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Editorial

When is Surgery an Option for Infectious Bowel Disease?

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Abstract

Inflammatory bowel disease is comprised of two major disorders: Crohn's disease and ulcerative colitis. Crohn's disease is a chronic inflammatory, idiopathic disorder that affects any part of the gastrointestinal tract from the mouth to the anus, while ulcerative colitis is a chronic inflammatory, idiopathic disorder that affects the large bowel. Despite the new and ever expanding array of medications for the treatment of inflammatory bowel disease, there are still clear indications for operative management.

Keywords: Crohn's Disease; Ulcerative Colitis; Surgical Treatment; Inflammatory Bowel Disease; Laparoscopic Surgery

Introduction

Crohn's disease is a chronic inflammatory, idiopathic disorder that affects any part of the gastrointestinal tract from the mouth to the anus. Individuals with Crohn's disease often experience periods of symptomatic relapse and remission. The estimated prevalence of Crohn's disease is about 322 cases per 100,000 persons among adults in Europe and 43 per 100,000 among children. It is considered a disease of the rich, as the estimated prevalence of Crohn's disease is higher in urban areas and upper socioeconomic classes. It is thought that this is due to increased access to the health care system [1].

The exact cause of Crohn's disease remains unknown. Several factors (genetic, environmental, immunologic, dietary etc.) are thought to play a role to the pathogenesis of Crohn's disease. IBD1 gene located on the chromosome 16 is believed to contribute to the pathogenesis of the disease. Furthermore, infectious agents such as Mycobacterium paratuberculosis, Pseudomonas species, Listeria species, environmental factors such as tobacco use and dietary factors such as a diet high in fatty acids have all been implicated in the pathogenesis of Crohn's disease. Crohn's disease is a chronic inflammatory disease that affects primarily the superficial mucosa and then spreads to the deeper mucosal areas, causing transmural inflammation. Granuloma formation is pathognomonic of Crohn disease. The granulomas extend through all layers of the intestinal wall and into the mesentery and the regional lymph nodes.

Clinical manifestations of the disease include constitutional symptoms such as low grade fever, weight loss, and fatigue. As mentioned before, Crohn's disease affects any part of the gastrointestinal tract. Oral manifestations such as oral ulcers are quite common. When the small intestine is affected, diarrhea, malabsorption, weight loss and abdominal pain are the predominant symptoms. When the colon is affected (terminal ileum is the most common affected part of the gastrointestinal tract), the patients report diffuse pain accompanied by diarrhea with blood and mucus in the stool. Extra-intestinal manifestations are also quite common in Crohn's disease. It also affects skin, joints, eyes, liver and bile duct. Arthritis is the most common extra-intestinal manifestation. Central or axial arthritis, such as sacroiliitis, or ankylosing spondylitis, may also occur. Examination of the skin reveals erythema nodosum and pyoderma gangrenosum. Eye manifestations include uveitis, iritis, and episcleritis. Primary sclerosing cholangitis occurs in approximately 5 percent of patients [2,3].

Complications of the Crohn's disease include: Fistulae that may develop between the diseased bowel and a variety of adjacent tissues, abdominal abscesses and small bowel obstruction and perianal abnormalities (abscesses and fistulae). It is worth mentioning that in threatening the above mentioning situations, a consultation with a surgeon is often needed [4].

The diagnosis of Crohn's disease is based on laboratory tests, imaging studies and more invasive procedures, such as endoscopic visualization and biopsy. Routine laboratory studies that are quite useful in the diagnostic process of Crohn's disease include: CBC count, chemistry panel, liver function tests and inflammatory markers. It is worth mentioning that fecal calprotectin is a new marker that is proven to be quite useful in the diagnostic process and in the prediction of clinical relapse of inflammatory bowel disease. Specifically, the fecal calprotectin is a calcium- and zinc-binding protein, which for practical purposes can be considered to be neutrophil-specific. Fecal calprotectin test is a functional quantitative measure of intestinal inflammation. Therefore, fecal calprotectin tests are quite useful in gastroenterologists, in order to distinguish inflammatory bowel disease from other common diseases, such as irritable bowel syndrome. Except from the above, calprotectin also predicts imminent clinical relapse with an 80% sensitivity and accuracy in patients with established, relatively

asymptomatic inflammatory bowel disease (ulcerative colitis or Crohn's disease) [5].

