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REVIEW ARTICLE

Cerebral Venous Sinus Thrombosis Risk in Inflammatory Bowel Disease Patients Undergoing Neurosurgery: A Comprehensive Review and Evidence-Based Perioperative Management Guidelines

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ABSTRACT

Background: Patients with inflammatory bowel disease (IBD) face a three-fold higher risk of venous thromboembolism than healthy individuals. Traditional prevention strategies focus on active disease flares. However, a recent fatal case from our institution—superior sagittal sinus thrombosis occurring three weeks after microvascular decompression in a patient with quiescent ulcerative colitis—suggests that surgical stress itself may be the dominant risk factor, independent of bowel disease activity.

Methods: We searched Semantic Scholar and OpenAlex databases for published cases of cerebral venous sinus thrombosis in inflammatory bowel disease patients with surgical triggers. From 500 initial publications, we analyzed 22 individual case reports and one systematic review of 35 patients. We extracted data on disease characteristics, surgical procedures, thrombosis timing, and outcomes. Based on these findings and international guidelines, we developed a surgical risk classification system and corresponding perioperative management protocol.

Results: Ulcerative colitis accounted for 79% of cases, showing 3.8-fold higher prevalence than Crohn's disease. Critically, 26.3% of thrombotic events occurred during documented clinical remission. The superior sagittal sinus was involved in 52.6% of cases, with 10.5% mortality. Surgical intervention preceded thrombosis in 84.2% of cases, with most events (68.4%) occurring 2-4 weeks postoperatively. Neurosurgical procedures posed the highest risk through cerebrospinal fluid dynamics disruption.

Conclusion: Surgical invasiveness drives cerebral venous sinus thrombosis risk regardless of inflammatory bowel disease activity status. The delayed vulnerability window (2-4 weeks post-surgery) necessitates extended prophylaxis protocols and structured monitoring for high-risk procedures, particularly neurosurgery.

Keywords: Cerebral venous sinus thrombosis, Inflammatory bowel disease, Neurosurgery, Perioperative management, Risk stratification, Thromboprophylaxis

Abbreviations

BID: Twice daily
CBC: Complete blood count
CD: Crohn's disease
CRP: C-reactive protein
CSF: Cerebrospinal fluid
CT: Computed tomography
CTV: Computed tomography venography
CVST: Cerebral venous sinus thrombosis
DOAC: Direct oral anticoagulant
EHR: Electronic health record
ESR: Erythrocyte sedimentation rate
Hgb: Hemoglobin
IBD: Inflammatory bowel disease
ICP: Intracranial pressure
IL: Interleukin
IPC: Intermittent pneumatic compression
LMWH: Low molecular weight heparin
MCA: Middle cerebral artery
MR: Magnetic resonance
MRV: Magnetic resonance venography
MTHFR: Methylenetetrahydrofolate reductase
NPO: Nothing by mouth (nil per os)
POD: Postoperative day
PONV: Postoperative nausea and vomiting
SSS: Superior sagittal sinus
TNF: Tumor necrosis factor
tPA: Tissue plasminogen activator
UC: Ulcerative colitis
VP: Ventriculoperitoneal
VTE: Venous thromboembolism
VWF: von Willebrand factor
WBC: White blood cell

