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EDITORIAL

Intestinal Behçet's disease: A review of clinical diagnosis and treatment

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Abstract

Behçet's disease (BD) is a chronic inflammatory disorder prone to frequent recurrences, with a high predilection for intestinal involvement. However, the efficacy and long-term effects of surgical treatment for intestinal BD are unknown. In the current issue of *World J Gastrointest Surg*, Park *et al* conducted a retrospective analysis of 31 patients with intestinal BD who received surgical treatment. They found that elevated C-reactive protein levels and emergency surgery were poor prognostic factors for postoperative recurrence, emphasizing the adverse impact of severe inflammation on the prognosis of patients with intestinal BD. This work has clinical significance for evaluating the postoperative condition of intestinal BD. The editorial attempts to summarize the clinical diagnosis and treatment of intestinal BD, focusing on the impact of adverse factors on surgical outcomes. We hope this review will facilitate more precise postoperative management of patients with intestinal BD by clinicians.

Key Words: Intestinal Behçet's disease; Diagnosis; Treatment; Surgery; Recurrence

Core Tip: Intestinal Behçet's disease (BD) is the gastrointestinal manifestation of BD, characterized primarily by intestinal ulcerations. The differential diagnosis of intestinal BD is challenging, the disease course is prolonged, and surgical intervention is often necessary to achieve cure or remission. Despite surgical treatment, postoperative recurrence and reoperation rates remain high. Understanding the timing of surgery and factors associated with postoperative recurrence is critical for standardized surgical and follow-up management of patients with intestinal BD.

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INTRODUCTION

Behçet's disease (BD) is a chronically relapsing multisystem inflammatory disorder primarily characterized by vasculitis [1], with an etiology remaining elusive. Genetic polymorphisms, encompassing IL23R/IL12RB2, ERAP1, and HLA-B51, are postulated as significant contributors to the dysregulation of inflammatory responses in BD pathogenesis[2-5]. Additionally, microbial factors such as herpes simplex virus infection[2,6], increased colonization of Streptococcus mutans [7], and intestinal microbial dysbiosis[8] provide clues to its underlying pathomechanisms. BD can involve multiple organs or systems, including recurrent oral and genital ulcerations, intestinal involvement, arthritis, ocular manifestations, skin lesions, as well as vascular and neural involvement[1,9]. The distribution of BD in the population varies widely (0.64-420/100000)[10], with a predilection for countries along the "ancient Silk Road" route, stretching from the Mediterranean, Middle East, to East Asia[11]. Prevalence is lower in Nordic and North American regions[12]. Notably, the diversity of intestinal involvement in BD seems more common in East Asian countries, including South Korea and Japan[13].

Intestinal BD, a significant subtype of BD, exhibits two distinct forms of intestinal pathology. One manifests as mucosal inflammation and ulceration triggered by neutrophilic venulitis, while the other involves ischemic injury caused by vasculitis[14]. Characterized by diverse, fluctuating courses, repeated remissions and relapses[15], intestinal BD can affect any part of the gastrointestinal tract[16] and potentially involve extraintestinal organs such as the liver[17], pancreas[18], or spleen[19]. Notably, the onset of intestinal BD symptoms typically lags behind those of extragastrointestinal manifestations[20]. Approximately 5-10% of patients with systemic BD develop intestinal BD approximately 7.35 years after initial disease onset[13], and approximately 75% patients require hospitalization[21].

Due to unsatisfactory control of disease symptoms with empirical medication, intestinal BD can lead to complications, such as anemia[22], and even increased risk of hematologic malignancies[23]. Severe cases can cause intestinal perforation or massive gastrointestinal bleeding, necessitating intestinal resection. Notably, 30.5% of patients with intestinal BD are more likely to require emergency surgical intervention[24]. Postsurgical recurrence is common, with the possibility of requiring second or multiple surgeries. Park *et al*[25] argue in "Short- and long-term outcomes of surgical treatment in patients with intestinal Behcet's disease" that significant inflammation may be a key component in postsurgical recurrence of intestinal BD. Therefore, exploring the clinical course following abdominal surgery and identifying predictors of clinical outcomes are essential for deepening understanding of intestinal BD and individualizing treatment approaches.

