Small bowel vasculitis? what a gastroenterologist should know – from diagnosis to management

João Carlos Gonçalves[a,b,c], *Bruno Rosa*[a,b,c] *and José Cotter*[a,b,c]

Purpose of review
This article provides gastroenterologists with an overview of small bowel involvement in systemic vasculitis. Though various vasculitides can impact the small bowel, we highlight those with a more frequent and clinically significant GI involvement.

Recent findings
Recent advances, including increased accessibility to cross-sectional imaging, capsule endoscopy and device-assisted enteroscopy, have improved detection of gastrointestinal manifestations in systemic vasculitis. Studies have also explored the genetic and inflammatory pathways involved in these diseases, although high-quality evidence on diagnosis and treatment remains limited, leading to reliance on expert consensus.

Summary
Small bowel involvement is common in Behçet's disease and small vessel vasculitis, presenting with symptoms ranging from mild to severe, including massive bleeding, ischemia, and perforation, often indicating a poorer prognosis. Diagnosis is challenging, but in patients with a known or suspected history of vasculitis, it should prompt contrast-enhanced abdominal imaging and endoscopic evaluation. Treatment decisions should be made collaboratively by a multidisciplinary team, with immunosuppressive therapy remaining the cornerstone.

Keywords
Behçet's disease, small bowel, vasculitis

INTRODUCTION

Vasculitis is characterized by inflammation and damage to blood vessels, with clinical presentations varying based on the vascular bed affected. Gastrointestinal involvement occurs as part of a broader systemic disease, and symptoms are often nonspecific. In the small bowel, injury can occur through two main mechanisms, the vascular damage to medium-sized vessels, which may lead to ischemia and transmural necrosis, or mucosal and/or submucosal damage in cases involving small vessel vasculitis. In patients with known or suspected vasculitis, clinicians should have a lower threshold to perform a dedicated gastrointestinal diagnostic work-up, aiming to prevent severe complications, such as bowel perforation or intestinal ischemia.

EPIDEMIOLOGY AND ETIOLOGY

According to the 2012 Chapel Hill Consensus Conference nomenclature [1] vasculitides can be categorized by the vessel size that is predominantly affected. As such, they can be classified into large vessel vasculitis, medium vessel vasculitis, and small vessel vasculitis. Additionally, some forms, such as variable vessel vasculitis, can affect vessels of any size, or may occur secondary to systemic autoimmune diseases or infections. The conditions that involve the gastrointestinal system more frequently are small vessel vasculitis, such as the immunoglobulin A (IgA) and antineutrophilic cytoplasmic antibodies (ANCA) associated vasculitis, Polyarteritis Nodosa (PAN) that causes inflammation of the medium-sized vessels and Behçet's disease (BD) that causes variable vessel vasculitis. In Table 1, we list

[a]Gastroenterology Department, Unidade Local de Saúde do Alto Ave, Guimarães, [b]Life and Health Sciences Research Institute (ICVS), School of Medicine, University of Minho, Braga and [c]ICVS/3B's - PT Government Associate Laboratory, Braga/Guimarães, Portugal

Correspondence to Bruno Rosa, Department of Gastroenterology, Unidade Local de Saúde do Alto Ave, Rua dos Cutileiros, Creixomil, Guimarães 4835-044, Portugal. Tel: +351 253 540 330; e-mail: brunorosa@ulsaave.min-saude.pt

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KEY POINTS

- All vasculitides can lead to gastrointestinal injury, with Behçet's disease and small vessel vasculitis being the most prevalent.
- The diagnostic workup relies on cross-sectional imaging and endoscopy, with a low threshold for these investigations in patients with a known history of vasculitis.
- Behçet's disease and Crohn's disease may present with similar clinical features.
- Treatment requires immunosuppression, however, given the complexity and multisystemic nature of vasculitis, a multidisciplinary approach is recommended.

the systemic vasculitis with a more frequent involvement of the small bowel by vessel size.

Behçet's disease

BD is a chronic, relapsing, multisystemic vasculitis that can affect any vessel type of any size, primarily presenting with recurrent oral and/or genital ulcers, uveitis, and characteristic skin lesions. The causal mechanisms are not fully understood, but a complex association with genetic predisposition due to specific HLA genes, vascular endothelial hyper-activation and dysregulated immune response has been studied [2]. It is most prevalent in countries along the historic Silk Road, including the Mediterranean region, the Middle East, and East Asia, with lower prevalence in North America and Northern Europe. Gastrointestinal involvement in BD is a major concern due to its association with significant morbidity and mortality [2,3].