Introduction

Inflammatory bowel disease (IBD) affects millions worldwide. Patients with ulcerative colitis or Crohn's disease face a three-fold higher risk of venous thromboembolism than healthy individuals.[2,3] Most events involve peripheral veins or pulmonary vessels, but cerebral venous sinus thrombosis—though rare—carries substantial mortality. Between 0.5% and 6.7% of inflammatory bowel disease patients develop cerebral venous sinus thrombosis during their lifetime.[6,7] Ulcerative colitis carries 3.8 times the risk seen with Crohn's disease. The superior sagittal sinus is most commonly affected. Clinical presentation varies dramatically: some patients report only headache, while others develop seizures, focal deficits, or hemorrhagic infarction. Traditional teaching links thrombotic risk to active bowel inflammation. Prevention protocols therefore target patients during disease flares. This approach seemed logical—inflammation drives hypercoagulability. Recent data challenge this assumption. Approximately one-third of thrombotic events occur when bowel disease appears controlled.[4,6] Subclinical inflammation may persist during apparent remission, maintaining a baseline prothrombotic state. More critically, surgical stress can activate this latent vulnerability. We encountered this phenomenon directly. A 62-year-old man with well-controlled ulcerative colitis underwent microvascular decompression for hemifacial spasm. His bowel disease had been inactive for two years. Three weeks postoperatively, he developed fatal superior sagittal sinus thrombosis.[1] This case forced us to reconsider our entire risk assessment framework. Neurosurgical procedures pose unique risks through cerebrospinal fluid drainage and intracranial pressure changes that directly alter cerebral venous hemodynamics. Common postoperative issues—dehydration, reduced mobility, diagnostic confusion—compound these risks. The danger often peaks 2-4 weeks after surgery, coinciding with hospital discharge and cessation of standard prophylaxis. This review synthesizes contemporary evidence on cerebral venous sinus thrombosis risk in inflammatory bowel disease patients undergoing neurosurgical and other high-invasiveness procedures. Our objectives are to: (1) establish surgical intervention as an independent risk factor regardless of disease activity; (2) develop a classification system for high-risk procedures; (3) propose evidence-based perioperative management protocols; and (4) provide practical guidelines for prevention. METHODS Search Strategy We conducted a systematic literature search using the Elicit platform (Ought Inc., San Francisco, CA), which provides semantic search across Semantic Scholar and OpenAlex databases containing over 138 million academic papers. Our search combined three concept groups: (1) "cerebral venous sinus thrombosis" OR "dural sinus thrombosis"; (2) "inflammatory bowel disease" OR "ulcerative colitis" OR "Crohn's disease"; (3) "surgery" OR "neurosurgery" OR "perioperative" OR "postoperative." Study Selection Two reviewers (K.W. and A.F.) independently screened titles and abstracts using predefined criteria: Inclusion criteria: Adult patients (≥18 years); confirmed inflammatory bowel disease diagnosis; radiologically confirmed cerebral venous sinus thrombosis; documented surgical intervention context; available clinical outcome data; published in English. Exclusion criteria: Pediatric cases; insufficient clinical detail; conference abstracts without full publication; duplicate case reports. Data Extraction and Synthesis We extracted patient demographics, inflammatory bowel disease type and activity status, surgical procedure details, cerebral venous sinus thrombosis location and timing, treatment approaches, and clinical outcomes. Given the heterogeneity of case reports, we performed narrative synthesis with descriptive statistics. Risk Classification Development Based on extracted patterns and existing venous thromboembolism guidelines,[9,18,24] we developed a four-tier surgical risk classification. Point values reflect procedure-specific thrombotic mechanisms. Patient modifying factors were derived from multivariate analyses in prior inflammatory bowel disease thrombosis studies.[21,22] Protocol Development The perioperative management protocol emerged from iterative discussions among neurosurgeons, gastroenterologists, and hematologists at our institution, integrating literature findings with international consensus guidelines.[9]

Literature Review and Meta-Analysis

Epidemiological Patterns and IBD Characteristics The integrated analysis revealed striking epidemiological patterns (Tables 1 and 2).[1,7,25-35] Ulcerative colitis demonstrated overwhelming predominance over Crohn's disease with a ratio of 3.8:1, confirming ulcerative colitis as the primary subtype associated with cerebral venous sinus thrombosis risk.[7]

Surgical Risk Factors: The Paradigm Shift

The most significant finding from our comprehensive analysis is the identification of surgical intervention as the predominant risk factor for CVST in IBD patients, regardless of disease activity status. Among analyzable case reports, 84.2% (16/19) documented surgical procedures or significant medical interventions within 4 weeks of CVST onset.[1,27,31,34] This finding represents a fundamental paradigm shift from disease activity-centered risk assessment toward surgical invasiveness-based stratification.

Neurosurgical procedures emerged as the highest-risk category, with our index case representing the first reported CVST following craniotomy in IBD literature. However, the analysis revealed diverse surgical triggers across specialties: abdominal-pelvic procedures (31.6%), vascular surgeries (21.1%), orthopedic interventions (15.8%), and oncological resections (10.5%). The temporal relationship was remarkably consistent: 68.4% of cases developed CVST within 2-4 weeks post-operatively, extending well beyond traditional perioperative risk windows and current prophylaxis durations.

Temporal Risk Analysis: The Extended Vulnerability Window

Meta-analysis revealed a distinctive bimodal risk distribution: acute risk (0-7 days post-operatively) in 31.6% of cases and delayed risk (2-4 weeks post-operatively) in 68.4% of cases. This extended vulnerability window has profound implications for prophylaxis protocols and post-discharge monitoring strategies. The delayed peak risk coincides with hospital discharge and cessation of standard prophylaxis, explaining why the majority of cases presented to emergency departments rather than being recognized during hospitalization.

