Gottumukkala Subba Raju, Peush Sahni, and Sachin Wani, Section Editors

Management of Small Bowel Crohn's Disease Strictures: To Cut, to Stretch, or to Treat Inflammation?

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This patient's case history (void of protected health information) was discussed in a multidisciplinary meeting including adult gastroenterologists (CL, BGF, FR), radiologists (MB, JGF), and a colorectal surgeon (SH) with an adult gastroenterologist (FR) serving as the moderator. We summarize the case history and discussion in this article.

Case History

A 27-year-old woman was diagnosed with ileocolonic Crohn's disease (CD) in 2020. She initially presented with postprandial abdominal cramping, diarrhea and a 30-pound weight loss over 1 year. Index colonoscopy at time of diagnosis showed a strictured ileocecal valve with ulcerations (Simple Endoscopic Score for CD [SES-CD] ileal segment score: 11; ileal subscores: size of ulcers: 3, ulcerated surface: 3, affected surface: 2, narrowing could not be passed with an adult ileocolonoscope: 3). There were erosions and ulcerations noted in the cecum Figure 1*A*). Her laboratory investigations revealed iron deficiency anemia (hemoglobin of 108 g/L, mean corpuscular volume of 78 fL, and ferritin of 14 μg/L) with an elevated C-reactive protein of 9.1 mg/L. Fecal calprotectin was not submitted. Intestinal ultrasound (IUS) examination revealed 15 cm of thickened terminal ileum with a maximal bowel wall thickness of 9.8 mm (normal $\leq$3 mm), moderate inflammation within the wall of the bowel, and the surrounding echogenic inflammatory fat (modified Limberg 3). The submucosa layer was thickened and echogenic suggestive of chronicity. There was luminal narrowing at 2 mm (normal $\leq$2 cm) with prestenotic dilation at 2.5 cm and wall stratification loss (Figure 1*B*–*D*). The remainder of the colon was normal. Histopathology of the affected areas showed moderately active inflammation with changes of chronicity.

Question: At this time, what would you recommend as the next best step in management?

