations to take biopsies from both the duodenal bulb and the second and third portion of the duodenum when endoscopy is used for diagnosis.

Discussing diagnosis, Dr. Murray provided an overview of the genetic component of celiac disease and added that too often, specialists fail to ask patients about a family history of celiac disease. Diagnosis includes villous atrophy with chronic inflammation in the proximal small intestine while the patient is eating a diet containing gluten and clinical response to a gluten-free diet. Dr. Murray stressed that biopsy can be avoided in selected patients with symptoms suggestive of celiac disease, tTg-IGA > 10 times ULN, a confirmatory blood sample that is positive for endomysial antibodies, and clinical response to a gluten-free diet. He added that he continues to routinely perform endoscopy for patients older than 40 years of age, due to the risk of malignancy or other endoscopic abnormalities.

For patients already on a gluten-free diet, serology and biopsies may normalize, but HLA typing can help rule out disease if HLA-DQ2/DQ8 are absent. A gluten challenge may be needed

Capsule Endoscopy Types of Involvement in Symptomatic Patients with Celiac Disease



Type of involvement	Extensive enteropathy	Limited duodenal enteropathy	Jejunal enteropathy
Frequency	22 (59%)	12 (32%)	1 (3%)
Description of lesion	Duodenal continuous and jejunal patchy	Duodenal continuous	Jejunal patchy

JOE MURRAY, MD

to confirm a diagnosis, but it is burdensome and can make patients ill, so Dr. Murray recommended against a gluten challenge for patients who are satisfied and whose symptoms have resolved on a gluten-free diet. If a gluten challenge is pursued, the diet should include at least one slice of gluten-containing bread per day for 6 weeks. If the serology is negative, Dr. Murray recommended continuing the challenge to 12 weeks and even beyond to account for delayed responders.

Dr. Murray then discussed antibody-negative villous atrophy and rare mimics. Differential diagnoses include drug-induced enteropathy, tropical sprue, Helicobacter pylori infection, CVID, peptic duodenitis, Crohn's disease, and infections, among others. He stressed that HLA testing is particularly useful when serology is negative, and added that explanations for negative serology include low/no gluten intake prior to testing, IgA deficiency, and borderline results. Young people are more likely to have lower tTg-IGA levels than older patients. If seronegative celiac disease is suspected, Dr. Murray emphasized the importance of following patients for a durable response to a gluten-free diet, following up biopsies for healing, and keeping an open mind about alternatives.

Turning to management, Dr. Murray recommended initially assessing adults for nutritional deficiencies, bone density, and pneumococcal and Shingles vaccine status. For patients older than 50, Dr. Murray recommended considering enterography given the risk of concomitant malignancy. The gluten-free diet can normalize antibodies, promote mucosal recovery, improve quality of life, and reduce complications, but adherence is challenging, with only approximately 75% of patients able to adhere to a strict gluten-free diet. Follow-up should include symptom review at 1 to 3 months, serology at 6 months, dietitian support, and optional repeat biopsy after 1 to 2 years.

Update on Treatment of Checkpoint Inhibitor Colitis in 2026

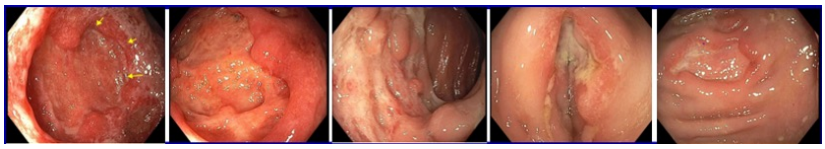
YINGHONG (MIMI) WANG, MD, PhD, MSc

Over the past decade, multiple major medical organizations, including MD Anderson, have released practice guidelines regarding the management of immunotherapy-related organ toxicities. Because patients who develop gastrointestinal toxicity have better overall survival than patients taking immunotherapy who don't develop gastrointestinal toxicity, Dr. Wang explained the goal is to control the colitis effectively so patients can resume or continue life-prolonging cancer therapy when appropriate.

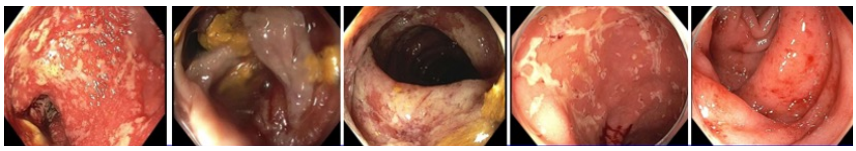
Dr. Wang then reviewed clinical presentation. Checkpoint inhibitor colitis commonly presents with diarrhea, fecal incontinence and urgency, as well as colitis symptoms including blood and mucus in the stool, fever, and abdominal pain. Onset of symptoms is most common within 2 to 3 months of starting therapy, but it can occur anytime from the first dose to up to 1 year after the last dose. Evaluation includes stool infectious work up, inflammatory stool markers, and colonoscopy or sigmoidoscopy with biopsy. She emphasized that CRP is less useful in this population because it can be affected by underlying cancer. Stool lactoferrin can help screen for inflammation, with a 70% sensitivity for endoscopic inflammation and a 90% sensitivity for histological inflammation. Calprotectin has a 91.4% and 86.4% sensitivity, respectively (Wang et al, 2018; Wang et al, 2025).