Table 1. Classification of systemic vasculitides by vessel size that may involve the small bowel

Vasculitides with predilection for small bowel involvement	
Variable vessel vasculitis	Behçet's disease
Large vessel vasculitis	Takayasu arteritis
Medium-vessel vasculitis	Polyarteritis nodosa
Small-vessel vasculitis	ANCA-associated: • Microscopic polyangiitis • Granulomatosis with polyangiitis • Eosinophilic granulomatosis with polyangiitis Immune complex-mediated: • IgA vasculitis
Vasculitis associated with systemic disease	Lupus vasculitis Rheumatoid vasculitis

There are two mechanisms for small bowel injury, neutrophilic infiltration that leads to mucosal inflammation and medium-vessel involvement that causes intestinal ischemia and infarction. There is also a geographical heterogeneity in the gastrointestinal involvement, as it is less common in Turkey and Saudi Arabia, where BD is prevalent, but more frequent in Japan and Europe [3]. Diagnosing BD remains challenging due to the absence of pathognomonic laboratory tests and the overlap of symptoms with other inflammatory diseases, such as Crohn's disease (CD).

Large vessel vasculitis: Takayasu and giant cell arteritis

Takayasu arteritis is an idiopathic large vessel vasculitis that usually affects young Asian females characterized by inflammation of the aorta and its primary branches. Gastrointestinal involvement is rare although occlusion of the large or medium mesenteric arteries can occur, resulting in ischemia of the small bowel [4]. Interestingly, co-occurrence of inflammatory bowel disease in Takayasu arteritis has been observed, although the reasons for such association remain unclear [5].

Giant cell arteritis involves the aorta and its cranial arteries, rarely involving other sites. The elderly are predominantly affected, and small bowel involvement is generally due to atherosclerosis.

Medium-vessel vasculitis: polyarteritis nodosa and Kawasaki disease

PAN is a type of medium-vessel vasculitis in which gastrointestinal involvement, especially of the small bowel, is relatively common and associated with a poorer prognosis [6]. Typically, patients are in their 40s or 50s and display a broad clinical syndrome involving multiple systems, including cutaneous lesions, renal injury, hypertension, peripheral neuropathy, as well as testicular and muscle damage. Hepatitis B virus (HBV) infection is a known risk factor for PAN, yet now it accounts for fewer than 10% of cases due to vaccination and widespread treatment [7]. When HBV-associated PAN does occur, it typically manifests within the first 12 months postinfection, coinciding with the presence of circulating antigen-antibody immune complexes [8]. Other etiologies were found to be the trigger of PAN including viral infections, drugs, solid and hematological malignancies and autoimmune diseases. Importantly, genetic syndromes that augment inflammatory response have been found to cause PAN, and this has led to an elucidation of the pathophysiological mechanisms involved in the disease [7].

Kawasaki disease is a vasculitis of unknown origin of small and medium caliber blood vessels, especially involving coronary arteries, affecting children under 5 years of age. Gastrointestinal involvement is rare, although there are some reports of small bowel ischemia with need for surgical resection [9].

Small-vessel vasculitis: antineutrophilic cytoplasmic antibodies-associated

ANCA-associated vasculitis predominantly affects small vessels with minimal or no immune deposits in the vessel walls, a characteristic termed 'pauci-immune'. This condition is typically associated with circulating ANCA antibodies and includes three primary subtypes, namely microscopic polyangiitis (MPA), granulomatosis with polyangiitis (GPA), and eosinophilic granulomatosis with polyangiitis (EGPA). While ANCA-associated vasculitis primarily involves the kidneys and lungs, gastrointestinal involvement can occasionally occur, particularly in EGPA.

Small-vessel vasculitis: immune complex-mediated

The IgA vasculitis, formerly known as Henoch–Schönlein purpura, strongly affects the small bowel. Despite being the most common vasculitis in children, around 10% of cases occur in adults. It is believed to arise from immune complex formation and deposition, leading to leukocytoclastic vasculitis in small vessels. An association with a recent upper respiratory infection is observed in about half of the cases [10,11].

Vasculitis associated with systemic disease

Systemic diseases such as systemic lupus erythematosus, long-standing Rheumatoid Arthritis or non-treated human immunodeficiency viruses (HIV) can present secondary vasculitis, which can affect the gastrointestinal tract. When overt abdominal symptoms are present small bowel involvement should be suspected and investigated.