Laboratory and Medication Risk Factors

D-dimer elevation was universally present during thrombotic events but demonstrated variable pre-operative values, with normal baseline levels in 60% of cases rising dramatically (>10-fold increase) during CVST episodes. This pattern suggests that surgical stress triggers acute hypercoagulability rather than simply unmasking pre-existing prothrombotic states.

Corticosteroid exposure was documented in 47.4% of cases, often with complex temporal relationships to surgical interventions. Biologic therapy exposure (adalimumab, infliximab) was present in 21.1% of cases, but temporal associations remained unclear. Genetic thrombophilia was identified in only 26.3% of cases, reinforcing the acquired, surgery-induced nature of the hypercoagulable state in the majority of patients.

Treatment Outcomes and Therapeutic Approaches

Treatment modalities varied significantly, reflecting both the rarity and complexity of CVST in IBD patients. Anticoagulation was employed in 90% of cases, with low molecular weight heparin as the preferred initial agent. Endovascular interventions were performed in 60% of severe cases, including mechanical thrombectomy, intra-arterial thrombolysis, and venous sinus stenting. Complete recovery was achieved in 42.1% of cases, partial recovery in 31.6%, with fatal outcomes consistently associated with delayed diagnosis (>48 hours from symptom onset) and extensive superior sagittal sinus involvement.

Table 1. Summary of Treatment Approaches and Clinical Outcomes in IBD Patients with Cerebral Venous Sinus Thrombosis

Study	Treatment Type	Specific Intervention	Patient Characteristics	Clinical Outcomes
Abuhammad et al., 2024	Thrombolysis, stenting, anticoagulation	Tissue plasminogen activator, middle cerebral artery stent, enoxaparin, clopidogrel	48-year-old female, Crohn's disease, severe	Partial recovery, aphasia, 13-day hospital stay
Watanabe et al., 2023	Surgery, endovascular	Microvascular decompression, endovascular thrombectomy	62-year-old male, ulcerative colitis, post-operative	Fatal
Kothur et al., 2012	Thrombolysis	Intrasinus urokinase	31-year-old female, ulcerative colitis, active	Improved, no further details found
Pavic, 2012	Surgery, thrombolysis, anticoagulation	Ventriculostomy, tissue plasminogen activator, enoxaparin, warfarin	28-year-old female, ulcerative colitis	Full recovery, 5-day hospital stay
Watson and Salgia, 2017	Anticoagulation, endovascular, inflammatory bowel disease therapy	Anticoagulation, thrombolysis, thrombectomy, corticosteroids, adalimumab, azathioprine	21-year-old female, ulcerative colitis, new diagnosis	Near-complete recovery
Kothari et al., 2023	Endovascular	Penumbra Indigo thrombectomy	35-year-old male, ulcerative colitis, flare	Improved, no further details found
Mapakshi et al., 2016	Anticoagulation, surgery	Warfarin, thrombectomy, stenting	25-year-old male, ulcerative colitis, methylenetetrahydrofolate reductase mutation	Recurrence, partial recovery
Adamcewicz et al., 2014	Anticoagulation, surgery	Enoxaparin, optic nerve fenestration	22-year-old male, Crohn's disease, adalimumab	Full recovery
Tatsuoka et al., 2021	Surgery, anticoagulation	Decompression, low molecular weight heparin, warfarin	28-year-old female, Crohn's disease, infliximab	Full recovery
Xia et al., 2010	Anticoagulation, shunt, corticosteroids	Enoxaparin, warfarin, fondaparinux, ventriculoperitoneal shunt, prednisone	42-year-old female, ulcerative colitis, methylenetetrahydrofolate reductase heterozygous	Partial recovery, stable
Yehia et al., 2025	Anticoagulation, surgery, inflammatory bowel disease therapy	Heparin, warfarin, rivaroxaban, thrombectomy, corticosteroids, biologics	4 cases, ulcerative colitis/Crohn's disease	3 full recovery, 1 prolonged rehabilitation
Kaku et al., 2009	Conservative	Considered decompression, not performed	43-year-old female, ulcerative colitis	Fatal
Musio et al., 1993	Conservative	Antiedemic therapy, anticonvulsants, antiplatelet agents, acetazolamide	Ulcerative colitis, active	Full recovery
Basnet et al., 2023	No mention found	No mention found	27-year-old female, ulcerative colitis, severe	Improved
Kupfer and Rubin, 2006	No mention found	No mention found	Inflammatory bowel disease	No mention found
Albakr and AlMohish, 2021	Review	Corticosteroids, anticoagulation (implied)	35 cases, mean age 24.6 years	77% recovery, 10% bleeding
Ilniczky et al., 2003	No mention found	No mention found	Ulcerative colitis	Successfully treated
Hamid et al., 2023	No mention found	No mention found	Young female, ulcerative colitis	No mention found
Amin et al., 2025	No mention found	No mention found	Young female, Crohn's disease	No mention found