Treatment options are individualized based on the severity of symptoms and the complications of the disease. In mild affected patients, such as patients with mild disease limited to the terminal ileum and/or colon and no complications, enteric-coated budesonide as the first line treatment for inducing remission. The use of 5-aminosalicylates (5-ASA) for Crohn's disease is controversial, and we limit its use to patients with mild Crohn's disease with limited ileocolonic involvement who prefer to avoid glucocorticoids. For patients with limited ileitis and mild symptoms, a slow release, oral 5-ASA agent is suitable, such as mesalamine (eg, Pentasa or Asacol). By contrast, sulfasalazine (the prodrug of 5-aminosalicylate) is less useful for ileitis because colonic bacteria must cleave the drug to release the active 5-ASA moiety, so it is reserved for cases of colitis. For low-risk patients with mild Crohn's disease who achieved remission with a glucocorticoid tapering and then discontinuing the glucocorticoid is recommended. For low-risk patients with mild Crohn's colitis who achieved remission with a 5-ASA agent (or sulfasalazine), the same agent should be used for long-term maintenance therapy [6-8]. On the contrast, patients with moderate to severe Crohn's disease ie patients younger than 30 years , with tobacco use with elevated C-reactive protein and/or fecal calprotectin levels, deep ulcers on colonoscopy ,long segments of small and/or large bowel involvement, perianal disease, extra-intestinal manifestations, history of bowel resections, first-line options for induction therapy include a biologic agent tumor necrosis factor-alpha (TNF) inhibitor (eg, infliximab) with or without an immunomodulator (eg, azathioprine [AZA], 6-mercaptopurine [6-MP], or methotrexate).After clinical, endoscopic, and histologic remission following induction with combination therapy, the same agents are used for one to two years [9-12]. For many patients with Crohn's disease, may have a continuous and progressive course of active disease, while approximately 20 percent of patients experience prolonged remission after initial presentation. There is an increased incidence of squamous cell carcinoma of the anus and skin, adenocarcinoma of the small bowel and duodenal neoplasia in patients with Crohn's disease [13-15].

Ulcerative colitis is a chronic inflammatory, idiopathic disorder that affects the large bowel. It is three times more common than Crohn disease.In the United States, about 1 million people are affected with ulcerative colitis. The exact cause of ulcerative colitis remains unknown. Several factors (genetic, environmental, immunologic, dietary etc.) are thought to play a role to the pathogenesis of the disease. A family history of ulcerative colitis (observed in 1 in 6 relatives) is associated with a higher risk for developing the disease. Disease concordance has been documented in monozygotic twins. Unlike Crohn's disease, smoking is negatively associated with ulcerative colitis. Dietary factors such as milk consumption may exacerbate the disease. Last but not least, psychological and psychosocial stress factors can play a role in exacerbations of ulcerative colitis.

Ulcerative colitis is a chronic inflammatory disease that affects colonic mucosa. It involves erosions and/or ulcers and individuals with Crohn's ulcerative colitis often experience periods of symptomatic relapse and remission. As mentioned above, ulcerative colitis affects predominantly the colon. Clinical manifestations of the disease include rectal bleeding and diarrhea with mucus in the stool. Lower abdominal pain is also a common symptom. Extra-intestinal manifestations are also quite common in ulcerative colitis. These include uveitis, pyoderma gangrenosum, pleuritis, erythema nodosum, ankylosing spondylitis, and spondyloarthropathies. Among them arthropathies are the most common, with an incidence of 39% and primary sclerosing cholangitisis is a potentially serious condition, which can often lead to liver failure. Complications of ulcerative colitis include: Severe bleeding happens in up to 10 percent of patients. Massive hemorrhage occurs in up to 3 percent of patients [16,17]. Urgent colectomy may be needed in these cases [18]. Another complication is fulminant colitis with more than 10 stools per day, bleeding, abdominal pain,and distension. Patients with fulminant colitis are at high risk of developing toxic megacolon. Perforation of the colon most commonly occurs as a consequence of toxic megacolon and requires surgical intervention [16].