CLINICAL PRESENTATION

A diagnosis of vasculitis should be considered in patients with constitutional symptoms, such as fever, malaise and weight loss in combination with evidence of single and/or multiorgan dysfunction. Small bowel vasculitis presents variably, often with overlapping gastrointestinal symptoms that mimic other conditions, such as intermittent or constant abdominal pain due to ischemia or inflammation, gastrointestinal bleeding manifesting as hematemesis or melena, diarrhea and weight loss linked to malabsorption or reduced appetite. In the adequate clinical scenario, i.e. in patients with known or suspected vasculitis with overt gastrointestinal symptoms, a dedicated diagnostic workup should be performed.

Intestinal BD lacks specific symptoms, but it should be suspected in patients presenting with abdominal pain, melena or hematochezia, abdominal mass, diarrhea, and weight loss [12]. BD can involve any part of the gastrointestinal tract, with symptoms often appearing several years after initial manifestations, particularly oral ulcers. The ileocecal region is the most affected, with the majority of patients presenting with one or a few ulcers that are typically large, round or oval, and deep. These ulcer features significantly increase the risk of complications, such as perforation, often requiring emergency surgery [13,14].

The hallmark lesions of PAN are microaneurysms, which are frequently observed in hepatic, splenic, mesenteric, and renal arteries [15]. Therefore, abdominal pain is a prominent symptom in most PAN patients, often resembling intestinal angina. This pain is likely due to transmural necrotizing inflammation of the mesenteric vessels, leading to bowel ischemia, most commonly affecting the small bowel [16]. In a retrospective study, approximately one-third of PAN patients with gastrointestinal involvement developed acute abdominal symptoms due to bowel perforation [17]. Other manifestations in PAN may include nausea, vomiting, diarrhea, hematochezia, and melena.

In MPA and GPA, gastrointestinal involvement is relatively uncommon [18]. In a retrospective Swedish study, only 6.5% of patients reported symptoms such as abdominal pain and gastrointestinal bleeding [19]. Endoscopic evaluation for suspected gastrointestinal hemorrhage may reveal areas with redness and swelling due to vasculitis but may not always identify a clear source of bleeding.

In EGPA manifestations such as abdominal pain, gastrointestinal bleeding, nausea, vomiting, diarrhea and constipation are common and are associated with a worse prognosis [20]. A Japanese multicentric retrospective study examining 19 EGPA patients reported that 42.1% had gastrointestinal involvement and most of these patients underwent extensive endoscopic evaluation, including capsule endoscopy and/or ballon assisted enteroscopy [21]. Interestingly, the small bowel was the most affected organ, with findings such as mucosal erythema, erosions, and ulcers reported in the enteroscopy procedures. Histopathological findings in EGPA include

eosinophilic infiltration, small-vessel vasculitis, and extravascular granulomas, though these features are detected less frequently in endoscopic biopsies (up to 30%) compared to skin tissue samples.

The primary clinical manifestations of IgA vasculitis include a palpable purpura, which typically precedes other symptoms, arthritis, colicky abdominal pain, and renal impairment. Gastrointestinal involvement is seen in approximately half of patients, usually presenting after the onset of the rash. The symptoms can range from mild, such as nausea, vomiting, and mild abdominal pain, to severe complications including gastrointestinal hemorrhage, bowel ischemia, necrosis with perforation, and intussusception [22].

The abdominal pain in IgA vasculitis is primarily due to submucosal hemorrhage and edema, with purpuric lesions often visible during endoscopy. A Japanese multicenter retrospective study of 33 adult patients with IgA vasculitis showed that all had skin and gastrointestinal involvement, with lesions identified throughout the digestive tract, particularly in the small intestine [21]. These findings included erythema, erosions, ulcers, and hematomas. However, histopathological alterations in endoscopic biopsies were detected in only 17.1% of cases, compared to 83.9% in those of the skin. The diagnosis of IgA vasculitis in adults can be challenging, especially if the gastrointestinal symptoms precede the typical purpuric rash [23].

DIAGNOSTIC APPROACH

The median time for reaching a diagnosis of systemic vasculitis is approximately seven months, with significant variability among subtypes [24]. Therefore, a detailed physical examination is warranted as well as laboratory tests, imaging studies, and endoscopic evaluation. Physical examination may reveal abdominal tenderness and guarding, particularly in severe cases, suggesting ischemia or perforation. Extra-intestinal manifestations can hint for a systemic disease, therefore a detailed skin, cardiovascular and neurological examination are mandatory.