A. Initiate anti-tumor necrosis factor therapy (with or without immunomodulator).

B. Initiate anti-interleukin (IL)-12/IL23 or anti-IL-23 inhibitor therapy.

C. Initiate Janus kinase inhibitor therapy.

D. Endoscopic balloon dilation.

E. Ileocolic resection.

F. Answer A or E.

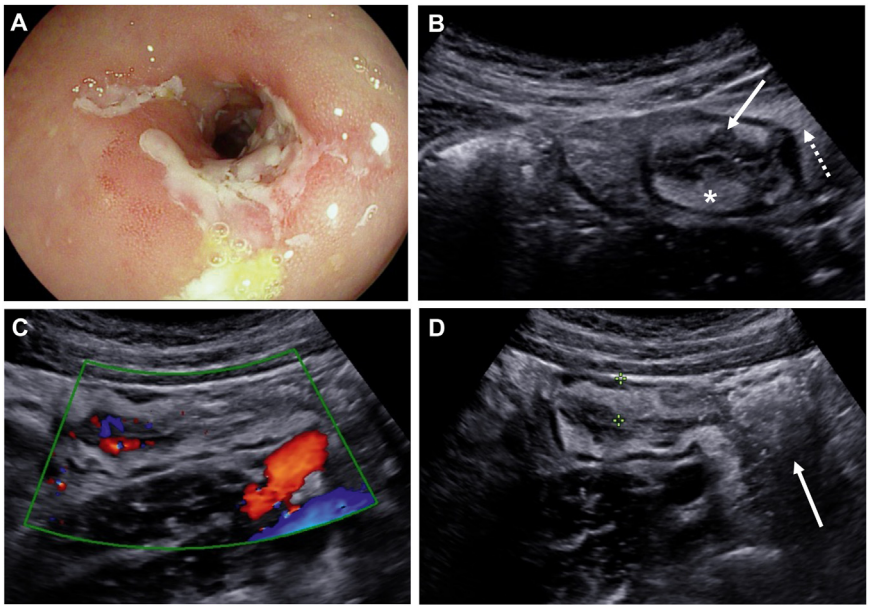

Figure 1. (*A*) Narrowed patulous ileocecal valve of patient with Crohn's disease. The endoscopist was unable to intubate the terminal ileum with a pediatric ileocolonoscope. Simple endoscopic score 11 (size of ulcers: 3, ulcerated surface: 3, affected surface: 2, narrowing cannot be passed with ileocolonoscopy: 3). Crohn's disease stricture on intestinal ultrasound. (*B*) Thickened Crohn's disease terminal ileum in cross-sectional view with echogenic thickened submucosa (*asterisk*), narrowed lumen, surrounding inflammatory fat (*dashed arrow*), and loss of echo stratification (*arrow*). (*C*) Longitudinal view of ileum with long chains of hyperemia with signals in perienteric fat (modified Limberg 3 color Doppler signal). (*D*) Longitudinal view of ileal stricture with thickened bowel wall (*calipers*), and prestenotic dilation with posterior wall shadowed by gas (*arrow*).

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Answer (Page 1283): Initiate Anti-Tumor Necrosis Factor Therapy (With or Without Immunomodulator) or Ileocolic Resection

The correct answer is F.

Multidisciplinary Case Discussion

FR: This patient's clinical presentation is not uncommon for a patient with CD. CD is a form of inflammatory bowel disease and results from a combination of environmental factors, a genetic predisposition, and a dysregulated immune system leading to chronic relapsing and remitting inflammation. Inflammation can lead to tissue damage in the bowel wall, which ultimately culminates in complications, such as stricture formation. Just like in our patient, strictures can present at diagnosis in approximately 10% of patients.[1,2] Strictures are clinically relevant because they lead to symptoms of obstruction and are a major cause of patient morbidity.

FR: Dr Lu, can we make a diagnosis of stricturing ileal CD by symptoms alone or do we need additional modalities?

CL: Symptoms alone are not specific enough to diagnose stricturing ileal CD over luminal inflammation alone as there is an overlap in clinical presentation between the two. We need additional modalities to confirm the diagnosis and for this we recommend endoscopy and cross-sectional imaging.[2] Small bowel strictures on endoscopy are defined by the inability to pass an adult or pediatric ileocolonoscope through the stricture (examples for endoscopy images for strictures of different severities can be found in Figure 2). It has to be noted, however, that this metric is highly dependent on the endoscopist and patient factors and hence reliability is not high.[3] In clinical practice we, therefore, consider cross-sectional imaging as optimal to diagnose strictures, also because this allows us to view extra-enteric complications, such as abscesses, internal penetrating disease, or malignancy.[4] This is relevant because internal penetrating disease frequently coexists with strictures and its presence influences clinical decision-making.

FR: Dr Lu nicely delineates the need to look beyond symptoms to diagnose stricturing CD and our reliance on endoscopy and cross-sectional imaging. In fact, clinically, we noted a disconnect between symptoms and the presence of strictures. Despite luminal narrowing and clear signs for strictures on endoscopy or imaging, the patient may have no or minimal symptoms. This may be related to patients changing their diet to avoid high residue components or to other factors we are yet to understand.

FR: Dr Lu, are there validated endoscopic scoring systems for stricturing CD?

CL: There are no validated endoscopic scoring indices dedicated to stricturing disease. The 2 current validated endoscopic scores for luminal CD, the SES-CD and the CD-Endoscopic Index of Severity, contain subscores for stenosis. Although this subscore has been used to assess drug response of luminal stenosis, it was found to have the lowest reliability of among the subscores of SES-CD.[3]

FR: Dr Baker, as an abdominal radiologist, can you walk us through the accuracy of cross-sectional imaging for diagnosis of stricturing CD?

MB: Cross-sectional imaging is required and able to accurately diagnose stricturing CD.[5] Cross-sectional imaging is also recommended before making treatment decisions and for following treatment response, as there may be complicating penetrating disease. All 3 modalities—computed tomography enterography (CTe), magnetic resonance enterography (MRe), or IUS examination—have a comparable accuracy for diagnosing CD strictures. IUS has a sensitivity of 80%–100%, specificity of 63%–100%; CTe has a sensitivity of 85%–100% and a specificity of 39%–100%; and MRe has a sensitivity of 75%–100% and a specificity of 91%–100%.[5] Based on patient and center preference, all 3 modalities can be used interchangeably. At the time of diagnosis, CTe or MRe are preferred at least once to image all of the proximal small bowel to understand the complete extent of the patient's disease (eg, proximal strictures, skip lesions with inflammation, perianal disease and extra-enteric complications), which may not be able to be visualized endoscopically or with IUS examination owing to bowel gas.[6]