Treatment options in ulcerative colitis are individualized based on the severity of symptoms of the disease. Patients with mild clinical disease are considered those who have ≤4 stools per day with or without small amounts of blood, no signs of systemic toxicity and a normal C-reactive protein (CRP) and/or erythrocyte sedimentation rate (ESR). Patients with moderate clinical disease may have frequent (four to six per day) loose, bloody stools, mild anemia, and abdominal pain that is not severe. Patients have no or minimal signs of systemic toxicity. Patients with a severe clinical disease typically have frequent loose bloody stools (≥6 per day) with severe cramps and evidence of systemic toxicity as demonstrated by a fever (temperature ≥37.8°C), tachycardia (heart rate ≥90 beats per minute), anemia (hemoglobin <10.0 g/dL), and/or an elevated CRP or ESR. For patients with mild disease, mesalazine is the drug of choice in inducing remission and preventing relapse [19-23]. For patients with severe disease, the use of glucocorticosteroids is necessery in inducing remission. For patients with steroid-resistant disease, monoclonal antibodies such as infliximab, adalimumab, and golimumab are used [24,25].

Generally, anal and colon lesions can occur due to infectious and neoplastic etiology, and a prompt and multidisciplinary approach may prevent poor outcomes [26]. Patients with ulcerative colitis are at increased risk for colorectal cancer. The risk appears to be highest in patients with pancolitis and begins to increase 8 to 10 years following the onset of symptoms in patients with pancolitis. In one prospective study, the incidence of colorectal cancer was 2.5 percent after 20 years and 7.6 percent after 30 years of disease [27].

Surgery as an Option

Indications for operation in Crohn's disease are failure of medical management (most common reason), extra-intestinal manifestations (25%), intestinal obstruction, fistulas with associated abscess or stricture, perforation, bleeding and cancer. Resection is the most commonly performed surgical procedure for small bowel Crohn's disease. Other surgical options for the treatment of small bowel Crohn's disease include bypass operations or ileostomy formation. These procedures can also be performed laparoscopically with decreased morbidity and length of stay in the hospital [28,29]. Laparoscopic minimally invasive surgery should be preferred to open surgery due to its advantages [30].

Indications for operation in ulcerative colitis are failure of medical management (most common reason), risk of malignancy and severe extra-intestinal manifestations of ulcerative colitis.

Worsening signs and symptoms of colitis, including numerous bloody stools per day, fever, elevated heart rate, anemia, elevated sedimentation rate, radiographic evidence of colonic distension, and abdominal distension with tenderness on exam are indications for an emergent surgery. Total proctocolectomy with end ileostomy remains the operative standard against which all other resections for ulcerative colitis are compared. Surgical options of ulcerative colitis in the emergent setting is aimed toward removing the inflamed bowel while minimizing morbidity, and a total abdominal colectomy and end ileostomy is the procedure of choice [28,29]

Conflict of Interest

There are no conflicts of interest to declare.

