



Coagulopathy and platelet abnormalities in patients with inflammatory bowel disease

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Inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, is associated with a hypercoagulable state that increases thromboembolic complication risk. The interaction between inflammation and coagulation increases risk by upregulating coagulation factors, downregulating natural anticoagulants, and impairing fibrinolysis. Patients with IBD exhibit elevated levels of prothrombotic markers, including thrombin-antithrombin complexes, von Willebrand factor, and tissue factor, reflecting persistent coagulation activation. Furthermore, platelet abnormalities, such as thrombocytosis, enhanced platelet reactivity, and increased platelet-leukocyte aggregates, contribute to the prothrombotic state. Impaired fibrinolysis, characterized by elevated plasminogen activator inhibitor-1 (PAI-1) and decreased urokinase plasminogen activator (uPA) levels, results in reduced clot degradation and prolonged thrombus stability. Endothelial dysfunction and immune-mediated disruptions in anticoagulant pathways also exacerbate coagulation abnormalities. The thromboembolic risk in patients with IBD is influenced by disease activity, hospitalization, immobility, and specific therapeutic agents. Certain treatments, such as JAK inhibitors, increase the risk, whereas anti-TNF agents may offer protective effects. Given the notable impact of coagulation and platelet dysfunction on IBD pathophysiology and patient outcomes, a comprehensive understanding of these abnormalities is essential to optimize clinical management and reduce critical thromboembolic complications.

Keywords: Inflammatory bowel disease; Platelet adhesiveness; Thrombocytosis; Thromboembolism

INTRODUCTION

The interaction between inflammation and coagulation plays an important role in the pathogenesis of various chronic inflammatory diseases and influences disease progression and severity [1,2]. Systemic inflammation is a potent prothrombotic stimulus. Inflammatory mechanisms contribute to coagulation abnormalities by up-regulating coagulation factors, down-regulating natural anticoagulants, and inhibiting fibrinolytic activity [2,3]. In addition to their role in modulating plasma coagulation pathways, inflammatory mediators have been implicated in augmenting platelet reactivity [1,2]. In vivo studies have shown that natural anticoagulants not only prevent thrombosis but also

inhibit inflammatory activity, suggesting that coagulation and inflammation interact [3].

Inflammatory bowel disease (IBD), which includes Crohn's disease (CD) and ulcerative colitis (UC), is associated with a hypercoagulable state that substantially increases the risk of venous and arterial thromboembolism [1,4,5]. It has also been demonstrated that coagulation abnormalities and platelet dysfunction are closely associated with the clinical presentation of IBD [2,6]. Venous thromboembolism (VTE) occurs in the general population at a rate of 0.7–1.5 per 1,000 people, although it varies somewhat by race and gender, and is one of the leading causes of death in hospitalized patients [2,5,7]. Risk factors included age, surgery, genetic factors, trauma, hospitalization, malignancy, central venous

catheter use, and prior venous thrombosis [7-10]. CD and UC are also independent risk factors for VTE, which is approximately 2–3 times more common in patients with IBD than in the general population [5,9,11]. Hospitalized IBD patients with VTE had a higher risk of death than hospitalized patients without VTE, and patients with IBD had a higher risk of VTE recurrence than those without IBD [1,12,13]. Since the occurrence of thromboembolic events is associated with poor prognosis in IBD patients, as well as they role as a major cause of mortality [12,14], this review focuses on coagulation and platelet abnormalities in IBD patients.

COAGULOPATHY IN IBD

Patients with IBD exhibit a persistent prothrombotic state characterized by excessive thrombin generation, endothelial dysfunction, and an imbalance between procoagulant and anticoagulant factors. Elevated levels of prothrombin fragment 1+2, thrombin-antithrombin complexes, and fibrinogen have been consistently observed in patients with IBD, even during periods of disease inactivity [10,15]. These markers reflect ongoing activation of the coagulation cascade. A marked increase in von Willebrand factor (VWF), coupled with reduced activity of a disintegrin-like metalloproteinase with thrombospondin motif type 1 member 13 (ADAMTS13), a metalloprotease responsible for VWF cleavage, contributes to endothelial dysfunction and promotes thrombosis. VWF is a large multimeric glycoprotein that plays a crucial role in platelet adhesion and aggregation, particularly under high shear stress conditions. Elevated levels of VWF are often associated with systemic inflammation and endothelial damage, as observed in various conditions, including IBD, hypertension, and other prothrombotic states. ADAMTS13 is a metalloprotease that cleaves ultra-large VWF multimers into smaller, less prothrombotic forms. Reduced ADAMTS13 activity leads to the accumulation of large biologically active VWF multimers that enhance platelet adhesion and aggregation, thereby increasing the risk of thrombosis [16,17]. The hypercoagulable state in IBD is further supported by the upregulation of tissue factor (TF) expression, which promotes the initiation of the extrinsic coagulation cascade. TF are key initiators of the extrinsic coagulation cascade. TF expression is increased in monocytes and microparticles in IBD. This upregulation contributes to the generation of thrombin and formation of fibrin, thereby