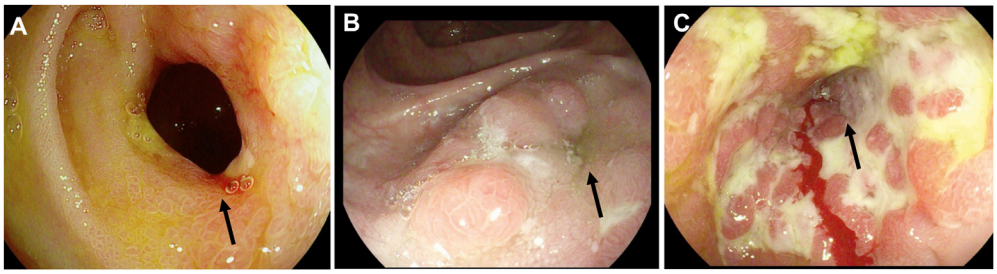

Figure 2. Examples for endoscopy images for strictures of different severities. (*A*) Distal ileal stricture traversable with pediatric colonoscope (*arrow*). (*B*) Deformed ileocecal valve with orifice too narrowed for colonoscope (*arrow*). (*C*) Severely ulcerated distal ileal stricture unable to be intubated (*arrow*).

FR: One important decision point in treating strictures, also in our patient here, is to distinguish the degree of inflammation from fibrosis within a stricture. Inflammation alone can lead to luminal narrowing and wall thickness, which may respond very well to anti-inflammatory therapy, whereas strictures with a high fibrotic component may be better served with dilation or resection.

FR: Dr Fletcher, are there imaging modalities that can be used to distinguish if a stricture contains predominantly either inflammation or fibrosis?

JGF: Cross-sectional imaging can assess the presence and extent of bowel inflammation with robust accuracy compared with histopathology and endoscopy.[5] However, IUS, CTe, and MRe are unable to accurately determine the exact combination of inflammation, fibrosis, and muscular hypertrophy/hyperplasia, which coexist in most CD strictures.[5,7] Magnetization-transfer MR imaging has shown promise when compared with a well-defined histopathological reference standard for distinguishing nonfibrotic and mildly fibrotic strictures from those with greater degrees of fibrosis[8,9]; however, this work is not replicated widely, and its ability to distinguish fibrosis from muscular hypertrophy is unknown. However, CTe and MRe are able to accurately and reproducibly measure small bowel stricture morphology, such as stricture length, wall thickness and prestricture dilation[10,11] and those morphological features may be used after medical treatment to determine response to therapy. In my experience, predominantly inflammatory strictures can respond dramatically and can show a marked shortening or even undergo complete resolution of associated small bowel dilation with a decrease in bowel wall thickness. Those changes may portend better long-term outcomes and can affect the decision for surgical resection or not.[12] Unfortunately, transmural healing will occur in a small minority of patients with CD stricture. Endoscopy with or without endoscopic biopsies, or biomarkers are also unable to determine the degree or extent of fibrosis present in a stricture.[2] Hence, the benefit of imaging strictures is diagnosis, exclusion of penetrating complications, determining the degree of obstruction, and assessing response to medical treatment rather than assessing their exact composition. Currently, our ability to understand the relative composition of strictures can only be inferred from changes on imaging findings (length, wall thickness, and associated small bowel dilation) over time and while on treatment. There are several other experimental techniques under investigation for IUS, CTe and MRe, several of which hold promise.[13] Validation studies are awaited before we can implement them in clinical practice. One recent example is the use of radiomics, which provides quantitative imaging descriptors from routine radiographic images.[7] Those descriptors can then be linked with the histopathologic features within a stricture, such as inflammation or fibrosis. This is a rapidly moving field, and more information is to come soon.

FR: This is important information. So, we are not able to determine how much fibrosis is in a given stricture. This is an area of large unmet need as inflammation and fibrosis invariably coexist within each stricture in varying degrees. Another observation we still must learn more about is the contribution of the thickening of the muscularis propria to luminal narrowing in CD strictures. Also, here imaging techniques are being developed to delineate the contribution of this compartment.