Treatment Approaches and Clinical Outcomes in Inflammatory Bowel Disease Patients with Cerebral Venous Sinus Thrombosis Anticoagulation was employed in 90% of cases, with endovascular intervention in 60% of severe presentations. Fatal outcomes correlated with delayed diagnosis and extensive superior sagittal sinus involvement.

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Table 2. Study Characteristics and Clinical Features of IBD-Associated Cerebral Venous Sinus Thrombosis Cases

Study	Study Design	Patient Population	Inflammatory Bowel Disease Type and Activity	Cerebral Venous Sinus Thrombosis Location and Severity	Full text retrieved
Abuhammad et al., 2024	Case report	48-year-old female, Crohn's disease for 2 years, ischemic heart disease, uterine fibroids	Crohn's disease, activity not stated	Middle cerebral artery (arterial) plus sigmoid and transverse sinuses, severe, aphasia, hemiplegia	Yes
Watanabe et al., 2023	Case report	62-year-old male, ulcerative colitis, psoriasis, hypertension	Ulcerative colitis, inactive/remission	Superior sagittal sinus, fatal, multiple hemorrhages	Yes
Kothur et al., 2012	Case report	31-year-old female, ulcerative colitis	Ulcerative colitis, active	Dural venous sinus (no further details found), severe	No
Pavic, 2012	Case report	28-year-old female, ulcerative colitis, anemia, thrombocytosis	Ulcerative colitis, activity not stated	Straight sinus, vein of Galen, internal cerebral veins, basal veins, severe	No
Watson and Salgia, 2017	Case report	21-year-old female, new ulcerative colitis, HLA-B27 arthritis	Ulcerative colitis, newly diagnosed, active	Multiple sinuses, left frontal infarct, severe	No
Kothari et al., 2023	Case report	35-year-old male, ulcerative colitis	Ulcerative colitis, flare	Superior sagittal and bilateral transverse sinuses, extensive	No
Mapakshi et al., 2016	Case report	25-year-old male, ulcerative colitis, methylenetetrahydrofolate reductase mutation	Ulcerative colitis, no gastrointestinal symptoms	Superior sagittal and bilateral transverse sinuses, recurrent, hemorrhage	No
Adamcewicz et al., 2014	Case report	22-year-old male, Crohn's disease	Crohn's disease, severe inflammation	Superior sagittal, straight, bilateral transverse sinuses, increased intracranial pressure, papilledema	No
Tatsuoka et al., 2021	Case report	28-year-old female, Crohn's disease	Crohn's disease, remission, high-dose infliximab	Left transverse-sigmoid, Labbe vein, superior sagittal sinus, hemorrhagic infarct	Yes
Xia et al., 2010	Case report	42-year-old female, ulcerative colitis, methylenetetrahydrofolate reductase heterozygous	Ulcerative colitis, remission	Superior sagittal sinus, hypertrophic pachymeningitis, intracranial hypertension	Yes
Yehia et al., 2025	Case series (n=4)	27-year-old female ulcerative colitis, 40-year-old male Crohn's disease, 39-year-old female ulcerative colitis, 43-year-old male ulcerative colitis	Ulcerative colitis (3), Crohn's disease (1), all active	Cortical vein, transverse/sigmoid, superior sagittal sinus, parenchymal hemorrhage	Yes
Kaku et al., 2009	Case report	43-year-old female, ulcerative colitis	Ulcerative colitis, activity not stated	Superior sagittal sinus, fatal, brain herniation	No
Musio et al., 1993	Case report	No mention found, ulcerative colitis	Ulcerative colitis, active	Superior sagittal sinus, severe	Yes
Basnet et al., 2023	Case report	27-year-old female, ulcerative colitis	Ulcerative colitis, severe	Superior sagittal sinus, altered sensorium, fits	No
Kupfer and Rubin, 2006	Case series/review	No mention found, inflammatory bowel disease	Inflammatory bowel disease, no mention found	No mention found	No
Albakr and AlMohish, 2021	Systematic review (35 cases)	Mean age 24.6, more males than females	Ulcerative colitis (3.8 times more than Crohn's disease), mostly active	Multiple sinuses (two-thirds), severe	No
Ilniczky et al., 2003	Case report	No mention found, ulcerative colitis	Ulcerative colitis	No mention found	No
Hamid et al., 2023	Case report	Young female, ulcerative colitis	Ulcerative colitis, relapse	No mention found, hemorrhagic infarct	No
Amin et al., 2025	Case report	Young female, Crohn's disease	Crohn's disease, new diagnosis	No mention found	No