Laboratory testing should include a complete blood count, inflammatory markers such as C-reactive protein and erythrocyte sedimentation rate, a minimal biochemical panel with liver and renal function, serum cryoglobulins, as well as screen for viral hepatitis and HIV. A urine analysis should be performed to investigate the occurrence of glomerulonephritis. Blood cultures are necessary in the setting of fever to exclude bacteremia. Additional tests should include stool analysis for pathogenic bacteria, viruses, and parasites, especially in cases where gastrointestinal infection is a consideration.

Investigation of autoimmunity, guided by the clinical presentation, can further assist in establishing a diagnosis. A positive antinuclear antibody (ANA) test may indicate systemic lupus erythematosus, particularly when accompanied by low serum complement levels. Specific autoantibodies, such as ANCA, are positive in approximately 90% patients with GPA and MPA, though the positivity rate is lower in EGPA, ranging from 30% to 50% [25]. ANCA targets include either proteinase 3 or myeloperoxidase, and this distinction is diagnostically significant, as antibodies against the former are more associated with GPA, while antibodies against the latter are more frequent in MPA.

Abdominal CT angiography is useful in evaluating suspected small bowel BD and PAN, as it can reveal abnormalities in the mesenteric arteries [26,27]. When gastrointestinal symptoms are present, a comprehensive endoscopic examination is recommended, including upper and lower gastrointestinal endoscopy and capsule enteroscopy. This approach aids in identifying mucosal alterations that can support the diagnosis, such as mucosal hematomas typical of IgA vasculitis or 'volcano'-shaped ulcers characteristic of BD, which tend to be fewer than six in number and localized to discrete regions in the ileocecal area. While endoscopic biopsies can provide valuable information, the sensitivity is low and tissue samples from another organ, such as the skin, muscle and/or nerve, can be useful to increase diagnostic yield [26].

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of systemic vasculitis with associated small bowel involvement is extensive and includes a range of gastrointestinal and systemic conditions that may present with similar symptoms. In this section we will focus on distinguishing between BD and CD, which will be a challenge to the gastroenterologist (Table 2). BD often includes genital ulcers, papulopustular skin lesions, and neurological symptoms, which are uncommon in CD. In contrast, perianal disease (e.g., fistulas and abscesses), bowel strictures, and fistulas are frequently observed in CD but are rare in BD. Endoscopic findings also help differentiate these conditions, as CD typically presents with irregular, longitudinal ulcers and a cobblestone appearance, while BD shows round or oval 'punched-out' ulcers, generally larger than 1 cm, often localized in the ileocecal region. Histologically, CD is marked by noncaseating granulomas, whereas BD is characterized by nonspecific neutrophilic or lymphocytic phlebitis, with no granulomas.

Table 2. Key differences between Crohn's disease and Behçet's disease

	Crohn's disease	Behçet's disease
Perianal disease	• Frequent	• Rare
Skin manifestations	• Erythema nodosum • Pyoderma gangrenosum • Oral ulcers	• Recurrent oral and genital ulcers • Papulopustular skin lesions • Positive pathergy test
Neurological symptoms	• Rare	• Common (neuro-Behçet syndrome)
Endoscopic findings	• Large and irregular ulcers with segmental distribution • Aphtous ulcers • Luminal stenosis and fistulas	• Large volcano-shaped ulcers, few in number • Focal distribution • No aphthous lesions
Histological features	• Noncaseating granulomas	• Neutrophilic infiltration and lymphocyte aggregation surrounding blood vessels

Intestinal tuberculosis is also a concern, especially in endemic areas, causing constitutional symptoms and a ileocecal predominant injury [3]. The correct distinction between small bowel vasculitis as part of a systemic disease from intestinal tuberculosis will impact on treatment strategies and outcomes.

Nonsteroidal anti-inflammatory drug-induced small intestinal ulcers can closely mimic the clinical and endoscopic findings of BD and CD. Therefore, obtaining a detailed history of its use is crucial.

MANAGEMENT STRATEGIES

The treatment of systemic vasculitis with small bowel involvement should be discussed with a multidisciplinary team, as various organs are typically affected. Overall, treatment has the primary goal of remission induction to control active disease and prevent further organ damage, typically using standard to higher doses of glucocorticoids, often combined with an immunosuppressive agent [26]. For PAN associated with viral hepatitis, treatment of underlying disease with antivirals is suggested in the remission phase as well [28]. Once remission is achieved, treatment shifts to maintaining disease control with lower drug doses to reduce toxicity. Continuous monitoring is crucial to assess disease activity, prevent recurrence, and detect any drug-related side effects.