FR: Dr Holubar, when should surgical resection of ileal strictures be considered?

SH: Identifying the appropriate timing for surgical resection in patients with small bowel stricturing CD may be challenging when perienteric complications, such as internal penetrating disease or abscesses, are absent. Indications for surgery include inflammatory masses, abscess, perforation, or suspicion of dysplasia or malignancy associated with a stricture, as well as failure to thrive with poor nutritional status.[14] In this case, our patient does not have any perienteric complications. However, she has poor nutritional status with a loss of 30 pounds over the past year and immediate surgical resection at this time without optimization may not be the best option for her. Nutritional status may be evaluated using a number of tools such as the Malnutrition Universal Screening Tool or Malnutrition Inflammatory Risk Tool. Given that inflammation is present in the stricture, choosing the best anti-inflammatory therapy first is a reasonable choice. Having said that, early resection of patients with limited luminal ileal CD before or instead of biologic therapy has demonstrated efficacy.[15,16] Although these data are interesting and relevant, it has to be noted that this work was performed in a highly selected patient population and the results might not be generalizable to the general CD population or patients with stricturing CD, like in our case. Having said that, should the patient have a preference for surgical resection, we can offer this option after nutritional optimization, typically with exclusive enteral nutrition, and aggressively treating the anemia with parenteral iron infusions.[17]

FR: Dr Lu, would this patient be a candidate for endoscopic balloon dilation?

CL: If inflammation is not severe, defined by the presence of deep ulcerations, and the stricture is <5 cm in length, accessible by endoscopy, and nonangulated with absence of concomitant CD features, such as abscess, phlegmon, internal penetrating disease, dysplasia or malignancy, then endoscopic balloon dilation may be considered.[2,18] This procedure needs to be accompanied with or followed after start of effective anti-inflammatory therapy. In this patient's case, endoscopic balloon dilation is not indicated owing to the length of stricture at 15 cm and because the degree of inflammation is significant.

FR: Can you walk us through how successful endoscopic balloon dilation is?

CL: If choosing the right patient, as delineated prior, this approach is quite effective. In a systematic review assessing endoscopic balloon dilation in CD strictures, 51.8% (36.0–63.6) of patients required a repeat dilation within 12 months and 30.1% (17.4–40.9) needed a surgical resection. Dilating a stricture >5 cm with endoscopic balloon dilation has a shorter time to surgical resection and is usually technically more challenging. There is an 8% increased risk for surgery with every

once centimeter increase in length of a stricture.[2,18] Hence in routine practice we do not dilate strictures >5 cm, as is the case in our patient. Serial dilation is an option in case the patient redevelops symptoms. Generally speaking, dilation is a temporizing measure for strictures that can be useful in conjunction with effective medical therapy.

FR: Thank you for sharing this with us. Can you provide some information about the technical aspects of balloon dilation in amendable patients?

CL: The luminal diameter of the stricture influences the initial balloon size. I usually choose a starting balloon diameter of 1–2 mm greater than the minimal stricture lumen. We recommend use of graded balloons and perform a maximum amount of 3 dilation steps. The balloon inflation time per diameter is one minute. An adequate luminal diameter at the end of dilation therapy, regardless of the number of sessions, is 15–18 mm. The time to reassessment after dilation depends on the endoscopic and imaging appearance of the stricture before dilation, but largely is based on patients' symptom recurrence. This approach is in line with a recently published global consensus project.[2]

FR: We now established that our patient is not a candidate for dilation as the stricture is too long. Dr Holubar mentioned surgical resection is an option. When should a stricturoplasty be performed on a small bowel CD stricture?

SH: Stricturoplasty is generally reserved for patients with short or multiple small bowel strictures as it conserves bowel length without resection. For terminal ileal strictures, an ileocolic resection is typically the procedure of choice, but if there are upstream small bowel strictures concurrent strictureplasties may be performed. In general, stricturoplasty should not be performed when perienteric complications are present.[2]

FR: Let's talk about medical therapy. Currently, no selective antistricture therapies are available. We hence rely on anti-inflammatory treatment approaches. Dr Feagan, which medical therapy should be chosen in this case?