DIAGNOSIS OF INTESTINAL BD

Intestinal BD is characterized by well-demarcated deep ulcers with smooth surrounding mucosa, most commonly affecting the ileocecal region[26]. Clinical symptoms include nausea, vomiting, abdominal pain, diarrhea, gastrointestinal bleeding, and perforation[27]. The systemic and intestinal symptoms and genetic origins of intestinal BD are remarkably similar to those of other inflammatory intestinal disorders, such as Crohn's disease (CD) and intestinal tuberculosis, making differential diagnosis difficult[28,29]. Approximately half of patients with intestinal BD are misdiagnosed as having CD[30].

Endoscopy[31,32] and capsule endoscopy[33] are the best and most widely used methods for diagnosing and assessing the progression of inflammatory intestinal diseases. These procedures allow for the observation of ulcer size and location [34], as well as mucosal healing[35], which aid in evaluating the severity and prognosis of intestinal BD. Additionally, endoscopy can facilitate the development of scoring systems to predict the clinical course after surgical resection of intestinal BD[36]. Endoscopic and histologic examinations are also effective in distinguishing intestinal BD from CD[29, 30]. Currently, colonoscopy is not an accurate predictor of the Disease Activity Index for Intestinal BD (DAIBD) scores. However, volcanic ulcers and ulcer counts obtained through colonoscopy are independent predictors of DAIBD[15]. Therefore, endoscopy remains crucial in diagnosing intestinal BD and assessing disease activity indices.

Computed tomography enterography is a valuable imaging modality that can enhance clinical differentiation between intestinal BD and CD by assessing the characteristics of intestinal images and analyzing body composition[37,38]. However, some studies raised concerns that computed tomography (CT) may expose intestinal BD to ionizing radiation, potentially causing damage and exacerbating inflammatory injury[39]. Therefore, a more cautious approach to CT examination management is necessary for patients with intestinal BD.

In clinical practice, assessing intestinal BD activity is critical for selecting appropriate treatment regimens. One widely used and relatively simple scoring system is the DAIBD[40]. The DAIBD incorporates eight parameters: Overall general health, fever, extraintestinal manifestations, abdominal pain, abdominal mass, abdominal tenderness, intestinal complications, and frequency of loose bowel movements within a week. The total score is calculated by adding the inactive (≤ 19), mild (20-39), moderate (40-74), and severe (≥ 75) categories[40]. However, additional markers or models, such as biodiversity indices[41] and endoscopic scoring systems[36], are needed to further evaluate and define intestinal BD activity.

Non-invasive tests are more amenable to screening and real-time assessment of diseases, facilitating their widespread application. Hou *et al*[42] discovered that interleukin-6 (> 7 pg/mL), hemoglobin (< 130 g/L), C-reactive protein (CRP) (> 10 mg/L) and erythrocyte sedimentation rate (ESR) (≥ 15 mm/H), suggest the presence of intestinal symptoms in patients with BD. Lee *et al*[43] also reported proteomic findings indicating that high serum amyloid A implies intestinal involvement in patients with BD. Elevated inflammatory markers can indicate the occurrence of intestinal BD[44]. Notably, CRP and ESR levels are significantly higher in patients with severe intestinal BD compared to those with mild disease [45], and serum procalcitonin levels have unique advantages in assessing the severity of intestinal BD infection[46]. In individuals with intestinal BD, albumin levels are also lowered due to increasing disease activity[45]. Additionally, the exploration of novel markers for intestinal BD exhibits tremendous potential, offering unique advantages in diagnosis. The soluble triggering receptor expressed on myeloid cells-1[47], fecal calprotectin[48,49], fecal lactoferrin[50], anti-alpha-enolase antibody[51], and lipoprotein-associated phospholipase A2[52] may emerge as diagnostic and activity monitoring markers for intestinal BD. Furthermore, anti-Saccharomyces cerevisiae antibody was higher in 44.3% of patients with intestinal BD, and positively correlated with surgical rates[53]. Moreover, the detection of heat shock protein family A member 6[54] and maltase-glucoamylase[55] can assist in distinguishing intestinal BD from CD.