Endoscopic presentations range from normal-appearing mucosa to severe inflammation with deep ulcerations. Severe endoscopic features are associated with steroid-refractory disease, need for biologic therapy in addition to corticosteroids, and hospitalization. Early endoscopy, and the earlier induction of biologic therapies, can improve outcomes, leading to less IV steroid, shorter duration on steroid, shorter symptom duration, less ICU admission and lower colitis recurrence. When endoscopy is not feasible to assess the effect of therapy, fecal calprotectin can serve as surrogate marker to predict colitis remission and guide the treatment duration with a study by Wang et al published in 2021 demonstrating a specificity for histologic remission of 85% and for endoscopic remission of 94%.

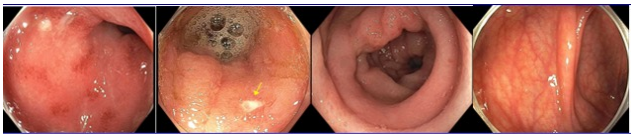
Endoscopy Features



Severe inflammation with large deep ulcers



Moderate inflammation with erythema, exudate, superficial ulcers



Mild inflammation with patchy erythema, aphtha, edema, or normal mucosa

IMC has wide spectrum of endoscopic presentations from mild to severe level.

Wang et al Inflammatory Bowel Disease 2018;24(8):1695-1705.

YINGHONG (MIMI) WANG, MD, PhD, MSc

Treatment depends on severity. Grade 1 disease may be managed with supportive care, including loperamide, fibre, and diphenoxylate/atropine. If this is unsuccessful, immunosuppressants including mesalamine or cholestyramine can be effective. For grade 2 or higher disease, immunosuppressants remain first-line, followed by biologics when needed. Investigational therapies included fecal transplant and tocilizumab. Dr. Wang argued that biologics should be introduced before repeated steroid failures, with a real-world study (Wang et al, 2019) showing earlier introduction of a biologic results in shorter symptom duration, fewer steroid tapers and attempts, and fewer hospitalizations. Infliximab and vedolizumab have similar efficacy, with response rates of 85% to 90%; infliximab may work faster while vedolizumab may be associated with shorter steroid exposure and lower recurrence (Zou, Wang, et al 2021). For refractory cases, ustekinumab and tofacitinib show promise.

Recurrence occurs in roughly one-third of patients overall, with higher risk after CTLA-4 therapy, severe index colitis, and patients requiring immunosuppressant therapy. Concurrent biologic therapy during rechallenge may reduce recurrence (Badran, et al, 2023), however long-term safety data on this approach is lacking.

Dr. Wang cautioned against unnecessary antibiotics on patients with immune checkpoint inhibitors. Infection should be treated when confirmed, but prophylactic antibiotics are not recommended as antibiotics, especially anaerobic, can worsen colitis severity and overall survival.

Investigational FMT shows great promise for checkpoint inhibitor colitis. MD Anderson performed the first FMT for refractory checkpoint inhibitor colitis in 2017, with rapid symptom resolution and endoscopic improvement. Ongoing trials evaluating FMT in both refractory and frontline settings. MD Anderson has demonstrated response rates of FMT around 80% to 90% within 30 days and generally self-limited adverse events. Adverse events typically resolve with conservative management in 24 hours. Importantly, FMT has sustainable effect over 12 months, enabling successful ICI rechallenge, though patients may need a second FMT after approximately 1 year.

Update on Treatment of IBS in 2026

COLLEEN PARKER, MD, MSc, FRCPC

Dr. Parker shared data outlining the significant major personal and health-system costs of IBS, noting that new data presented at DDW 2026 estimated the global prevalence of IBS to be approximately 10%. The new Rome V Criteria for IBS stipulates that patients must have recurrent, intermittent abdominal pain or discomfort at least 3 days per month in the past 3 months with symptom onset of at least 6 months prior to diagnosis. Pain or discomfort must be associated with at least two of the following: relation to defecation, change in stool frequency, or change in stool form. Dr. Parker reviewed IBS subtypes including IBS-C (IBS with constipation), IBS-D (IBS with diarrhea), IBS-M (IBS mixed), and IBS-U (IBS undefined). She emphasized that confidently diagnosing IBS can reassure patients and help them adhere to management approaches.

Dr. Parker focused on diet and pharmacotherapy, while explaining that behavioural therapy, education, and lifestyle management are also important components of IBS care. The low-FODMAP diet has the strongest evidence, and has shown to improve bloating, abdominal pain, and stool form in IBS-D. However, a low-FODMAP diet can worsen constipation and should be used in IBS-C only when bloating persists after bowel habits are controlled. The low FODMAP diet is not a long-term elimination diet: patients should restrict FODMAP foods for 6 weeks, then reintroduce foods methodically, ideally with the guidance of a dietitian. Prolonged restriction can cause weight loss, nutritional deficiencies, microbiome changes, and disordered eating. Other options include the NICE diet and the Mediterranean diet. Gluten-free diets are not routinely recommended as a first-line approach, though some patients without celiac disease report benefit.