enhancing the hypercoagulable state. For instance, TF-bearing microparticles are elevated in IBD patients, potentially linking inflammation to coagulation activation [18]. TF-dependent pathways are substantially involved in the inflammatory responses in IBD. TF expression in immune cells and microparticles promotes procoagulant activity and amplifies inflammatory responses. The role of TF in IBD is further supported by its ability to activate protease-activated receptors, which mediate proinflammatory signaling, thus linking coagulation with inflammation [19]. Increased levels of TF expression in patients with IBD have also been associated with enhanced thrombin generation and localized thrombosis, which are characteristic features of the hypercoagulable state of IBD [20]. Additionally, a dysregulated protein C pathway and decreased levels of natural anticoagulants, such as protein S and antithrombin III, further exacerbate the prothrombotic environment. The protein C pathway plays a crucial role in anticoagulation, inflammation regulation, and the maintenance of vascular integrity. Disruption of this pathway, as observed in conditions such as IBD, leads to impaired downregulation of coagulation and inflammation. This is exacerbated by the reduced levels of protein S and antithrombin III, which are essential natural anticoagulants [1-3,10,14,21].

The coagulation cascade in IBD is dysregulated at multiple levels and involves both extrinsic and intrinsic pathways. The extrinsic pathway is primarily activated by TF that play a central role in the initiation of coagulation. TF is upregulated in inflamed intestinal mucosa and systemic circulation in patients with IBD. TF binds to factor VIIa, initiating a cascade that leads to increased thrombin production [19,20,22]. This results in excessive fibrin deposition that contributes to intravascular thrombus formation.

The intrinsic pathway, which is typically initiated by factor XII activation, is also dysregulated in patients with IBD. Elevated levels of activated factors XI and IX suggest ongoing activation of the intrinsic pathway, which is potentially driven by endothelial injury and chronic systemic inflammation. Increased levels of activated factor XI (FXIa) have been reported in patients with IBD. FXIa contributes to the intrinsic pathway to thrombin generation and coagulation. Elevated FXIa levels have been observed in both CD and UC and are associated with thrombotic risks [23]. Similarly, elevated levels of activated FIXa have been identified in patients with IBD. FIXa collaborates with FVIIIa to form an intrinsic tenase complex that promotes thrombin generation. Elevated FIX

levels in active IBD suggest a persistent prothrombotic state [24]. Endothelial dysfunction and systemic inflammation in patients with IBD may drive activation of this intrinsic pathway by exposing the subendothelial matrix, which promotes factor XII activation. Inflammatory cytokines such as interleukin (IL)-1 and tumor necrosis factor- α (TNF- α) also enhance coagulation factor expression and activity [1,24]. The presence of autoantibodies against key intrinsic pathway components may indicate an immune-mediated component to coagulation abnormalities in IBD. Autoantibodies against coagulation factors, including antiphospholipid and antiprotein S, have been identified in patients with IBD [2,20]. They are believed to contribute to the prothrombotic state by impairing natural anticoagulation mechanisms, promoting thrombosis, and exacerbating the inflammatory response.

GUT DYSBIOSIS AND ENDOTOXINS

Gut dysbiosis and the associated endotoxins are major contributors to the pathophysiology of coagulopathy in patients with IBD. Gut dysbiosis, characterized by altered intestinal microbiota, increases intestinal permeability, leading to the translocation of bacterial endotoxins, such as lipopolysaccharides (LPS), into the systemic circulation [25]. Elevated LPS levels trigger inflammatory responses and activate the coagulation cascade via Toll-like receptor 4-dependent pathways, thereby promoting thrombus formation [26]. Additionally, microbiota-derived metabolites, such as trimethylamine-N-oxide, are associated with platelet hyperreactivity and increased thrombosis risk [27,28]. To treat gut dysbiosis and reduce the risk of dysbiosis-related thrombosis in patients with IBD, restoration of the gut microbiota balance through several methods, including probiotics, dietary interventions, and fecal microbiota transplantation, is being investigated [29-31]. These therapeutic approaches are expected to modulate intestinal permeability, reduce systemic inflammation, and subsequently attenuate coagulation activation and thromboembolic risk; however, further research is required.

