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REVIEW

Postoperative pseudomembranous colitis after abdominal surgery: pathogenesis, diagnosis and current management

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Abstract

Pseudomembranous colitis, a severe complication of antibiotic use, is primarily caused by *Clostridioides difficile* infection (CDI), and poses a significant risk to postoperative patients, especially following abdominal surgery. We reviewed the pathogenesis, diagnostic challenges, prevention strategies, and management approaches for postoperative pseudomembranous colitis in both adult and pediatric populations. Antibiotic-induced disruption of the normal gut microbiota enables colonization and toxin production by *C. difficile*, with additional influences from surgical factors and host susceptibility. Diagnostic methods include stool tests (Glutamate dehydrogenase (GDH) antigen, toxin Enzyme immunoassay (EIA), Nucleic acid amplification test (NAAT)) and endoscopy, with specific considerations needed for pediatric patients due to high colonization rates in infants. Prevention focuses on antibiotic stewardship, with treatment options ranging from oral vancomycin and fidaxomicin for mild-to-moderate cases to surgical intervention for fulminant disease. Fecal microbiota transplantation has emerged as an effective treatment for recurrent and severe cases. While most patients recover with appropriate treatment, CDI significantly prolongs hospitalization and increases readmission rates. Early recognition, prompt diagnosis, and tailored management are essential to improve outcomes in this potentially life-threatening complication of surgery.

Keywords

Pseudomembranous colitis; *Clostridioides difficile*; Postoperative infection; Antibiotic stewardship; Fecal microbiota transplantation

1. Introduction

Pseudomembranous colitis is a severe antibiotic-associated colitis most often caused by *Clostridioides difficile* infection (CDI). It is characterized by inflammatory pseudomembranes in the colon and presents with profuse diarrhea, abdominal pain and systemic illness. However, CDI can manifest in milder, more chronic forms, particularly among younger patients with recurrent episodes or those undergoing multiple antibiotic or immunosuppressive treatments [1, 2]. These patients may experience persistent, low-grade gastrointestinal symptoms that can be mistaken for other conditions, leading to underdiagnosis and delayed treatment [1, 2]. The chronicity of symptoms in such cases underscores the importance of considering CDI in differential diagnoses, even when presentations are atypical. Postoperative patients, especially after abdominal surgery, are at heightened risk due to perioperative antibiotic exposure and hospitalization. CDI accounts for approximately 15% of nosocomial infections [3]. In surgical populations, CDI is an important complication with significant morbidity. A national analysis found an overall CDI incidence of ~0.3% within 30 days after laparoscopic abdominal surgery, with higher rates (~1.0%) following colorectal procedures [4]. CDI was reported in ~0.4% of general surgery patients in 2019 [4]. These postoperative CDI cases carry a disproportionate impact—affected patients had hospital stays three times longer and a >4-fold higher 30-day mortality (1.8% *vs.* 0.2%) compared to surgical patients without CDI [3].

Both adult and pediatric patients can develop postoperative pseudomembranous colitis, though epidemiology and outcomes differ. The incidence of CDI in children has risen in recent years, with pediatric cases being more often community-associated (75%) than healthcare-associated [5]. Severe fulminant colitis remains rare in pediatrics but carries high morbidity when it occurs [5]. Nearly 4% of all CDI cases may progress to a fulminant course, requiring intensive care or surgery [6]. Given the potential severity, general surgeons must maintain vigilance for postoperative pseudomembranous colitis as a cause of postoperative diarrhea or sepsis. This review provides a comprehensive overview of pathogenesis, diagnostic challenges, prevention strategies, and management