BGF: The number of available therapies for CD has recently expanded. However, the effectiveness for medical therapy for stricturing CD remains uncertain. Most information is available for tumor necrosis factor (TNF) antagonists. The Intensive drug therapy versus standard drug therapy for symptomatic intestinal Crohn's disease strictures (STRIDENT) trial is the only randomized controlled trial evaluating the efficacy of the TNF antagonist adalimumab in small bowel CD strictures, either using standard dosing or an intensified dosing regimen. The primary end point in this important open-label trial was improvement in an obstructive symptom score at 12 months compared with baseline. Most patients (>60%) in both the intensive and standard treatment arms had an improvement in obstructive symptom scores without a difference between the 2 arms. This trial furthermore showed that a more intensive adalimumab dosing regimen was more effective than standard adalimumab therapy in improving stricture parameters on imaging.[19] However, prestenotic dilatation on imaging was not required for inclusion and a hence a subset of these patients did not exhibit prestenotic dilatation. Owing to this, the strictures may have had a high inflammatory, rather than fibrotic component. It remains uncertain if a comparable response to TNF antagonist would have been observed when using the now accepted so called CONSTRICT imaging criteria that require prestricture dilation and represent more advanced strictures.[6,20] The efficacy of TNF antagonists in stricturing CD is also supported by data from the single-arm open-label CREOLE trial as well as a systematic review summarizing observational studies.[21,22]

FR: Are there any data on newer agents for the therapy of CD strictures?

BGF: Data regarding the efficacy of other agents, including vedolizumab, ustekinumab, risankizumab, and upadacitinib in CD strictures remains limited. In a combined post hoc analysis that evaluated a subgroup of participants with endoscopic stenoses in the UNITI and IM-UNITI clinical trials for ustekinumab in luminal CD,[23] the SONIC[24] as well as the CT-P13 clinical trials,[25] 62.5% of patients with endoscopically nonpassable strictures at baseline had a passable stricture or resolution of the stricture after treatment.[26] Of these strictures, 71.5% were located at the ileum. Additionally, a post hoc analysis of the phase III ADVANCE, MOTIVATE, and FORTIFY studies assessed the efficacy of risankizumab in patients with passable and nonpassable stenoses defined by the SES-CD narrowing subscore.[27] Overall, 48.9% of patients with initially nonpassable stenoses had improvements or resolution of strictures after 12 weeks of induction with risankizumab. At week 52, 42.9% of these patients with baseline nonpassable stenoses had a sustained improvement. Finally, in another post hoc analysis of a phase III study with upadacitinib in luminal stenosis in moderate to severely active CD patients, 19% of patients with baseline stenosis had resolution at week 52 on upadacitinib 30 mg/d. However, this difference was not statistically significant compared with placebo.[28] Collectively, these findings underscore the need for dedicated studies in patients with stricturing disease that use standardized inclusion criteria and cross-sectional imaging to evaluate response.

Limited observational data indicate that anti-inflammatory therapy does not provide a long-term benefit for a large portion of patients. For example, 49% of patients in CREOLE underwent surgery within 4 years after the initiation of adalimumab therapy. Accordingly, surgery or endoscopic balloon dilation remain staples of management. In the case of our patient, it is reasonable to start on a TNF antagonist in combination with an immunomodulator. If a patient desires or requires a different drug class, anti-IL12/23 or anti-IL23 are also options. Upadacitinib is available in the United States only after failure of a TNF antagonist. Overall, the choice of medication must consider extraintestinal manifestations, comorbidities, and shared decision-making with the patient.

FR: Thank you, Dr Feagan. Are there clinical predictive factors of strictures that may aid in determining if a stricture is less likely to respond to biologics?

BGF: Predictors of nonresponse to biologics in stricturing disease have been poorly defined. The most commonly used predictors for nonresponse in clinical practice are longer stricture length, the presence of prestricture dilation, and the presence of internal penetrating disease associated with the stricture. The choice and positioning of biologics is comparable

to the choice and positioning of biologics in luminal small-bowel or ileocolonic CD. A recent global consensus on the management of small bowel CD strictures in clinical practice has described that bionaïve patients with fibrostenotic CD can be treated with anti-TNF agents with or without immunomodulators, or ustekinumab as a first-line option, regardless if the stricture is naïve or anastomotic.[29]

FR: Interesting that you mention naïve versus anastomotic strictures. Does it make a difference in approach if the patient has a naïve anastomotic stricture?