PLATELET ABNORMALITIES IN IBD

Thrombocytosis is frequently observed during the active

phase of IBD and is correlated with disease severity. It has been proposed as a potential marker of disease activity in patients with both CD and UC. Elevated platelet counts are directly correlated with systemic inflammation markers such as C-reactive protein (CRP) and IL-6 [2,32]. The increase in circulating platelets during active IBD is likely due to enhanced thrombopoiesis driven by inflammatory cytokines such as IL-6 and thrombopoietin. These cytokines are upregulated during inflammation, stimulate platelet production, and contribute to reactive thrombocytosis [33,34]. Increased thrombopoiesis results in platelet overproduction, leading to a hypercoagulable state [5,35]. In these patients, the mean platelet volume (MPV) often decreases, indicating increased platelet turnover owing to increased platelet demand. Reactive thrombopoiesis produces smaller but functionally hyperactive platelets, which increase procoagulant potential [32,34]. The reduction in MPV has been associated with the consumption of larger, more metabolically active platelets at sites of inflammation, further contributing to disease pathophysiology [2,36]. Platelet activation in IBD is dysregulated and is characterized by the upregulation of platelet surface glycoproteins, such as glycoprotein IIb/IIIa and P-selectin, which enhance platelet aggregation and adhesion [2,33,35]. The increased expression of these receptors facilitates the interactions between VWF and fibrinogen, amplifying clot formation [16,35,37]. Additionally, elevated levels of platelet-derived extracellular vesicles (P-LEVs), which contain high concentrations of phosphatidylserine and TF, potentiate thrombin generation, thereby sustaining the prothrombotic state. Phosphatidylserine on the surface of P-LEVs provides a negatively charged catalytic surface that supports the assembly of coagulation factor complexes, whereas TF acts as a direct initiator of the extrinsic coagulation pathway. Collectively, these properties enhance thrombin generation and contribute to a hypercoagulable state. P-LEVs promote excessive coagulation, and their presence has been linked to increased thrombotic events in patients with IBD [18,20,38].

The interplay between platelets and leukocytes in IBD exacerbates disease severity. Platelet-leukocyte aggregates, particularly platelet-neutrophil and platelet-monocyte complexes, are prominently elevated in patients with IBD. These aggregates act as key mediators of thrombosis and inflammation, thereby amplifying endothelial activation and tissue damage [1,2,39]. The binding of activated platelets to leukocytes enhances the secretion of pro-inflammatory cytokines such as TNF- α and IL-1 β , contributing to sustained

inflammation and endothelial dysfunction. Activated platelets engage leukocytes via P-selectin (on platelets) binding to P-selectin glycoprotein ligand-1 on leukocytes. This interaction promotes the secretion of inflammatory mediators such as TNF- α and IL-1 β by leukocytes, thereby exacerbating inflammation [37,40]. Furthermore, activated platelets upregulate CD40 ligand (CD40L), which plays a crucial role in perpetuating the inflammatory response by interacting with endothelial and immune cells. CD40L expressed on activated platelets can bind to CD40 on endothelial cells, leading to the upregulation of adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1) as well as the secretion of proinflammatory cytokines such as IL-8. These interactions facilitate leukocyte adhesion, transmigration, and recruitment to the sites of inflammation, thereby amplifying the inflammatory response [38,40,41].

Endothelial dysfunction in IBD is mediated by platelet-driven alterations in vascular integrity. Increased platelet activation leads to the secretion of pro-angiogenic and pro-thrombotic factors such as thrombospondin-1, serotonin, and platelet factor 4, which promote endothelial damage and vascular remodeling [2,39,42]. Moreover, platelets serve as reservoirs of miRNAs and inflammatory mediators that modulate immune responses [35,42]. Dysregulated platelet-endothelial interactions contribute to microvascular thrombosis, which can impair mucosal healing and exacerbate ischemic injury in the gastrointestinal tract [16,17,33,40].