approaches in both adult and pediatric populations. The diagnostic workup and management pathway for postoperative pseudomembranous colitis is illustrated in Fig. 1. This review article is particularly significant as it comprehensively synthesizes the current understanding of postoperative pseudomembranous colitis specifically following abdominal surgery, an area inadequately explored in existing literature. Unlike previous reviews that broadly address CDI without surgical context, this work uniquely integrates the pathogenesis, diagnostic challenges, preventive strategies, and advanced management approaches tailored explicitly for postoperative surgical patients. It emphasizes both adult and pediatric populations, highlighting the nuanced differences in epidemiology, diagnosis and outcomes, which have been less systematically discussed in earlier works. Furthermore, this article incorporates recent advancements such as fecal microbiota transplantation and surgical innovations like diverting ileostomy with lavage, offering clinicians up-to-date, evidence-based insights. Thus, this targeted review addresses critical gaps in current guidelines and provides practical recommendations aimed at improving patient outcomes in a population vulnerable to significant morbidity and mortality.

2. Pathogenesis of postoperative pseudomembranous colitis

2.1 Role of antibiotics and dysbiosis

The pathogenesis of pseudomembranous colitis centers on disruption of the normal colonic microbiota, usually by antibiotics, allowing *C. difficile* to overgrow and produce toxins. *C. difficile* is an anaerobic, spore-forming bacillus that can colonize the gut asymptomatically under normal conditions. Antibiotics (especially broad-spectrum agents) disturb the commensal bacteria that normally provide "colonization resistance", creating an environment conducive to the germination of *C. difficile* spores and vegetative growth [7]. Spores, which are ubiquitous in hospitals and can survive routine disinfection, reach the colon (often via the fecal-oral route) and remain dormant until they sense favorable conditions such as elevated levels of certain gut metabolites that accumulate after microbiota disruption [8, 9].

Once germinated, vegetative *C. difficile* produces exotoxins that injure the colonic epithelium. The two primary toxins, toxin A (TcdA) and toxin B (TcdB), inactivate Rho-family GTPases, leading to cytoskeletal disaggregation, loss of tight junctions, and massive inflammation of the mucosa [10]. The result is the characteristic pseudomembrane: a layer of necrotic debris, fibrin and neutrophils overlying the damaged mucosa.

2.2 Surgical factors

Surgery itself may contribute to pathogenesis through various mechanisms. Major abdominal operations often require perioperative antibiotic prophylaxis, which can precipitate CDI. Prolonged postoperative ileus can lead to fecal stasis and higher intraluminal toxin concentrations. Postoperative immune regulation, encompassing stress response and transfusions, together with the widespread use of acid-suppressing medications in postoperative patients for stress ulcer prophylaxis, may increase vulnerability [11]. Indeed, surgery is recognized as a risk factor for developing CDI, even as surgery can be a necessary intervention for treating severe CDI [12]. In children, abdominal surgeries (*e.g.*, for oncology or transplant) often coincide with intensive antibiotic exposure and immunosuppression, compounding the risk [13].

2.3 Host susceptibility

Not all patients exposed to *C. difficile* develop colitis—host factors are critical. Advanced age is one of the strongest risk factors in adults, as the elderly have diminished microbiome diversity and immune defenses; over 80% of fatal CDI cases occur in patients >65 years [12]. Comorbid conditions such as inflammatory bowel disease (IBD), end-stage renal disease, diabetes or malignancy increase risk, as does any immunodeficiency (including corticosteroid use or chemotherapy) [12]. Hypoalbuminemia and malnutrition have also been associated with more severe CDI in surgical patients [4].

Patients with IBD face a heightened risk of CDI, attributed to factors such as frequent antibiotic use, immunosuppressive therapies and underlying mucosal inflammation [14–16]. A systematic review and meta-analysis by Balram *et al.* [14] identified antibiotic exposure, biologic therapy, and colonic involvement as significant risk factors for CDI in IBD patients, with associated increases in both short- and long-term mortality. Additionally, corticosteroid use and active colonic inflammation have been recognized as modifiable risk factors, emphasizing the need for vigilant infection control and tailored therapeutic strategies [14–16]. Management complexities arise due to overlapping symptoms of IBD flares and CDI, necessitating prompt and accurate diagnosis.