For IBS-D, eluxadoline can improve stool consistency and urgency, although pain benefits are modest. It is contraindicated after cholecystectomy or in patients at risk of pancreatitis. Rifaximin may improve stool consistency, pain, and bloating after a 14-day course, but it remains unclear how often retreatment is necessary. As a clinical pearl, Dr. Parker said she rarely uses rifaximin for a chronic condition, but finds it beneficial for post-infectious IBS patients. Ondansetron can help stool form, frequency, urgency, and nausea in patients with IBS-D, with a modest effect on pain. Guidelines recommend against loperamide on a regular basis, because, although it can improve stool frequency and consistency, it can worsen pain and cause constipation when used regularly. Dr. Parker explained she prescribes loperamide on an as-needed basis, such as for a long flight.

Focusing on treating pain in IBS, Dr. Parker explained that peppermint oil can reduce overall symptom severity, including pain, but may worsen reflux. Enteric-coated formulations can reduce reflux and taking peppermint oil 30 minutes before a meal can reduce post-prandial symptoms. Antispasmodics can help as an as-needed or adjunctive therapy, especially in IBS-D, though anticholinergic side effects pose challenges. Dr. Parker explained that tricyclic antidepressants including amitriptyline and desipramine are effective for abdominal pain, but can cause constipation and drowsiness. SNRIs such as duloxetine can also improve abdominal pain and may help with overlapping chronic pain conditions. While the Canadian IBS guidelines indicate SSRIs for IBS, there is no evidence that SSRIs improve abdominal pain. Instead, SSRIs can be helpful when underlying anxiety or depression are suspected to be driving gut symptoms. Dr. Parker cautioned against combining SNRI and SSRI medications, but noted that tricyclic antidepressants can be safely combined with an SNRI or SSRI.

Dr. Parker closed by highlighting that adjunctive cognitive behavioural therapy and hypnotherapy can change pain-processing pathways and studies have demonstrated response rates ranging from 50% to 80%. Emerging options include digital apps, such as Nerva, microbiome therapies, mast-cell stabilizers, H1-receptor antagonists, new visceral pain modulators, and vagal nerve stimulation.

COLLEEN PARKER, MD, MSc, FRCPC

2026 Clinical Updates in Gastroenterology Symposium

Update on the Use of Incretins in 2026

WAQQAS AFIF, MD, MSc, FRCPC
JULIE LOVSHIN, MD, PhD, FRCPC

Dr. Lovshin reviewed the science, clinical evolution, and practical use of incretin-based therapies in 2026. She began by framing obesity as a chronic, relapsing medical condition that commonly overlaps with type 2 diabetes, cardiovascular disease, chronic kidney disease, and MASLD.

The discovery that oral glucose triggers greater insulin secretion than intravenous glucose led to an appreciation of the two most powerful incretins in humans, GLP-1 and GIP. GLP-1 is released from the gut after eating, stimulates glucose-dependent insulin secretion, and suppresses glucagon to increase glucose uptake. Dr. Lovshin reviewed the clinical development of GLP-1 receptor agonists, from liraglutide, approved in 2010, to once-weekly agents such as dulaglutide and the more powerful semaglutide. Semaglutide 2.4 mg produced weight loss of 12% to 14% (Davies et al, 2021), a major advance over earlier pharmacotherapies.

Tirzepatide, which activates both GIP and GLP-1 receptors, achieved a 21% body weight reduction at the 15 mg dose at 72 weeks in the SURMOUNT 1 trial. Dr. Lovshin noted that semaglutide and tirzepatide are now leading pharmacologic options, although tirzepatide is difficult to access while semaglutide is covered in Ontario for patients over 65.

Dr. Lovshin emphasized the importance of explaining to patients how the medications work by reducing appetite, to combat misinformation that patients can lose weight on incretin therapies without changing their diet. She recommended titrating the dose based on patients' self-reported appetite suppression and using the lowest effective dose for maintenance once patients are at a desired weight. Side effects, especially nausea and constipation, are common during titration, so clinicians should gradually increase the dose with patients who experience these side effects to ensure patient adherence. Dr. Lovshin advised against stopping the therapies as weight regain is common. Instead, switching to oral therapies for the long term may be preferred by patients. Patients taking incretin-based therapy should also be advised about protein intake, and resistance training to mitigate lean muscle loss.

Dr. Afif presented a short case discussion of a patient with mild ulcerative colitis, obesity, and MASH. The patient has a BMI of 38 kg/m^2, has been receiving vedolizumab every 4 weeks for 2 years, and continued to have mildly active ulcerative colitis. He also has elevated liver enzymes and evidence of fibrosis. Dr. Afif noted that obesity is common in IBD, affecting roughly 20% to 40% of patients, and may influence disease course and treatment response.