In summary, enhanced platelet activation, P-LEV formation, and platelet-leukocyte interactions in patients with IBD contribute substantially to the observed hypercoagulable state. These abnormalities not only increase the risk of thromboembolic complications but also play a critical role in perpetuating intestinal inflammation, creating a negative reinforcement between thrombosis and immune activation.

ALTERED FIBRINOLYSIS IN IBD

Fibrinolysis is substantially impaired in IBD, contributing to persistent thrombotic risk. The fibrinolytic system, which is responsible for breaking down fibrin clots and maintaining vascular patency, is dysregulated because of the interplay between inflammatory mediators, endothelial dysfunction, and coagulation abnormalities. Proinflammatory cytokines such as TNF- α , IL-1, and IL-6 are known to disrupt the fibrinolytic system.

These cytokines increase the levels of plasminogen activator inhibitor-1 (PAI-1), which inhibits tissue plasminogen activator (tPA) and urokinase plasminogen activator (uPA) [2,22]. Elevated levels of PAI-1, a key inhibitor of fibrinolysis, suppress tPA activity, resulting in prolonged clot stability and delayed thrombus resolution [43]. In IBD, PAI-1 overexpression is driven by inflammatory cytokines that exacerbate the prothrombotic state [44]. Concurrently, decreased uPA levels further disrupt fibrinolytic efficiency, limiting the ability to degrade fibrin deposits within the vasculature [2,45]. The reduction in uPA activity correlates with increased disease activity and endothelial injury, compounding the risk of thrombus formation [15]. In addition, alterations in the balance of thrombin-activatable fibrinolysis inhibitors (TAFI) further modulate fibrinolytic activity, with increased TAFI levels contributing to delayed fibrin degradation and a sustained hypercoagulable state [2,43,46].

Markers of excessive fibrin turnover, such as D-dimer and fibrin degradation products (FDPs), are elevated in patients with IBD, reflecting ongoing thrombus formation and impaired clearance. Increased circulating D-dimer levels have been associated with active disease states, suggesting the chronic activation of coagulation and subsequent ineffective fibrinolysis. Elevated FDP levels indicate persistent clot breakdown, but insufficient fibrinolytic activity to counteract excessive coagulation [15,47,48].

Additionally, autoantibodies against fibrinolytic proteins, including antiplasminogen and antiprotein C, have been detected in a subset of patients with IBD, suggesting a potential immune-mediated component of fibrinolytic dysfunction. These autoantibodies interfere with normal fibrinolysis, further exacerbating clot persistence and increasing the thrombotic risk [45,49]. Impaired endothelial protein C receptor (EPCR) function, commonly seen in IBD, also compromises the anticoagulant and fibrinolytic pathways, reducing the overall ability to counteract thrombosis. EPCR facilitates the activation of protein C by thrombin bound to thrombomodulin. Activated protein C exerts its anticoagulant effect by inactivating the coagulation factors Va and VIIIa. Decreased EPCR expression results in decreased activation of protein C, which is a key factor in regulating excessive coagulation [3,37]. This dysregulated fibrinolytic balance exacerbates the already increased thrombotic risk seen in IBD, particularly during disease exacerbation and in the presence of additional predisposing factors such as immobility, surgery, and corticosteroid use. Immobility due to

prolonged bed rest or reduced physical activity contributes to venous stasis, which is a key factor in the development of VTE. This is especially concerning during acute IBD flares or hospitalization [4,6]. Prolonged exposure to corticosteroids induces a prothrombotic state by modulating fibrinolysis and increasing the levels of fibrinogen and PAI-1, while suppressing tPA activity. High-dose corticosteroids have been reported to increase the risk of VTE by 3.31 times compared to lower doses [6,10,12,50]. IBD patients undergoing surgical intervention are at compounded risk because they are more likely to develop thromboembolism due to postoperative fibrinolytic inhibition [10,11].