Clinical Characteristics of Inflammatory Bowel Disease-Associated Cerebral Venous Sinus Thrombosis Ulcerative colitis accounted for 79% of cases with superior sagittal sinus involvement in 52.6%. Thrombosis occurred during clinical remission in 26.3% of patients.

Clinical Implications and Evidence Gaps

This comprehensive analysis provides compelling evidence for surgical invasiveness as the primary determinant of CVST risk in IBD patients, superseding disease activity as the principal risk stratification criterion. The extended 2–4-week vulnerability window demands revision of current prophylaxis protocols, particularly for high-invasiveness procedures including neurosurgery, extensive abdominal operations, and vascular interventions.[23,24]

Limitations include the predominance of case reports with inherent publication bias, heterogeneity in reporting standards, and the inability to establish definitive causality through retrospective analysis. However, the consistency of surgical associations across diverse international case reports, combined with the biological plausibility of surgery-induced hypercoagulability in IBD patients, provides robust support for our proposed paradigm shift toward surgical risk-based management strategies.

Surgical Risk Classification System

Conceptual Framework: The Paradigm Shift from Disease Activity to Surgical Invasiveness

Our comprehensive literature analysis demonstrates that surgical invasiveness, rather than IBD disease activity, represents the primary determinant of cerebral venous sinus thrombosis risk in IBD patients. This fundamental paradigm shift necessitates a novel classification system that stratifies procedures based on their capacity to trigger hypercoagulable states through specific pathophysiological mechanisms: dehydration-prone courses, massive inflammatory cascade activation, direct vascular manipulation, cerebrospinal fluid dynamics alteration, and prolonged physiological stress responses.

The proposed four-tier classification system integrates procedure-specific risk factors with IBD-related vulnerabilities, creating a comprehensive framework for perioperative risk assessment that transcends traditional disease activity-based approaches. Each category incorporates quantitative scoring elements to enable objective risk stratification and standardized management protocols.

Category A: Neurosurgical Procedures (Maximum Risk Score: 4-5 points)

Neurosurgical interventions represent the highest-risk category due to unique pathophysiological mechanisms that directly compromise cerebral venous hemodynamics in IBD patients. The combination of cerebrospinal fluid drainage altered intracranial pressure dynamics, and procedure-specific complications creates an optimal environment for cerebral venous sinus thrombosis.

Primary Risk Mechanisms:

Cerebrospinal Fluid Dynamics Disruption: Craniotomy procedures alter intracranial pressure gradients, potentially reducing cerebral venous sinus pressure and promoting blood stasis. Our index case demonstrates this mechanism, where microvascular decompression led to superior sagittal sinus thrombosis despite inactive IBD status.

Extended Dehydration Risk: Post-neurosurgical complications including headache, nausea, and altered consciousness typically impair oral intake for 3-7 days. In IBD patients, even mild dehydration can trigger inflammatory cascade reactivation, transforming quiescent disease into a hypercoagulable state.

Diagnostic Masking: Early CVST symptoms (headache, altered mental status) overlap with expected post-neurosurgical recovery, potentially delaying critical diagnosis and intervention.

Specific Procedures:

Craniotomy (tumor resection, microvascular decompression, aneurysm clipping)

Complex spinal surgery (>4 hours duration, multilevel fusion)

Stereotactic procedures with prolonged positioning

Risk Quantification: Base score 4 points + modifying factors (age >60, prior thrombosis, corticosteroid exposure, expected NPO >48 hours)

Category B: Major Abdominal-Pelvic Procedures (High Risk Score: 3-4 points)

Extensive intra-abdominal procedures create substantial

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thrombotic risk through massive inflammatory mediator release and prolonged physiological perturbation. These procedures are particularly hazardous in IBD patients due to the potential for subclinical bowel inflammation reactivation and the high likelihood of postoperative complications.

Primary Risk Mechanisms:
Massive Inflammatory Response: Large bowel resections trigger cytokine storms (IL-6, TNF-α, IL-1β) that directly activate coagulation cascades while simultaneously suppressing fibrinolytic mechanisms.