One possible explanation is altered drug pharmacokinetics, particularly with anti-TNF therapies, where patients with obesity may have higher drug clearance and require higher drug levels. Dr. Afif then addressed whether treating obesity could improve IBD outcomes. Early data suggest GLP-1-based therapies appear safe in IBD and do not increase flare risk, although gastrointestinal side effects may occur. Emerging evidence also suggests possible anti-inflammatory benefits independent of weight loss, with the ongoing COMMIT studies evaluating tirzepatide in ulcerative colitis and Crohn's disease.

WAQQAS AFIF, MD, MSc, FRCPC
JULIE LOVSHIN, MD, PhD, FRCPC


Julie
Lovshin
Waqqas
Afif

Update on the Treatment of MASLD

GIADA SEBASTIANI, MD

Dr. Sebastiani began by explaining why the terminology changed from NAFLD to MASLD in 2023. The older term was a diagnosis of exclusion whereas MASLD gives patients and clinicians a positive diagnosis based on hepatic steatosis and one or more cardiometabolic risk factor.

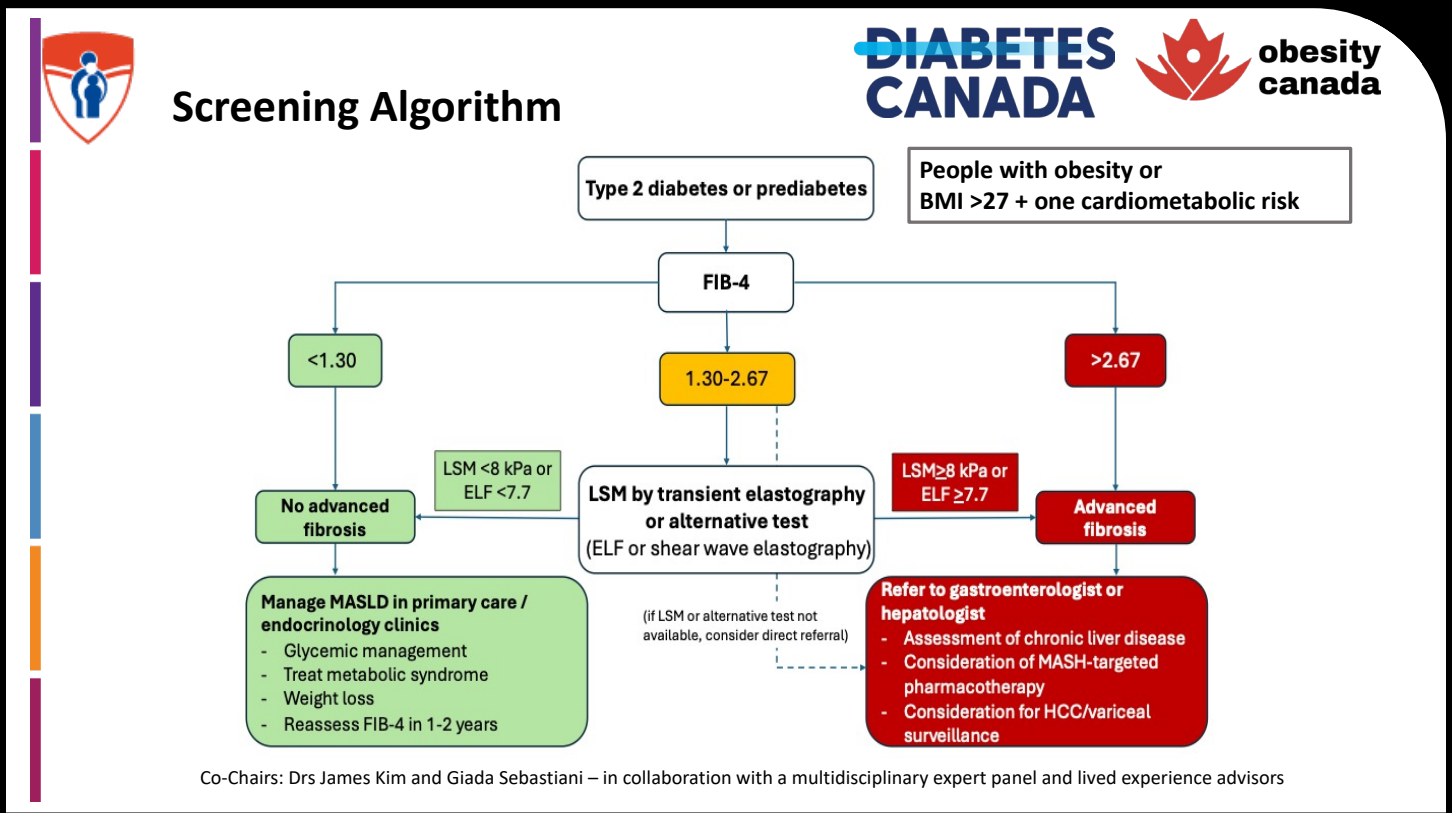
MASLD now affects roughly one in three adults worldwide and is the most common chronic liver disease globally. Projections suggest at least 14 million Canadians may be living with MASLD by 2030, alongside a doubling of cirrhosis, liver cancer, and liver transplantation related to MASLD.