The risk of VTE may increase or decrease with the development of advanced therapies for IBD. Tofacitinib is an oral JAK inhibitor that demonstrated efficacy in UC but was associated with an approximately 2.5-fold increased risk of pulmonary embolism and deep vein thrombosis at a dose of 10 mg twice daily [51]. A study on tofacitinib in patients with rheumatoid arthritis reported a 33% increase in major adverse cardiovascular events compared to TNF inhibitors, particularly in patients aged 50 years or older with cardiovascular risk factors [52]. As a result, the U.S. FDA has tightened restrictions on use in high-risk populations. Although the study was conducted in patients with rheumatoid arthritis, VTE has been reported with other JAK inhibitors such as upadacitinib and baricitinib; therefore, caution and monitoring are recommended [53,54]. Anti-TNF agents are associated with a decreased risk of VTE. Anti-TNF agents have been shown to reduce platelet activation, which is a critical factor in the development of thrombus formation, and improve the ability of endothelial cells to activate protein C, which is crucial for the regulation of coagulation; this helps prevent excessive coagulation and lowers the production of TF, an initiator of the coagulation cascade, thereby further decreasing the likelihood of thrombus formation [55].

COAGULOPATHY IN EASTERN AND WESTERN IBD PATIENTS

Hypercoagulable states associated with IBD are recognized as an important clinical problem in both Eastern and Western populations. However, there are some epidemiological and pathophysiological differences between these two populations. Asian patients with IBD demonstrate markedly lower absolute VTE incidence rates than Western patients

with IBD, with population-based studies reporting 8–18 per 10,000 person-years in Korea, Taiwan, and Japan versus 25–42 per 10,000 person-years in Western populations [56–61]. However, the relative risk compared with the general population remains remarkably similar, with both Asian and Western patients exhibiting a 2.0–3.6-fold increased risk of thrombotic events [56,62,63]. These epidemiological differences between Eastern and Western populations are often due to fundamental genetic differences in thrombophilia prevalence, with factor V Leiden and prothrombin G20210A mutations occurring in fewer Asian populations compared populations (5–8%) [64,65]. Additionally, Asian patients with IBD present with distinct hemostatic profiles, including lower baseline coagulation factor VIII and fibrinogen levels, which may contribute to their reduced overall thrombogenicity [56,66]. Despite these protective factors, the inflammatory-driven hypercoagulability mediated by elevated cytokines such as IL-6, TNF- α , and TF upregulation appears to follow similar pathophysiologic pathways across both populations.

Clinical management approaches for IBD-associated coagulopathy reflect these regional differences in thrombotic and bleeding risk profiles. While Western guidelines advocate routine pharmacological thromboprophylaxis with low-molecular-weight heparin for all hospitalized patients with IBD experiencing acute flares, uptake in Asian centers remains suboptimal at approximately 60%, primarily because of heightened concerns regarding gastrointestinal bleeding complications [56,63,67]. This conservative approach is not entirely unfounded, as Asian patients with IBD demonstrate higher rates of mucosal and variceal bleeding, compounded by potential intestinal tuberculosis overlap, which further complicates anticoagulation decisions [68]. When thrombotic events occur in Asian populations, the clinical outcomes appear comparable to those in Western patients, with similar five-year recurrence rates of approximately 15% after anticoagulation discontinuation [66,69]. Recent evidence suggests that Asian IBD patients may experience lower major bleeding rates on direct oral anticoagulants compared to Western counterparts (1.9 vs. 3.4 events per 100 patient-years) while maintaining similar efficacy [62]. Based on these findings, a personalized thromboprophylactic approach that integrates regional bleeding profiles, genetic predisposition to thrombosis, and careful consideration of disease activity markers is required.

Table 1. Prophylaxis of thromboembolic events in IBD patients

Patient group	Thromboprophylaxis	Duration	Consideration
Hospitalized (any cause)	Recommended for all	Entire hospitalization	Bleeding
Active flare - outpatient	With risk factors	Until clinical remission	Risk-benefit assessment
History of VTE	Long-term consideration	≥ 3-6 mo	Regular monitoring
Extended prophylaxis	High-risk patients only	2-8 wk	Cost-effectiveness
Post-surgical	Standard + extended	2-8 wk post-discharge	Thromboembolism vs bleeding risk

IBD, inflammatory bowel disease; VTE, venous thromboembolism.

THE PROPHYLAXIS OF THROMBOEMBOLIC EVENTS IN IBD PATIENTS

The management of thromboembolic events in patients with IBD requires a tailored approach that considers the disease activity, clinical setting, and individual risk factors (Table 1). The latest evidence from international consensus statements and clinical guidelines provides recommendations for thromboprophylaxis strategies appropriate for various clinical situations [50].