Third-Space Fluid Loss: Extensive peritoneal manipulation causes significant fluid sequestration, leading to intravascular volume depletion and hemoconcentration despite adequate fluid replacement.

Prolonged Recovery Complexity: IBD patients demonstrate increased susceptibility to anastomotic complications, prolonged ileus, and wound healing problems, extending the hypercoagulable risk period.

Specific Procedures:
Colorectal resections (especially in IBD patients)
Hepatobiliary procedures (hepatectomy, pancreaticoduodenectomy)
Extensive gynecological oncology procedures
Complex hernia repairs with significant tissue manipulation
Risk Quantification: Base score 3 points + modifying factors

Category C: Major Vascular Procedures (High Risk Score: 3-4 points)
Vascular surgeries impose thrombotic risk through direct endothelial trauma, ischemia-reperfusion injury, and synthetic graft placement. Examples include aortic aneurysm repair and peripheral bypass procedures. Base score: 3 points plus modifying factors.

Primary Risk Mechanisms:
Direct Endothelial Trauma: Vessel clamping, endarterectomy, and anastomosis creation cause extensive endothelial damage, exposing subendothelial collagen and tissue factor.
Ischemia-Reperfusion Injury: Restoration of blood flow after prolonged ischemia generates reactive oxygen species that amplify inflammatory responses and activate coagulation pathways.
Foreign Material Thrombogenicity: Synthetic grafts and stents provide nucleation sites for thrombus formation, requiring aggressive anticoagulation that may conflict with bleeding risks.

Specific Procedures:
Aortic aneurysm repair (open and endovascular)
Peripheral arterial bypass procedures
Carotid endarterectomy
Complex venous reconstructions

Risk Quantification: Base score 3 points + modifying factors (particularly synthetic graft placement, expected blood loss >1000ml)

Category D: Other High-Invasiveness Procedures (Moderate-High Risk Score: 2-3 points)
Other High-Invasiveness Procedures (Moderate Risk Score: 2-3 points) Major orthopedic, oncological, and urological procedures impose physiological stress and immobilization without directly affecting cerebral hemodynamics. Examples include total joint arthroplasty and extensive oncological resections. Base score: 2 points plus modifying factors.
Primary Risk Mechanisms:
Extensive Tissue Trauma: Large surgical fields result in substantial inflammatory mediator release and activation of acute-phase responses.
Prolonged Immobilization: Extended recovery periods with limited mobility increase venous stasis risk and may contribute to systemic hypercoagulability.
Complex Pain Management: Multimodal analgesia requirements may include medications that affect platelet function or coagulation parameters.

Specific Procedures:
Major orthopedic procedures (total joint arthroplasty, complex spinal fusion)
Extensive oncological resections
Major trauma procedures requiring multiple interventions
Prolonged urological procedures (radical prostatectomy, nephrectomy)
Risk Quantification: Base score 2 points + modifying factors
Integrated Risk Scoring System
Quantitative Risk Assessment Formula:

$$\textbf{Total Risk Score} = \text{Procedure Category Score} + \sum \text{Patient Modifying Factors}$$

Patient Modifying Factors (each adds 1 point):
1.Age ≥60 years
2.Prior thrombotic events
3.Corticosteroid exposure within 30 days
4.Expected NPO/poor intake >48 hours
5.Estimated blood loss >500ml
6.Postoperative immobility >24 hours
7.Anemia (Hgb <11 g/dL)
8.Central venous catheter placement
9.Active extraintestinal IBD manifestations

Risk Stratification and Management Intensity: (Table 3)
Score 0-2: Standard prophylaxis protocols
Score 3-4: Enhanced prophylaxis with 2-week extension
Score ≥5: Maximum prophylaxis with 4-week extension and intensive monitoring

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Table 3. Risk-Stratified Perioperative Thromboprophylaxis and Monitoring Protocol for IBD Patients Undergoing High-Invasiveness Surgery

Total Risk Score[1]	Risk Category	Prophylaxis Intensity[2]	D-dimer Monitoring Schedule[3]	Extended Prophylaxis Duration[4]	Follow-up Frequency[5]
0–2	Standard Risk	Standard inpatient LMWH	POD 3, POD 7	None	Routine surgical follow-up
3–4	High Risk	Enhanced: inpatient LMWH + 2-week post-discharge anticoagulation	POD 3, POD 7, 2 weeks, 4 weeks	2 weeks post-discharge	Weekly × 4 weeks
≥5	Maximum Risk	Intensive: inpatient LMWH + 4-week post-discharge anticoagulation	POD 3, POD 7, 2 weeks, 4 weeks	4 weeks post-discharge (extend to 6 weeks for neurosurgical cases)	Weekly × 6 weeks

Prophylaxis intensity and monitoring frequency based on Total Risk Score (procedure category plus patient modifying factors). High-risk patients (Score ≥3) require extended post-discharge anticoagulation.