Dr. Sebastiani identified obesity and type 2 diabetes as the two main risk factors for MASLD. Approximately 70% of Canadian adults are overweight or obese. Among people living with obesity, MASLD prevalence may reach 80%, and significant liver fibrosis affects 20% of patients with obesity. Type 2 diabetes is the strongest risk factor for MASLD. About 65% of people with type 2 diabetes have MASLD, and 15% of patients with type 2 diabetes may already have advanced fibrosis.

IBD is another emerging high-risk population. A Canadian study found patients with IBD have a 25% higher MASLD prevalence than the general population (Palumbo et al, 2019), with MASLD occurring at younger ages among patients who have IBD. Age, BMI, and triglyceride levels were associated with MASLD and fibrosis in the IBD cohort.

Because the disease is frequently asymptomatic, patients may present only once cirrhosis or decompensation has developed. Dr. Sebastiani argued that targeted screening is therefore essential. Clinicians should screen high-risk groups for fibrosis, including all patients with type 2 diabetes. The first-line tool is FIB-4. A score below 1.3 has a high negative predictive value, while a score above 2.7 indicates high risk for fibrosis and should prompt referral. For scores in the intermediate range, the Diabetes Canada guidelines recommended second-line testing, which is typically done with FibroScan. Additionally, Obesity Canada recommends fibrosis risk assessment for people with obesity as well as those who are overweight and have metabolic risk factors.

Discussing treatment, Dr. Sebastiani emphasized



GIADA SEBASTIANI, MD

that lifestyle modification remains foundational. The best results come from combining diet, aerobic exercise, and resistance training. The Mediterranean diet has the strongest evidence base, although it should be adapted to the patient's culture and preferences. She offered two practical "pearls": coffee is liver-protective, with three cups of black coffee daily associated with less advanced liver disease, while sugar-sweetened and fructose-rich drinks should be eliminated because of their strong association with liver fat and oxidative stress.

Pioglitazone has longstanding evidence in MASLD-related liver fibrosis, while the next iteration of the Diabetes Canada guidelines is expected to more strongly support semaglutide based on recent evidence. The ESSENCE trial showed semaglutide improved both steatohepatitis as well as liver fibrosis, resulting in Health Canada to recommend semaglutide for as a treatment for MASH with F2 to F3 fibrosis. In 2024, resmetirom became the first FDA-approved therapy for MASH, though it is not yet available in Canada. Dr. Sebastiani also reviewed incretin-based therapies, noting that weight loss of more than 5% can reduce MASH and weight loss of more than 10% can improve fibrosis. Other agents under investigation include survodutide, tirzepatide, and retatrutide.

Finally, Dr. Sebastiani cautioned that rapid weight loss can worsen sarcopenia, which has been associated with increased mortality and end-stage liver outcomes in patients with MASLD. She recommended more gradual weight loss and protein supplementation as well as resistance training in patients with liver disease taking incretin-based therapies. Another note of caution was the high rate of cardiovascular complications in patients with MASLD. Dr. Sebastiani noted that statins are safe, even in patients with cirrhosis, and can reduce cardiovascular morbidity and mortality.