MEDICAL TREATMENT OF INTESTINAL BD

Currently, the conventional treatment for intestinal BD remains empirical, with aminosalicylates recommended for mild cases[56]. However, a significant proportion of patients, particularly those who are young (< 35 years) or have high levels of CRP or a high intestinal BD score, exhibit poor response to these medications[57]. For moderate to severe cases, glucocorticoids and immunomodulators such as thalidomide, thiopurine, cyclophosphamide, and methotrexate, used alone or in combination, are necessary[2]. Nevertheless, there remains a risk of disease recurrence, exacerbation of gastrointestinal bleeding, venous thrombosis, and infection[58,59]. Tumour necrosis factor-α (TNF-α) inhibitors, such as infliximab and adalimumab, are often used to treat severe and refractory cases of intestinal BD[60,61]. Patients with intestinal BD localized outside the ileocecal region may also have a greater need for anti-TNF-α immunotherapy[56]. Those who respond poorly to anti-TNF-α therapy have a higher likelihood of undergoing surgical intervention[60], and combination therapy with thalidomide has been suggested[62]. The future holds promise for the discovery and development of additional biologic agents, such as baricitinib[63] and calcineurin inhibitors[64], as novel therapeutic options for severe and refractory intestinal BD[27].

SURGICAL TREATMENT OF INTESTINAL BD

Surgical intervention is often necessary in patients with intestinal BD who present with medically refractory disease, fistula or abscess formation, intestinal obstruction, or abdominal mass. Emergency surgery is required for intestinal perforation and severe gastrointestinal bleeding. The cumulative rate of surgical intervention in patients with intestinal BD is not low: 20% after one year, 27%-33% at five years, and 31%-46% at ten years following diagnosis[60]. Studies have shown that the progression of intestinal BD to more extensive ocular and ileal disease is an important indicator for needing surgery treatment[65]. Findings suggested history of appendectomy and high DAIBD score at diagnosis increase likelihood of intestinal surgery[21]. However, there is no difference in short-term outcomes between laparoscopic and open surgery for intestinal BD patients[66].

Postoperative complications, including anastomotic leak, abscess or fistula formation, wound infection, intestinal obstruction, bleeding, and perforation, can significantly impact recovery of patients with intestinal BD. Elevated CRP levels immediately postoperatively significantly increase risk of postoperative complications[67]. Combined use of glucocorticoids and immunosuppressants postoperatively can significantly reduce incidence of postoperative complications [68].

PROGNOSIS OF SURGICAL INTERVENTION

Postoperative recurrence is a substantial contributor to the ongoing non-healing of intestinal BD. Approximately 13%-75% of patients with intestinal BD experience postoperative recurrence, carrying high risk of disease recurrence (Table 1). Therefore, regular post-surgery follow-up is strongly recommended[36]. The endoscopic-intestinal BD scoring system, utilizing parameters such as number and size of ulcers, can aid disease recurrence assessment[36]. As shown in Table 1, various factors influence intestinal BD prognosis post-surgery. Among these, elevated CRP levels identified by Kang *et al* [67] and Jung *et al*[69], and emergency surgery identified by Park *et al*[70], are poor postoperative prognostic factors in intestinal BD. These findings are consistent with the results of the current study by Park *et al*[25].