Pediatric patients present a somewhat different risk profile: infants <1 year frequently carry *C. difficile* without illness (colonization rates >40% in infants) [17], presumably due to lack of toxin receptors and immature immune response. Thus, infants can harbor the organism but rarely manifest pseudomembranous colitis [18]. Severe CDI in children tends to occur in those with significant comorbidities (*e.g.*, cancer, organ transplant or complex chronic illnesses) who have disruption of gut flora from antibiotics or prolonged hospitalization [19].

Collectively, postoperative pseudomembranous colitis arises from the convergence of antibiotic-mediated dysbiosis, exposure to *C. difficile* (often in the healthcare environment), and host vulnerabilities. This synergy is especially pronounced in hospitalized surgical patients who receive broad-spectrum antibiotics. The incidence and risk factors for postoperative CDI are listed in Table 1 (Ref. [3–5, 12, 19–21]).

3. Diagnostic challenges and tools

Diagnosing postoperative pseudomembranous colitis can be challenging due to overlapping postoperative symptoms and limitations of diagnostic tests. Surgical patients may have postoperative ileus or diarrhea from other causes (*e.g.*, enteral feeding, medications), making clinical recognition more challenging. A high index of suspicion is needed for any post-abdominal surgery patient with unexplained diarrhea

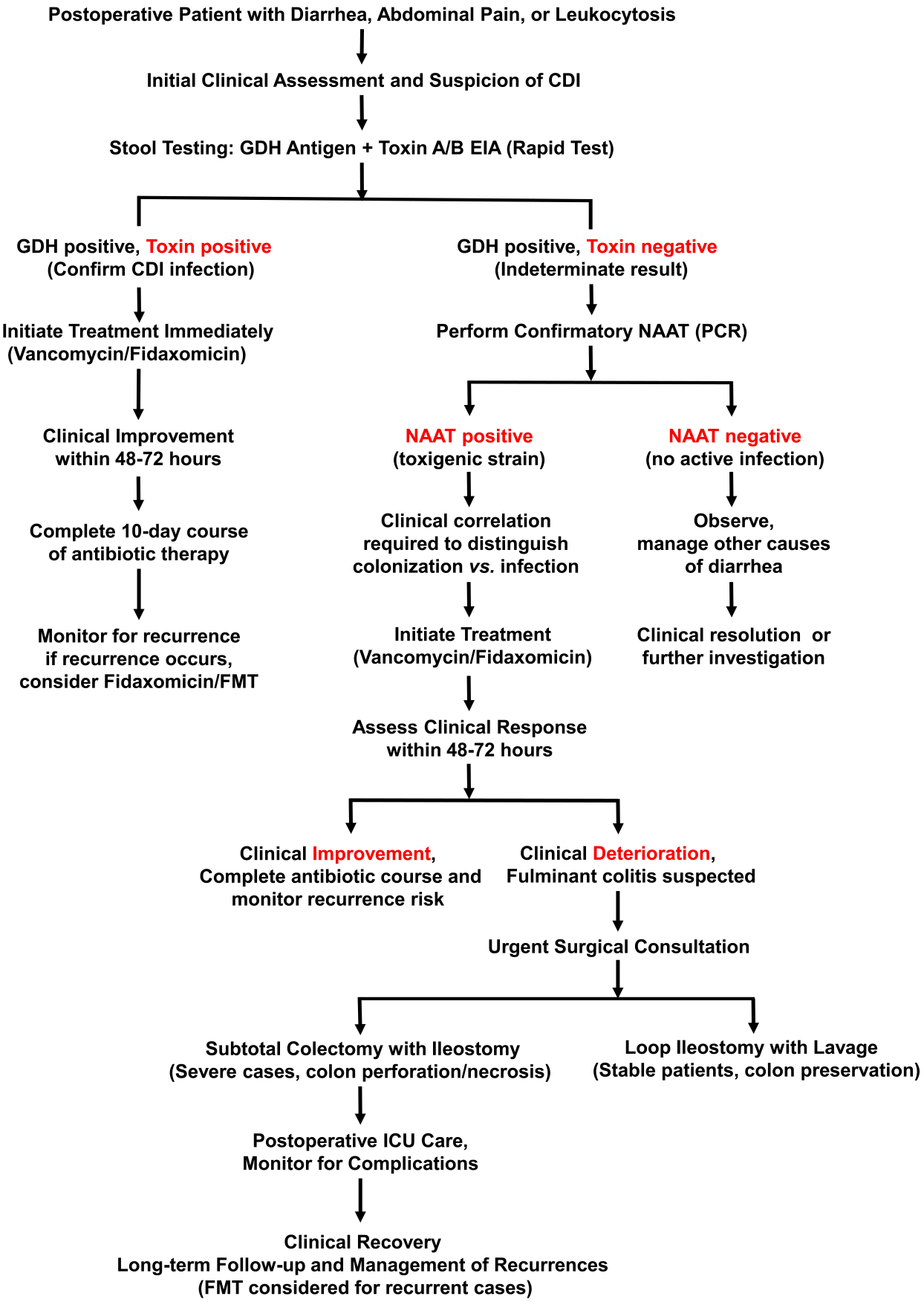

FIGURE 1. The illustration of the diagnostic workup and management pathway for postoperative pseudomembranous colitis. CDI: *Clostridioides difficile* infection; GDH: Glutamate dehydrogenase; EIA: Enzyme immunoassay; NAAT: Nucleic acid amplification test; PCR: Polymerase chain reaction; ICU: Intensive care unit; FMT: Fecal microbiota transplantation.

TABLE 1. Incidence and risk factors for postoperative CDI.

Population	Incidence	Major risk factors