CL: This question remains unanswered. It is unclear if anastomotic strictures involve different disease processes relative to naïve strictures. We do not yet fully understand if both respond differently to medical therapies. At this time, the diagnostic criteria for both forms of strictures are identical[20] and approach to therapy is similar.[2] Available data suggest that outcomes between the 2 are comparable. In CD patients with small bowel stricture who were exposed to a TNF antagonist treatment previously, the global consensus recommended treatment with ustekinumab, endoscopic balloon dilation, or surgery.[29] This recommendation was made for both naive or anastomotic strictures. However, this consensus was completed before IL-23 and Janus kinase inhibitors were approved for CD, and these agents may now be considered for this clinical scenario as well.

FR: How long should one treat a stricture before deciding that the stricture is refractory to medical treatment?

BGF: This is an important question. Based on clinical experience, anti-inflammatory medical therapy should only be used if there is active inflammation present as identified on cross-sectional imaging, laboratory markers, and/or endoscopy. We suggest that a lack of clinical or biochemical response by 12 weeks (no change in symptoms or inflammatory markers), and insufficient response by 6 months (no change in symptoms or inflammatory markers or cross sectional imaging) because decision points to deem a stricture refractory to medical management. At 6 months, we recommend optimization of medical therapy if inflammation is present. Endoscopic balloon dilation for strictures <5 cm may be considered if symptoms persist and inflammation has largely resolved, provided there are no contraindications to the procedure. Alternatively, surgical intervention is a viable option if symptoms continue or if inflammation is resistant to medical therapy.

Further Information on the Clinical Case

The patient was started on adalimumab at a standard dosing without concurrent immunosuppressive therapy. The patient experienced initial symptomatic improvement and gained weight; however, her symptoms recurred 4 months later. A bedside follow-up ultrasound examination at 6 months revealed active inflammation and similar findings compared with before therapy initiation, with a bowel wall thickness of 9.5 mm, continued abnormal color Doppler signal in the bowel wall (modified Limberg 2), and perienteric inflammatory fat. The prestenotic dilation was now increased to 3.3 cm. An adalimumab trough concentration returned at 6 μg/mL and the patient received a dose intensification to adalimumab 40 mg weekly. Fecal calprotectin submitted before dose escalation was 1,020 μg/g.

Three months after dose intensification, she remained intermittently symptomatic. MRe imaging 6 months after dose escalation revealed similar findings to her baseline and follow-up IUS scans. The terminal ileum continued to have 15 cm of disease with a maximal bowel wall thickness of 1.0 cm and a luminal diameter of 1.0 mm with prestenotic dilation of 3.0 cm. Repeat therapeutic drug monitoring revealed an adalimumab trough level of 14 μg/L. The patient had minimal symptoms with mild abdominal cramping that improved with an altered diet of less fruits and vegetables. The patient denied having nausea, vomiting, or frequent bloating. She described occasional bloating.

FR: Dr Fletcher, can you walk us through guidance for imaging follow-up of nonresected stricturing CD?

JGF: No clear guidance exists on follow-up imaging of existing stricturing CD after starting medical therapy. Frequency of follow-up imaging is largely driven by accessibility to imaging and patient symptoms.[2] In those with access to IUS, follow-up at 6 and 12 months after medication initiation and every 6 to 12 months thereafter for ongoing follow-up is considered a reasonable choice.[30] Because CTe use is associated with radiation exposure and MRe access may be limited by payors or long waiting lists, these imaging modalities can be used on a yearly basis or as needed during symptomatic exacerbations. However, increased frequency of imaging is at the discretion of the clinician, and sooner time points are acceptable for recurrence or worsening of symptoms and/or laboratory investigations.

Additional medical therapy with either risankizumab or upadacitinib, or surgical resection were discussed with the patient after the MRe findings. Surgical referral and consultation were completed. The patient declined surgical resection and wished to pursue alternative medical therapy. She was started on risankizumab and a prednisone taper. After 6 months, her IUS findings did not improve, and inflammatory markers remained elevated (fecal calprotectin 1,211 μg/g, C-reactive protein 16.4 mg/L). As a result, she proceeded with ileocolic resection with ileocolonic anastomosis. Risankizumab was continued postoperatively because it was believed that the tissue damage was already too severe at start of risankizumab preoperatively to consider this a therapy failure. Histopathology from her surgical resection showed patchy chronic active enteritis with ulceration. Marked amounts of smooth muscle hyperplasia and fibrosis in the submucosa was noted (Figure 3). There was no evidence of dysplasia. A colonoscopy at 6 months after ileocolic resection with ileocolonic anastomosis revealed no evidence of recurrence of CD (Rutgeert's i0).