Concerning the management of BD, immunosuppressive drugs used parallel those in CD. The management of small bowel BD generally requires glucocorticoids combined with an immunosuppressive agent to minimize long-term steroid dependence [29]. Prednisone is started at 0.5–1 mg/kg daily, with tapering once symptoms improve. Azathioprine is initiated concurrently, starting at 50 mg/day and titrating up to 2.5 mg/kg/day, with regular blood monitoring due to toxicity risks. If symptoms are not controlled with glucocorticoids and azathioprine, TNF-alpha inhibitors (e.g., infliximab or adalimumab) might be added. Studies show these biologics are effective and safe, especially when combined with azathioprine [30–33]. Sulfasalazine and messalazine are other treatment options for inducing and maintaining remission in mild cases, but lower evidence exists to support their use [3,27]. As with CD, surgery is reserved for severe cases but carries a risk of recurrence [34▪▪], thus continued medical therapy is essential postsurgery. Enteral nutrition should be considered, as it can be effective for induction therapy and is particularly indicated for patients with refractory disease and severe activity [27].

CONCLUSION

Small bowel involvement is possible in nearly every systemic vasculitis, with symptoms that can range from mild to severe, including gastrointestinal bleeding, ischemia, or perforation, often signaling a poorer prognosis. Conditions such as BD, PAN, and small-vessel vasculitis frequently impact the gastrointestinal tract and require a tailored diagnostic approach when symptoms are present. The use of cross-sectional imaging and capsule endoscopy has significantly advanced the detection and understanding of small bowel involvement in systemic vasculitis, uncovering a higher incidence of small bowel injury than previously recognized. Effective management relies on early diagnosis, immunosuppressive therapy, and a multidisciplinary approach due to the systemic complexity of these cases. Figure 1 illustrates the key manifestations of small bowel vasculitis, the diagnostic approach, and general treatment strategies to assist clinicians in identifying these patients effectively.

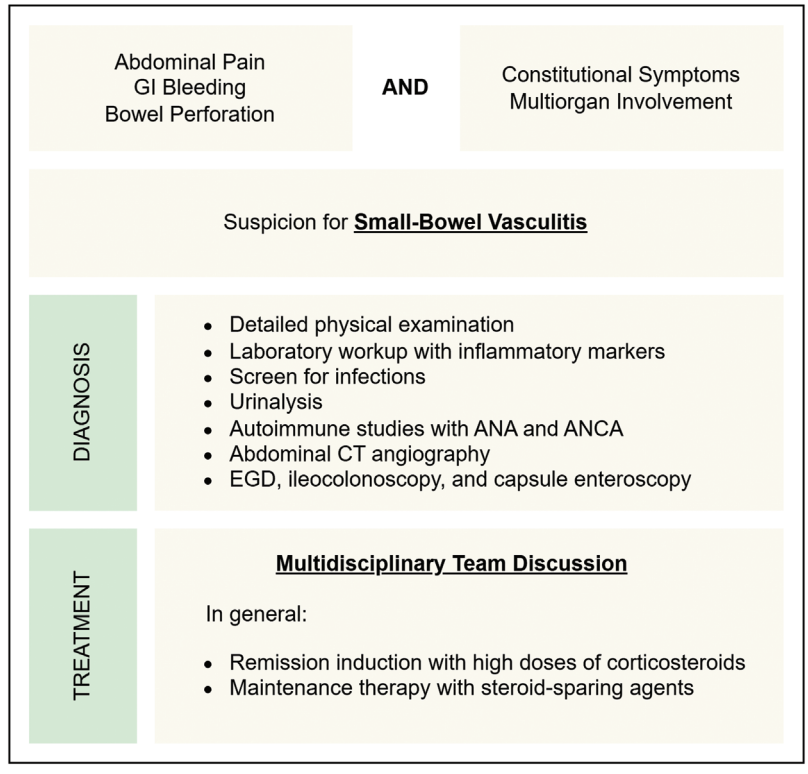

FIGURE 1. Approach to the patient with suspected small bowel vasculitis. EGD, esophagogastroduodenoscopy.

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Conflicts of interest

There are no conflicts of interest.

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Papers of particular interest, published within the annual period of review, have been highlighted as:

- ■ of special interest
- ■■ of outstanding interest

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In this study, the authors investigated the long-term outcomes of surgical treatment for intestinal BD and identify the predictive factors for recurrence and reoperation.