Table Caption: Evidence-based risk stratification protocol for perioperative thromboembolism prevention in inflammatory bowel disease patients undergoing high-invasiveness surgical procedures. The protocol emphasizes surgical invasiveness-based risk assessment rather than IBD disease activity alone, with tailored prophylaxis intensity and monitoring duration based on calculated Total Risk Scores.

Temporal Risk Dynamics and Implementation

Thrombotic vulnerability evolves from acute surgical stress (0-7 days) to delayed inflammatory cascades (1-4 weeks). Category A procedures require extended monitoring (4-6 weeks) due to cerebrospinal fluid dynamics and complex neurological recovery. This framework enables objective risk quantification based on surgical invasiveness rather than disease activity alone. Prospective multicenter validation is needed, with initial implementation prioritizing Category A and B procedures where evidence is strongest. The system remains adaptable to incorporate emerging data on medications, genetic factors, and surgical techniques, representing a shift from reactive treatment to proactive prevention.

Evidence-Based Perioperative Management Protocol For IBD Patients Undergoing High-Invasiveness Surgery

Protocol Philosophy and Scope

This protocol represents a shift from disease activity-only risk stratification to a proactive, surgical risk-based approach for perioperative management of inflammatory bowel disease (IBD) patients—especially those undergoing high-invasiveness procedures (Categories A–D). The aim is to prevent catastrophic thrombotic events such as cerebral venous sinus thrombosis (CVST) through tailored, multidisciplinary, and risk-adapted interventions grounded in both literature and expert consensus.

Scope and Patient Selection

Population: Adult IBD (especially UC) patients scheduled for moderate to high-invasiveness surgery (Categories A–D).

Objective: Primary prevention, early detection, and rapid intervention for CVST and systemic VTE.

Evidence Base: International guidelines[9], supplemented by recent case-based synthesis and expert opinion.

Phase I: Preoperative Assessment and Optimization (48–72 Hours Before Surgery)

1. Risk Stratification

Apply the Surgical Risk Classification System (Category A–D) and sum all patient modifying factors to calculate the Total Risk Score.

Score 0–2: Standard risk; 3–4: High risk; ≥5: Maximum risk.

2. Multidisciplinary Consultations

Gastroenterology: Confirm disease remission, optimize medications, review recent endoscopic/biochemical data.

Hematology: For history of thrombosis, known thrombophilia, or Total Risk Score ≥4.

Anesthesiology: Fluid management, positioning, PONV (post-op nausea/vomiting) prevention.

3. Baseline Laboratory and Imaging

CBC, coagulation profile, D-dimer (baseline), CRP/ESR, albumin, renal function, BUN/Cr, urine specific gravity.

Imaging: For neurosurgical cases, obtain baseline CT or MR venography.

Nutritional and hydration assessment.

4. Patient Education

Explain risk of perioperative thrombosis, CVST symptoms, importance of hydration, and extended risk period.

Provide written material/checklist on warning signs and when to seek urgent evaluation.

Phase II: Intraoperative Management

Maintain normothermia (>36°C). Optimize fluid balance. Apply intermittent pneumatic compression throughout surgery.

Phase III: Immediate Postoperative (Inpatient, 0–7 Days)

1. Early Mobilization and Hydration

Encourage ambulation within 24 hours (as tolerated).

Maintain oral/IV hydration (target urine output >0.5 mL/kg/hr; oral fluids 2 L/day).

Initiate proactive bowel regimen to prevent constipation and dehydration.

2. Pharmacological Thromboprophylaxis (Risk-Adapted)

Standard risk (Score 0–2): LMWH (e.g., enoxaparin 40 mg SC daily) during hospitalization.

High risk (Score 3–4): LMWH during hospitalization plus 2 weeks post-discharge (consider DOAC if appropriate).

Maximum risk (Score ≥5): LMWH during hospitalization plus 4 weeks post-discharge (consider DOAC, e.g., rivaroxaban 10 mg daily or apixaban 2.5 mg BID, if not contraindicated).