All hospitalized patients with IBD, regardless of the cause of admission, received pharmacological thromboprophylaxis throughout their inpatient stay. This recommendation is based on evidence that hospitalized patients with IBD face a markedly elevated risk of VTE, even during periods of clinical remission, with hazard ratios of 1.7 compared to those without IBD [50]. Low molecular weight heparin or fondaparinux is preferred over unfractionated heparin as they demonstrate superior efficacy and safety profiles, with reduced rates of pulmonary embolism, deep vein thrombosis, major bleeding, and heparin-induced thrombocytopenia [50,70]. Thromboprophylaxis did not increase the risk of gastrointestinal bleeding in patients with active IBD [50,70].

The relationship between IBD activity and thrombosis risk provides a basis for establishing preventive strategies. During active disease flares, particularly in outpatient settings, the relative risk of VTE increases dramatically, with hazard ratios reaching 15.8 compared to the general population [71]. However, in the case of outpatients without additional risk factors, the absolute thrombosis risk remains relatively low; therefore, it is not efficient to implement uniform prophylaxis in this group. Ambulatory patients with active IBD should be considered for thromboprophylaxis only when additional major risk factors are present, such as previous thromboembolic events, prolonged immobilization, active malignancy, or concurrent autoimmune diseases [50,72].

The decision to extend thromboprophylaxis beyond hospital discharge requires careful evaluation of individual risk factors and clinical circumstances. Extended prophylaxis should be considered for patients with multiple risk factors, including age > 45 years, multiple hospital admissions, intensive care unit stays, hospitalization > 7 days, use of central venous catheters, and concurrent sepsis [50,73]. The optimal duration typically ranges from 2-8 weeks post-discharge, with the specific duration determined by the persistence of risk factors and the achievement of clinical remission. Recent evidence suggests that 91% of readmissions owing to VTE occur within 60 days of discharge, with the highest risk occurring within the first 10 days, supporting the rationale for extended prophylaxis in selected high-risk patients [74]. Patients with post-surgical IBD represent a particularly high-risk population, with extended thromboprophylaxis using low-dose rivaroxaban (10 mg daily for 30 days), demonstrating major reductions in post-discharge VTE rates (4.8-0.6%) while maintaining acceptable bleeding risk profiles [75].

CONCLUSION

IBD, comprising CD and UC, is closely associated with a hypercoagulable state driven by dysregulation of coagulation pathways, platelet abnormalities, and impaired fibrinolysis (Table 2). A persistent prothrombotic environment in patients with IBD increases the risk of thromboembolic events, which are associated with poor clinical outcomes and higher mortality rates. The interaction between inflammation and coagulation is an important factor in IBD pathogenesis in patients, with inflammatory mediators amplifying coagulation and vice versa. Understanding the mechanisms underlying coagulation and platelet dysfunction in IBD is crucial to improve risk stratification and develop therapeutic strategies. Certain IBD treatments, such as anti-TNF agents, may

Table 2. Coagulopathy and platelet abnormalities in patients with inflammatory bowel disease

Pathophysiologic component	Alteration	Clinical significance
Coagulation cascade	Tissue factor ↑ Thrombin generation ↑ Fibrinogen ↑	Increased thromboembolic risk
Natural anticoagulants	Protein C ↓ Protein S ↓ Antithrombin III ↓	Impaired anticoagulation Increased thrombosis
Fibrinolysis	PAI-1 ↑ uPA activity ↓	Reduced clot degradation Persistent thrombus
Gut dysbiosis and endotoxins	LPS ↑ TMAO ↑	Increased systemic inflammation Platelet hyperreactivity
Platelet abnormalities	Thrombocytosis ↑ Platelet activation markers (P-selectin) ↑	Hypercoagulability Correlation with disease activity
Endothelial dysfunction	VWF ↑ ADAMTS13 ↓	Enhanced platelet adhesion Endothelial injury

PAI-1, plasminogen activator inhibitor-1; uPA, urokinase plasminogen activator; LPS, lipopolysaccharide; TMAO, trimethylamine N-oxide; VWF, von Willebrand factor; ADAMTS13, a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13.

reduce thromboembolic risk, whereas others, such as JAK inhibitors, have the potential to produce thromboembolic events and should be monitored carefully. Given the high risk of thromboembolic complications in patients with IBD, a multidisciplinary approach integrating anticoagulation strategies, disease activity management, and individualized treatment plans is necessary to optimize patient outcomes and minimize thromboembolic complications.

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