Adults	0.3–0.4% of abdominal operations [3] 1.0% after colorectal surgery [4]	• Age >65 years [12] • Recent antibiotics, including clindamycin, cephalosporins, fluoroquinolones [12] • High surgical severity: ASA ≥4, emergency surgery [3, 4] • Preoperative infection or sepsis [3] • Immunosuppression [12] • Comorbidities (IBD, renal failure, malignancy) [12] • PPI use [12]
Pediatrics	0.3% of pediatric surgeries [20] 9 per 100,000 pediatric inpatients (HA-CDI) [21]	• Recent antibiotics (OR ~2×) [19] • Prolonged hospitalization (OR ~14×) [19] • Prior hospitalization (OR ~3.7×) [19] • Immunodeficiency (OR ~4×) or cancer (OR ~3×) [19] • GI surgery or transplantation [19] • Acid suppressants (OR ~2×) [19] • Age <2 years: high colonization rates [5]

ASA: American Society of Anesthesiologists; IBD: Inflammatory bowel disease; PPI: Proton pump inhibitor; OR: Odds ratio; GI: Gastrointestinal; HA-CDI: Healthcare-Associated Clostridioides difficile Infection.

(especially if profuse and foul-smelling), abdominal pain or leukocytosis—particularly if they received antibiotics [22]. Distinguishing *C. difficile* colonization from active infection is another key challenge, as hospitalized patients (and young children) can carry *C. difficile* without disease [23]. The diagnostic tools for *C. difficile* and their performance are listed in Table 2 (Ref. [10, 23]).

3.1 Stool toxin testing

The gold standard for confirming pseudomembranous colitis is detecting *C. difficile* toxin in stool [24]. The most common method is enzyme immunoassay (EIA) for toxins A and B. Toxin EIAs are rapid (results in hours) and highly specific (approximately 92–98%), but lack sensitivity [23, 25]. A meta-analysis found that toxin EIAs detect only ~75% of CDI cases (sensitivity ~53–85% in various studies) [21]. Thus, a negative toxin EIA does not definitively rule out CDI, especially in a high-risk clinical scenario [24, 26]. Because of this, many laboratories now use a multi-step algorithm: an initial screening test with high sensitivity, followed by a confirmatory toxin assay [24, 26].