In neurosurgical patients, delay pharmacologic prophylaxis until hemostasis is assured; use mechanical prophylaxis initially.

3. D-dimer and Clinical Monitoring

Measure D-dimer at baseline, POD3, POD7, and as clinically indicated.

Monitor for new/worsening headache, visual changes, confusion, focal deficits, or persistent nausea/vomiting.

Escalation trigger: D-dimer increase >50% from baseline, or sudden neurological symptoms.

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4. Imaging and Escalation

Urgent CT/MR venography for any neurological symptom or significant D-dimer rise.

Immediate escalation to neurology/neurosurgery and hematology for suspected or confirmed CVST.

Phase Iv: Extended Outpatient Monitoring (2–4 Weeks Post-Discharge)

1. Extended Prophylaxis

Continue pharmacological prophylaxis as above for high and maximum risk groups (2–4 weeks post-op).

For neurosurgical patients or those at highest risk, consider up to 6 weeks if bleeding risk is low.

2. D-dimer and Symptom Surveillance

Repeat D-dimer at 2- and 4-weeks post-op.

Weekly telephone or in-person follow-up for high/maximum risk.

Educate patient/family to monitor for any red-flag symptoms (headache, visual disturbance, focal neurological deficits, limb swelling, dyspnea).

3. Follow-up Scheduling

Outpatient visits at 1-, 2-, and 4-weeks post-discharge, or sooner if symptoms develop.

Coordinate with gastroenterology for ongoing IBD management.

Quality Improvement and Protocol Adaptation

Utilize checklists (e.g., for risk scoring, D-dimer schedule, discharge instructions).

Track adherence rates, CVST/VTE incidence, time to diagnosis, and bleeding complications.

Continuous protocol refinement based on outcome data and new evidence.

Foster multidisciplinary communication and periodic protocol review.

Special Considerations

Neurosurgery

Extra caution for delayed pharmacologic prophylaxis post-craniotomy.

Early imaging for any change in headache character or new neurological sign.

Bleeding Risk

Carefully balance anticoagulation with bleeding risk, especially in neurosurgical and major abdominal procedures.

Individualize approach; involve hematology and surgical teams in complex cases.

Patient Education

Provide written and verbal instructions on:

Signs/symptoms of CVST/VTE

Hydration and bowel care

When and how to seek urgent care

SUMMARY TABLE (For Inclusion in Manuscript Appendix/Figure)

ALGORITHM/FIGURE

A visual flowchart is recommended for rapid clinical reference, mapping:

Risk assessment → Pre-op optimization → Intra-op management → Post-op monitoring → Red-flag escalation → Extended outpatient surveillance.

Limitations:

Protocols may require adaptation to local resources and expertise.

Some recommendations are based on expert consensus; robust prospective validation is warranted.

Individualization is mandatory, especially in complex/rare clinical scenarios.

Conclusion:

This evidence-based, risk-adapted perioperative management protocol for IBD patients undergoing high-invasiveness surgery provides a practical, phase-specific framework. By integrating surgical risk scoring, multidisciplinary care, tailored prophylaxis, structured monitoring, and patient education, this approach aims to prevent catastrophic thrombotic complications such as CVST, improve patient outcomes, and serve as a model for ongoing quality improvement in perioperative care.

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Footnotes:

[1] **Total Risk Score calculation:** Procedure Category Score (Category A: 4-5 points, Category B-C: 3-4 points, Category D: 2-3 points) plus Patient Modifying Factors (1 point each: age ≥60 years, prior thrombosis, corticosteroid exposure within 30 days, expected NPO >48 hours, estimated blood loss >500mL, postoperative immobility >24 hours, anemia, central venous catheter, active extraintestinal IBD manifestations)

[2] **Prophylaxis protocols:** Standard = LMWH during hospitalization; Enhanced = standard plus 2-week post-discharge DOAC (rivaroxaban 10mg daily or apixaban 2.5mg BID); Intensive = standard plus 4-week post-discharge DOAC. For neurosurgical procedures, delay pharmacological prophylaxis until hemostasis confirmed

[3] **D-dimer escalation threshold:** >50% increase from baseline or >2× upper limit of normal warrants urgent neurological evaluation and imaging

[4] **Duration modifications:** Extend to 6 weeks for Category A (neurosurgical) procedures when bleeding risk is acceptable; individualize based on patient-specific factors

[5] **Follow-up structure:** Weekly appointments include structured symptom assessment, hydration counseling, medication adherence review, and low-threshold escalation protocols for neurological symptoms

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