FR: Very interesting case. There remains substantial heterogeneity among histologic scoring systems for assessing CD strictures. None of the currently available indices have had formal validity or reliability testing. In particular, muscular

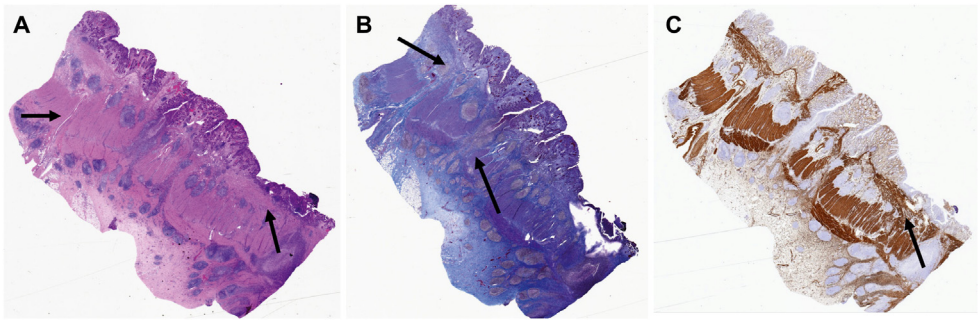

Figure 3. Full-thickness histopathology of the patient's stricture resection. (*A*) Smooth muscle hyperplasia and fibrosis in submucosa (*arrows*) on hematoxylin and eosin stain. (*B*) Excessive extracellular matrix deposition is stained in blue (*arrows*) on Masson trichrome stain. (*C*) Immunostain for smooth muscle actin shows smooth cells and bundles in the submucosa highlighted in brown (*arrow*). Each panel represents the same area in subsequent cuts from the same tissue