Treatment and Management of Microscopic Colitis in 2026

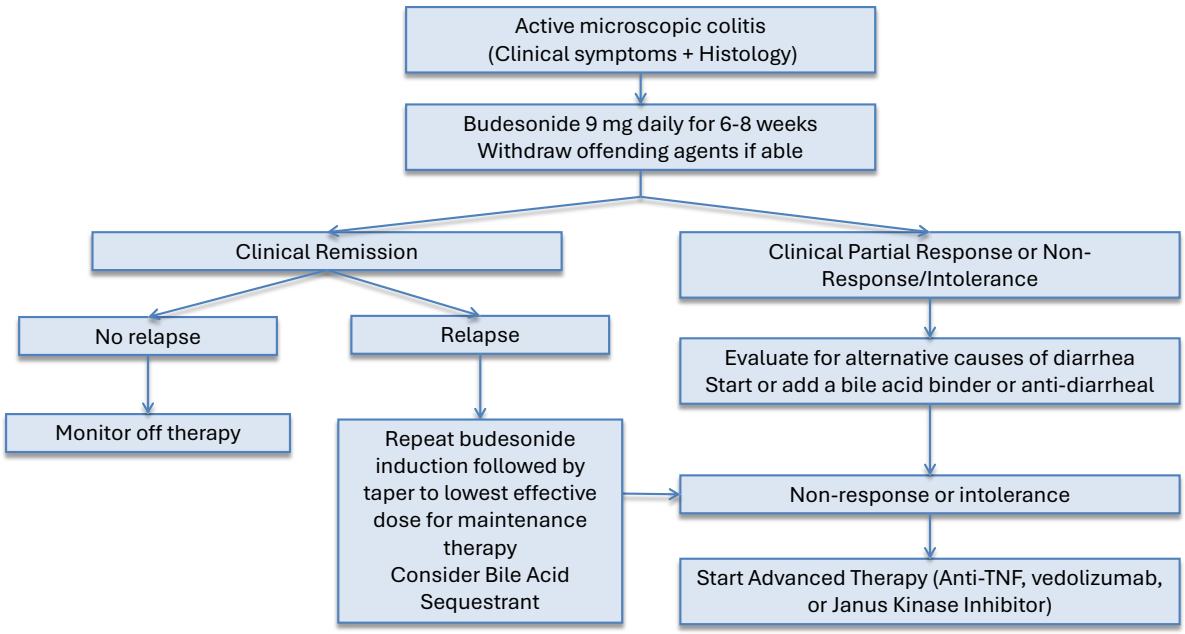
KRISTIN BURKE, MD, MPH

Dr. Burke opened with the case of a 75-year-old woman with a history of hypothyroidism and 5 years of watery diarrhea. The patient had five to seven Bristol type 7 stools daily, nocturnal stools, severe urgency, weekly incontinence, cramping, and major lifestyle disruption, including cancelling planned trips. Her infectious workup was negative, thyroid function was normal on levothyroxine, and she had a normal esophagogastroduodenoscopy and normal duodenal biopsies. Colonoscopy appeared nearly normal, with only mild erythema and edema, but biopsies confirmed microscopic colitis. Dr. Burke stressed that diagnosis requires biopsies of both the right and left colon, because rectal or rectosigmoid biopsies alone will miss 73% and 29% of cases, respectively.

Microscopic colitis was first described in 1976, and its incidence rose until about 2010. Swedish population data suggest a lifetime risk of 0.87% in women and 0.37% in men (Bergman and Khalil, 2019). Pathogenically, environmental triggers act on the gut microenvironment in genetically predisposed people, producing an inappropriate immune response.

A small study of nine patients with collagenous colitis showed that when the fecal stream was diverted, diarrhea resolved and the collagen layer normalized; when intestinal continuity was restored, disease recurred. Small case-control studies show enrichment of pro-inflammatory oral-associated taxa (*Veillonella dispar*) and loss of protective microbes (*Akkermansia muciniphila and Bacteroides stercoris*). Bile acid malabsorption may also contribute, with a study of 35 patients finding a significant decrease in the expression of the farnesoid X receptor in the right and left colon of patients with microscopic colitis, compared with controls. Many environmental risk factors have been associated with disease development, including proton pump inhibitors, SSRIs, NSAIDs, hormone therapy, oral contraceptives, diuretics, checkpoint inhibitors, older age, female sex, smoking, lean BMI,

Microscopic Colitis Management Algorithm



Burke KE et al., Nature Review Disease Primers, 2021

enteric infections, and high consumption of coffee and red wine.

For active, histologically confirmed microscopic colitis, first-line therapy includes budesonide 9 mg daily for 6 to 8 weeks, as well as the withdrawal of possible offending agents when feasible. In a 2014 study, remission occurred in 84% of budesonide-treated patients versus 44% with mesalamine and 42% with placebo in the per-protocol group (Miehlke et al, 2014). However, relapse after stopping budesonide affects about 85% of patients. Low-dose maintenance budesonide is effective in treating recurrence in approximately 60% of patients at 1 year (Munch et al, 2016). Low-dose budesonide appears to be relatively safe in studies following patients for 4 to 5 years, although Dr. Burke cautioned that budesonide may not be appropriate for patients with osteoporosis.

Bile acid sequestrants such as cholestyramine, colesevelam, and colestipol can also be helpful, with a 2022 study of 79 individuals demonstrating a response rate of 76% in budesonide-naïve patients and dose reduction (50%) or discontinuation (20%) among budesonide-dependent patients. Dr. Burke discouraged immunomodulators due to limited efficacy and continued budesonide dependence in approximately 41% of patients (Cotter et al, 2017).

For partial response, nonresponse, or intolerance to budesonide, Dr. Burke advised reassessing for alternative causes of diarrhea, including celiac disease and thyroid dysfunction, and considering bile acid binders or antidiarrheals. A meta-analysis of small studies and case series published in 2024 showed a pooled induction remission rate of 63.5% with vedolizumab, versus 57.8% with infliximab and 39.3% with adalimumab, and a higher pooled maintenance remission rate with vedolizumab versus infliximab and adalimumab. However, a multicenter, retrospective study of 142 patients found that JAK inhibitors led to higher clinical response and remission than biologics in both collagenous and lymphocytic colitis.

Returning to the patient case, Dr. Burke explained that the patient failed multiple biologics but responded to upadacitinib, which led to normal bowel function and a major improvement in quality of life. She concluded by recommending moving to advanced therapies for patients who are steroid- and/or bile acid sequestrant-refractory.

Controversies in IBD: CRC Screening in IBD

SANJAY MURTHY, MD, MSc, FRCPC

Dr. Murthy opened by noting that CRC surveillance is time-consuming for physicians and patients and inconvenient for patients with IBD, as patients with IBD may undergo dozens of colonoscopies over a lifetime. CRC risk is about 1.5 to 2.5 times higher in patients with IBD than in the general population. About 20% of CRC cases are diagnosed prior to the recommended 8 to 10-year post-diagnosis surveillance initiation and 30% of CRC detected in patients with IBD are post-colonoscopy cancers (either missed or early interval cancers), a rate five times higher than the general population. Meanwhile, 80% to 90% of surveillance colonoscopies in individuals with IBD do not identify neoplasia. This raises the questions of whether current screening is adequate.