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References

1. Lovasz BD, Golovics PA, Vegh Z, Lakatos PL. New trends in inflammatory bowel disease epidemiology and disease course in Eastern Europe. Dig Liver Dis. 2013;45(4):269-76.
2. Chandrakumar A, Loeppky R, Deneau M, El-Matary W. Inflammatory Bowel Disease in Children with Elevated Serum Gamma Glutamyltransferase Levels. J Pediatr. 2019;215:144.
3. Shen B. Managing medical complications and recurrence after surgery for Crohn's disease. Curr Gastroenterol Rep. 2008;10(6):606-11.
4. Deneau M, Jensen MK, Holmen J, et al. Primary sclerosing cholangitis, autoimmune hepatitis, and overlap in Utah children: epidemiology and natural history. Hepatology. 2013;58:1392.
5. Bjarnason I. The Use of Fecal Calprotectin in Inflammatory Bowel Disease. Gastroenterol Hepatol (NY). 2017;13(1):53-56.
6. Pavlidis P, Gulati S, Dubois P, et al. Early change in faecal calprotectin predicts primary non-response to anti-TNFα therapy in Crohn's disease. Scand J Gastroenterol. 2016;51(12):1447-1452.
7. Lim WC, Hanauer S. Aminosalicylates for induction of remission or response in Crohn's disease. Cochrane Database Syst Rev. 2010;8:CD008870.
8. Ford AC, Bernstein CN, Khan KJ, et al. Glucocorticosteroid therapy in inflammatory bowel disease: systematic review and meta-analysis. Am J Gastroenterol. 2011;106(4):590-9.
9. Ford AC, Sandborn WJ, Khan KJ, Hanauer SB, Talley NJ, Moayyedi P. Efficacy of biological therapies in inflammatory bowel disease: systematic review and meta-analysis. Am J Gastroenterol. 2011;106(4):644-59.
10. Louis E, Mary JY, Vernier-Massouille G, et al. Maintenance of remission among patients with Crohn's disease on antimetabolite therapy after infliximab therapy is stopped. Gastroenterology. 2012:142(1):63-70.e5;quiz e31.
11. Lichtenstein GR, Loftus EV, Isaacs KL, Regueiro MD, Gerson LB, Sands BE. ACG clinical guideline: management of Crohn's disease in adults. Am J Gastroenterol. 2018;113(4):481-517.
12. Terdiman JP, Gruss CB, Heidelbaugh JJ, et al. American Gastroenterological Association Institute guideline on the use of thiopurines, methotrexate, and anti-TNF-α biologic drugs for the induction and maintenance of remission in inflammatory Crohn's disease. Gastroenterology. 2013;145:1459.
13. Farmer RG, Whelan G, Fazio VW. Long-term follow-up of patients with Crohn's disease. Relationship between the clinical pattern and prognosis. Gastroenterology. 1985;88:1818.
14. Solberg IC, Vatn MH, Høie O, et al. Clinical course in Crohn's disease: results of a Norwegian population-based ten-year follow-up study. Clin Gastroenterol Hepatol. 2007;5:1430.
15. Hemminki K, Li X, Sundquist J, Sundquist K. Cancer risks in Crohn disease patients. Ann Oncol. 2009;20:574.
16. Manser CN, Borovicka J, Seibold F, et al. Risk factors for complications in patients with ulcerative colitis. United European Gastroenterol J. 2016;4(2):281-287. doi:10.1177/2050640615627533
17. Lynch WD, Hsu R. Ulcerative Colitis. [Updated 2019 Dec 17]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459282/
18. Theofanis G, Saedon M, Kho SH, Mulita F, Germanos S, Leung E. Avoiding emergency stoma surgery with the use of sugar. Br J Nurs. 2017;26(22):S24-S26. doi:10.12968/bjon.2017.26.22.S24
19. Kornbluth A, Sachar DB. Ulcerative colitis practice guidelines in adults: American College Of Gastroenterology, Practice Parameters Committee. Am J Gastroenterol. 2010;105(3):501-23; quiz 524
20. Bernstein CN, Fried M, Krabshuis JH, et al. World Gastroenterology Organization Practice Guidelines for the diagnosis and management of IBD in 2010. Inflamm Bowel Dis. 2010;16(1):112-24.
21. McNamara D. New IBD guidelines aim to simplify care. Medscape Medical News. February 20, 2018.
22. Ford AC, Achkar JP, Khan KJ, et al. Efficacy of 5-aminosalicylates in ulcerative colitis: systematic review and meta-analysis. Am J Gastroenterol. 2011;106(4):601-16.
23. Lichtenstein GR, Ramsey D, Rubin DT. Randomised clinical trial: delayed-release oral mesalazine 4.8 g/day vs. 2.4 g/day in endoscopic mucosal healing--ASCEND I and II combined analysis. Aliment Pharmacol Ther. 2011;33(6):672-8.
24. Ford AC, Sandborn WJ, Khan KJ, et al. Efficacy of biological therapies in inflammatory bowel disease: systematic review and meta-analysis. Am J Gastroenterol. 2011;106(4):644-59. quiz 660.
25. Leblanc S, Allez M, Seksik P, et al. Successive treatment with cyclosporine and infliximab in steroid-refractory ulcerative colitis. Am J Gastroenterol. 2011;106(4):771-7.
26. Mulita F, Tavlas P, Iliopoulos E, Maroulis I. Giant anal warts. Clin Case Rep. 2020;00:1-2. https://doi.org/10.1002/ccr3.3021
27. Rutter MD, Saunders BP, Wilkinson KH, et al. Thirty-year analysis of a colonoscopic surveillance program for neoplasia in ulcerative colitis. Gastroenterology. 2006;130:1030.
28. Hwang JM, Varma MG. Surgery for inflammatory bowel disease. World J Gastroenterol. 2008;14(17):2678-2690. doi:10.3748/wjg.14.2678
29. Sica GS, Biancone L. Surgery for inflammatory bowel disease in the era of laparoscopy. World J Gastroenterol. 2013;19(16):2445-2448. doi:10.3748/wjg.v19.i16.2445
30. Francesk Mulita et al. Laparoscopic removal of an ingested fish bone from the head of the pancreas: case report and review of literature. Pan African Medical Journal. 2020;36:123. [doi:10.11604/pamj.2020.36.123.23948]