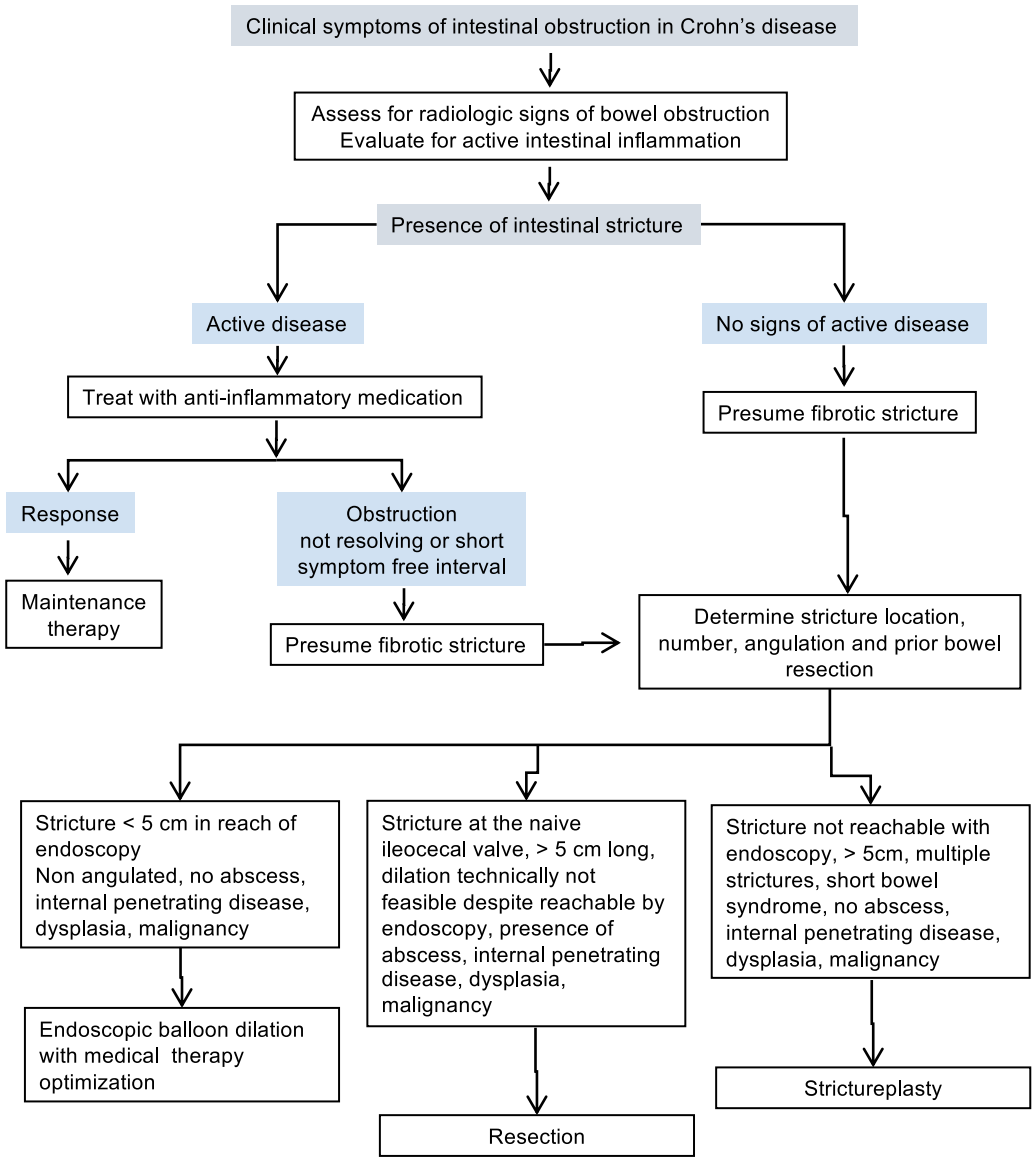

Figure 4. Treatment algorithm for stricturing Crohn's disease.

hypertrophy or hyperplasia are important pathological components of strictures lacking uniformity in grading and descriptions.

FR: Dr Feagan, can you provide a look into the future of antistricture therapy?

BGF: Despite major advances in the field, trials testing novel antifibrotic drugs in the intestine have not been performed. This was driven by lack of consensus on definitions and clinical trial end points, since antifibrotic drug candidates are already available for other organs.[31] The dire need for reliable definitions and the heterogeneity in approaches has now been overcome by the Stenosis Therapy and Anti-Fibrotic Research (STAR) Consortium. The STAR consortium recently created clear definitions for what defines a stricture and what constitutes improvement.[20] Multiple projects are now underway to build monitoring tools and end points for clinical trials, including a patient-reported outcome tool and stricture radiology indices.[10,11] This work has culminated in the first clinical trial for stricturing CD using an antifibrotic drug. In this trial, an orally delivered transforming growth factor-b1 signaling (ALK5) inhibitor is administered to patients with symptomatic strictures (NCT05843578). The trial is currently underway and the results are eagerly anticipated. In the future, a safe and effective antifibrotic agent may be used in combination with our anti-inflammatory medications.

FR: Thank you to our multidisciplinary panel, who walked us through a case of stricturing CD, but also provided a glimpse into the future. A clinical decision tool is depicted in Figure 4. The key take home points are:

1. Strictures are a common and serious complication in CD.
2. Strictures can be diagnosed with endoscopy, but the preferred approach is cross sectional imaging such as IUS, MRe, or CTe.
3. Diagnosis on cross sectional imaging is highly accurate, but we cannot determine the degree of fibrosis within a stricture.
4. Control of inflammation is the first step in therapy.
5. Endoscopic balloon dilation is indicated for strictures <5 cm, but imaging needs to be performed to exclude extraluminal complications or malignancy.
6. Serial dilations are feasible.
7. Perform resection in case of stricture with associated fistula, abscess, phlegmon, or malignancy.
8. Strictureplasty remains an option for patients with mid small bowel strictures, multiple strictures, or short gut.

Keywords: Fibrostenosis; Stricture; Intestinal Ultrasound; Computed Tomography Enterography; Magnetic Resonance Enterography.

Supplementary Material

Note: To access the Supplementary References 16–31 accompanying this article, visit the online version of *Gastroenterology* at www.gastrojournal.org, and at https://doi.org/10.1053/j.gastro.2024.08.030.

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