Dr. Murthy then reviewed how chronic, recurrent bouts of inflammation can increase CRC risk. Colonoscopy remains the main tool for detection, yet optimal colonoscopy surveillance may be limited by poor bowel preparation, active inflammation, post-inflammatory polyps, scarring, and inconsistent use of image enhancement. Some neoplastic lesions in patients with IBD may also be flatter, more indistinct, or ulcer-like, blending into the surrounding mucosa and making them more difficult to detect. However, 90% of CRC precursor lesions in IBD appear as conventional tubular or serrated adenomas.

To improve CRC prevention or early detection, surveillance should begin 8 to 10 years after IBD symptom onset rather than after the date of the IBD diagnosis. Each exam should be conducted under the optimal conditions, including high-quality bowel preparation, minimal inflammation, and use of image-enhancing adjuncts. Gastroenterologists should not merely look for protruding polyps, but also subtle mucosal differences, including changes in colour, vascularity, nodularity, elevation, and ulceration.

Reviewing adjunctive technologies, Dr. Murthy noted that head-to-head studies suggest virtual chromoendoscopy may be comparable to dye spray chromoendoscopy, leading the AGA to accept it as a practical alternative. Dr. Murthy said he uses virtual chromoendoscopy for routine surveillance, reserving dye spray for difficult or second-opinion cases. He highlighted the HELIOS trial, which suggested the benefit of dye spray may partly be a result of longer, more careful segmental reinspection rather than the dye itself. He also described the VISION-AI comparing high-definition/linked-colour imaging endoscopy plus artificial intelligence to chromoendoscopy.

Dr. Murthy addressed the notion of extensive non-targeted biopsies, noting the absence of any properly powered trials studying the utility of this practice and the disadvantages of low sampling potential, low neoplasia yield of 0.1% to 0.2%, and increased procedural time and costs associated with this practice. Dr. Murthy and colleagues recently led a Canadian randomized trial that found high-quality targeted sampling to be non-inferior to targeted sampling plus extensive random biopsies.

Dr. Murthy also highlighted data showing that cumulative inflammatory burden, rather than inflammation severity at the most recent colonoscopy, is predictive of neoplasia risk (Choi et al, 2019). In practice, he now recommends closer CRC surveillance in patients with a history of inflammation even if their most recent colonoscopy was normal. He also discussed a study from the Netherlands showing that patients with two negative exams (no active disease, dysplasia, post-inflammatory polyps, or strictures) had no advanced colorectal neoplasia after a median 3.5 years of follow-up, whereas advanced neoplasia was identified in those with one or more positive colonoscopies during the follow-up period (Hove et al, 2019).

Dr. Murthy highlighted that incorporating multiple risk factors into decision making regarding colorectal neoplasia surveillance timing may better risk stratify patients than the current "top risk factor" approach used in practice. Prediction tools such as UC-CaRE incorporate lesion size, incomplete resection, recent histologic inflammation, and multifocal neoplasia to estimate future advanced neoplasia risk among those with dysplasia identified during colonoscopy. Dr. Murthy also highlighted the IBD Advanced Colorectal Neoplasia (aCRN) Risk Assessment Tool, a multivariable prediction model of future advanced colorectal neoplasia risk that was developed at multiple centers in the Netherlands, U.S.A. and Canada. He concluded by describing the Canadian Dynamic-IBD study, which aims to develop a risk prediction model that can be applied at the point of care to guide the need for early versus deferred colonoscopy surveillance.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Update on the Treatment of EoE