Table 1 Surgical prognosis of intestinal Behçet's disease

Cases	Recurrence rate after surgery (%)	Factors associated with recurrence	Ref.
31	20.5	Preoperative increased CRP levels; Emergency surgery	Park *et al*[25]
54	68.5	Colonoscopy; Colonoscopic recurrence	Park *et al*[36]
8	75	Increased peripheral CD8+DR+ lymphocytes (%)	Naganuma *et al*[65]
90	57.8	Higher CRP level immediately after surgery	Kang *et al*[67]
72	58.3	Volcano-type ulcerations; Increased CRP levels; Intestinal perforations	Jung *et al*[69]
90	45.6	Initial emergency operation; Higher initial perioperative erythrocyte sedimentation rate	Park *et al*[70]
16 (Complete remission of intestinal lesions)	13	Incomplete remission of intestinal lesions; the history of intestinal perforation or fistula; did not take azathioprine	Choi *et al*[72]
27 (Incomplete remission of intestinal lesions)	43		
50 (5-ASA)	66.0	Thiopurine was not used postoperatively	Lee *et al*[73]
27 (Thiopurine)	37.0		
33 (Intestinal perforations)	42.4	NA	Moon *et al*[76]
40 (Early surgery)	35.0	Late surgery	Jung *et al*[77]
62 (Late surgery)	45.2		
9	55.6	Intraoperative endoscopy	Iida *et al*[78]
91	51.1	NA	Jung *et al*[79]
16	50.0	Volcano-type ulcerations	Kim *et al*[80]
91	35.2	Postoperative use of steroids; postoperative complications; high BMI	Baek *et al*[81]

CRP: C-reactive protein; 5-ASA: 5-aminosalicylic acid; BMI: Body mass index; NA: Not available.

In addition to identifying factors associated with postoperative recurrence, it is crucial to consider factors such as disease duration[45] to assess disease activity and severity. Postoperative systemic pharmacological treatment, particularly the use of immunosuppressants[71], is critical. Regular administration of azathioprine postoperatively also can result in partial or complete resolution of intestinal symptoms and, to some extent, reduce the recurrence rate of intestinal BD[72], outperforming the use of 5-aminosalicylic acid alone[73].

Especially in younger patients, greater attention should be paid to disease progression, as multiple clinical evidences suggested that they often exhibit more severe clinical symptoms and poorer prognoses[74-76]. Patients with severe courses tend to be younger than those with mild courses[45], resulting in higher demand for outpatient, inpatient treatment, and intestinal surgery[75]. They are also more prone to postoperative recurrence[76]. Therefore, it is essential to develop targeted treatment strategies for younger patients with intestinal BD, and to initiate immunosuppressant therapy early in the course of the disease[75].

The timing of surgery is worth discussing. Compared to patients with advanced BD undergoing surgery, those with intestinal BD receiving early intestinal surgery have better prognosis, with significantly reduced postoperative clinical recurrence and reoperation rates[77]. Anti-TNF-α medication can effectively treat refractory intestinal BD. If patients with intestinal BD get anti-TNF-α therapy and experience poor response (within one month), ESR > 42.5 mm/H, skin and joint symptoms, or geographic ulcers, surgical intervention may be required[60]. Therefore, it is crucial to individualize treatment plans based on risk factors, deciding whether to prioritize pharmacological therapy or early surgery as the primary treatment goal.

CONCLUSION

The pathogenesis of intestinal BD remains unclear, lacking radical treatment and often requiring surgical intervention[25, 76-81]. In a retrospective clinical analysis spanning nearly 11 years, Park *et al*[25]. found that the postoperative recurrence rate among patients with intestinal BD was 20.5%. Severe inflammatory variables, notably emergency surgery and increased CRP levels, were found as the main predictors of postoperative recurrence or reoperation. This work by Park *et al*[25] can assist clinicians in better assessing the surgical prognosis of intestinal BD and developing more precise, individualized treatment plans.

However, numerous areas require further improvement in future studies. Future research should examine age, inflammatory status, and drug treatment impacts on surgical outcomes, postoperative complications, and recurrence. Additionally, multicenter collaborations should further explore and refine prognostic risk factor assessment for intestinal BD through retrospective and prospective studies. Elevated CRP levels associate with surgical recurrence at various time points[67,69], so real-time CRP inflammatory marker monitoring before, during, and after surgery may aid real-time management of intestinal BD. These efforts could enable risk-stratified, tailored drug therapies and personalized patient monitoring.

FOOTNOTES

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