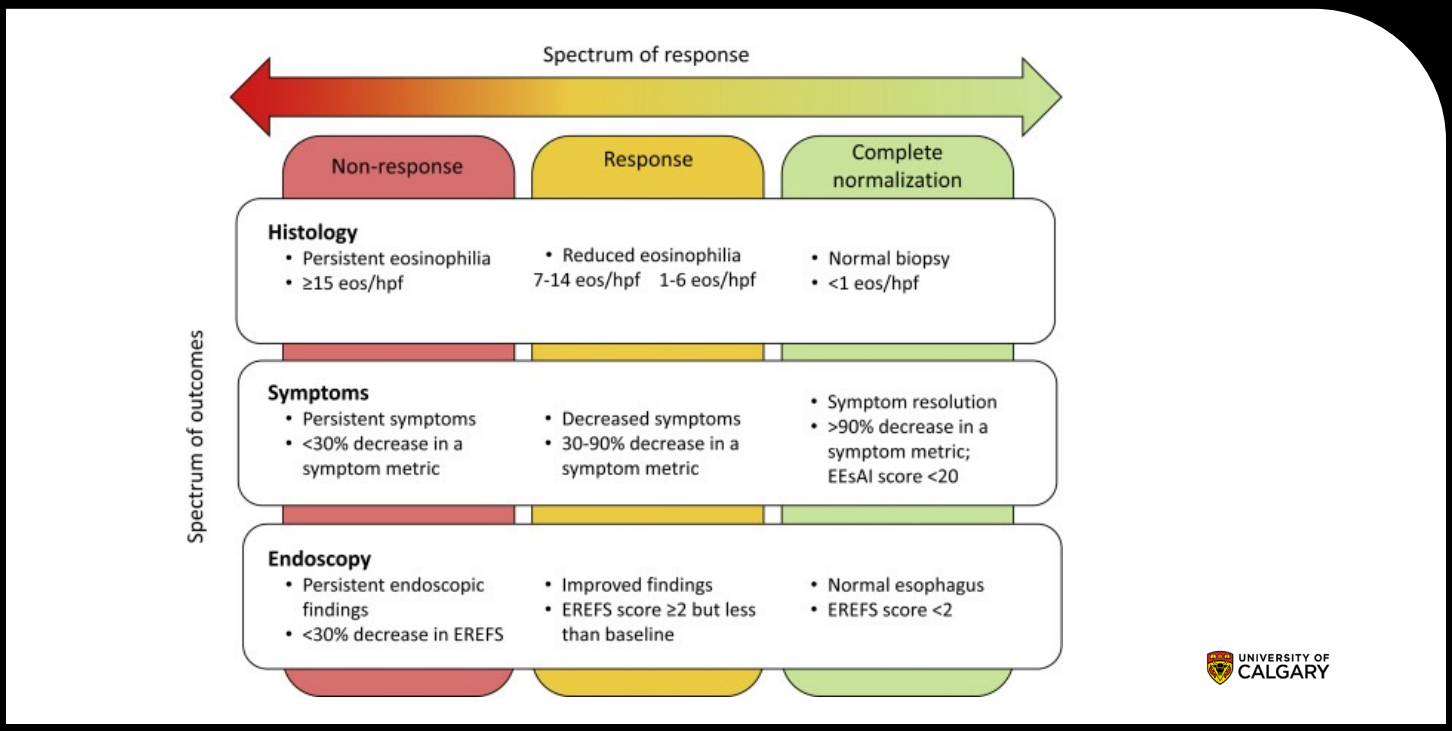
CHRISTOPHER MA, MD, MPH, FRCPC

Dr. Ma grounded the discussion in recently published Canadian consensus recommendations developed by allergists, gastroenterologist, pathologists and dieticians (Avinashi et al, 2025). He emphasized that EoE is a chronic, rather than episodic, disease. It is a non-IgE-mediated, type 2 inflammatory disease that is triggered and perpetuated by barrier dysfunction. While the "classic" EoE patient is often considered to be a young Caucasian man, Dr. Ma highlighted that almost half of new EoE diagnoses occur among women and EoE can occur in people of all ethnicities. He also noted the strong association with asthma, food allergy, CRSwNP, rhinitis, and atopic dermatitis.

The diagnostic criteria include symptoms, including food impaction and dysphagia, with esophageal biopsies showing ≥15 eosinophils/high-power field. Dr. Ma highlighted that patients should not undergo esophageal biopsy for asymptomatic EoE, due to the risk of false positive diagnoses. In addition, Dr. Ma discussed the importance of excluding other possible diagnoses. He emphasized the importance of high-quality and methodical endoscopy. Nine of 10 EoE patients have visible abnormalities if the esophagus is carefully inspected. At least four to six biopsies from two levels are recommended to ensure EoE is not missed. Canadian consensus recommendations additionally advise that stomach and duodenum biopsies be considered at first presentation to assess for other gastrointestinal disorders.

Reviewing treatment, Dr. Ma emphasized shared decision-making because available options all have trade-offs and long-term adherence is important. First-line options include PPIs, swallowed topical steroids, and empiric diet elimination. PPIs can induce histologic remission in approximately half of patients. However, PPIs should be stopped 3 to 4 weeks before diagnostic endoscopy if confirmation is needed. Dr. Ma emphasized that dilation solely treats strictures, and the goal is to achieve an esophageal diameter of 16 mm. Dr. Ma strongly discouraged six-food elimination, favouring one-food elimination of animal milk protein, which has been shown to be non-inferior to six-food elimination. Dr. Ma encouraged referrals to dieticians for support and advice and discouraged elemental diets due to poor taste, frequent need for enteral feeding, high cost, and negative impact on quality of life.

Oral budesonide tablets are highly effective, inducing histologic remission in up to 90% of patients, but relapse is very common and maintenance strategies are required in most cases.



CHRISTOPHER MA, MD, MPH, FRCPC

Weekly dupilumab is highly effective for steroid-refractory patients, but access remains limited in Canada because it is not publicly reimbursed for EoE. Dr. Ma encouraged compassionate use requests. When evaluating treatment response, Dr. Ma explained that the presence or absence of dysphagia alone does not accurately reflect disease control. The response evaluation should also include histology and endoscopy. While formal instruments are not required, Dr. Ma encouraged his peers to discuss quality of life with patients, highlighting a significant burden especially in adolescents.

Dr. Ma concluded by discussing the unanswered question of how long patients should stay on therapy. Long-term treatment is reasonable so long as disease control is achieved, but stronger evidence is still needed. The long-term treatment decision should depend on disease severity and shared decision-making, balancing the risks and benefits of treatment with the risks of complications.

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