3.2 GDH antigen and multi-step algorithms

C. difficile produces the enzyme glutamate dehydrogenase (GDH) in high quantities. GDH antigen EIAs serve as a sensitive initial screen for the presence of *C. difficile* organism (both toxigenic and non-toxigenic strains) [27, 28]. Reported GDH test sensitivity ranges from ~80% up to 100%, with specificity ~83–100% [23]. In practice, GDH is often combined with a toxin EIA: if GDH is negative, CDI is effectively ruled out (negative predictive value ~99%) [23]. If GDH is positive but toxin EIA is negative, a follow-up nucleic acid amplification test is typically performed to clarify infection versus colonization [29, 30].

3.3 Nucleic acid amplification tests (NAAT)

PCR-based tests (*e.g.*, real-time PCR for the *C. difficile* toxin B gene) are highly sensitive and increasingly used. NAAT can detect toxigenic *C. difficile* with sensitivity often >90% and specificity ~95–99% relative to culture [10]. A positive NAAT indicates the patient harbors a toxigenic strain, but it does not prove that active toxin production is causing disease at that moment [10]. Patients who are colonized may be NAAT-positive but toxin-negative [31]. For this reason, experts recommend NAAT be used as part of a two-step approach or that clinicians interpret NAAT results in context—only test patients with a compatible clinical picture, and avoid treating colonization [23]. In postoperative patients with ileus who cannot produce stool, PCR assay on colonic contents (via enema or during endoscopy) can sometimes secure the diagnosis.

3.4 Endoscopic diagnosis

Flexible sigmoidoscopy or colonoscopy can directly visualize pseudomembranes on the colonic mucosa, which is virtually pathognomonic for *C. difficile* colitis. Finding raised yellow plaques (pseudomembranes) on endoscopy confirms the diagnosis. However, endoscopy is not routinely required for diagnosis and carries risk (including perforation in an inflamed colon). Its sensitivity is also limited—pseudomembranes may be patchy or absent in early infection or mild cases. Endoscopic evaluation is most useful in fulminant cases requiring rapid diagnosis when stool tests are delayed or impractical [32].

3.5 Pediatric considerations

In children under 2 years, routine diagnostic testing for *C. difficile* is discouraged because of high colonization rates. A positive test in an infant is difficult to interpret and often does not indicate true disease [5]. For older children with post-surgical diarrhea, the same stool tests are used as in adults, but

TABLE 2. Diagnostic tools for *C. difficile* and performance.

Diagnostic test	Sensitivity	Specificity	Notes
GDH Antigen EIA	80–100%	83–100%	Detects *C. difficile* antigen; high NPV; initial screen; needs confirmatory toxin test [23]
Toxin A/B EIA	50–85%	91–98%	Rapid and specific for toxin; lower sensitivity [23]; confirms active toxin production
NAAT (PCR)	90–95+%	95–99%	Highly sensitive; detects toxin genes; can detect colonization; best in two-step algorithm [10]
Cell Cytotoxicity Assay	94–100%	99–100%	Historical gold standard; very sensitive/specific but slow (24–48 h); primarily research [10]
Endoscopy	50–60% for pseudomembranes	97% for pseudomembranes	Direct visualization; high specificity; limited sensitivity; useful if stool tests inconclusive; invasive [10]

GDH: Glutamate dehydrogenase; EIA: Enzyme immunoassay; NAAT: Nucleic acid amplification test; PCR: Polymerase chain reaction; NPV: Negative predictive value.

with caution to correlate with symptoms. Pediatric labs often require a child to have significant diarrhea plus risk factors before testing, to avoid over-diagnosis [33, 34].

In practice, an optimal diagnostic approach in a postoperative patient is to promptly send stool for a multi-step assay at the first suspicion of CDI. Most hospitals now employ an algorithm combining GDH and toxin EIA, with reflex NAAT, to balance sensitivity and specificity [10]. Rapid diagnosis allows early therapy and infection control measures to prevent spread [12].

4. Antibiotic stewardship and preventive strategies

Preventing postoperative pseudomembranous colitis hinges on prudent antibiotic use and infection control practices. Antibiotic stewardship is paramount: antibiotics are the single most important modifiable risk factor for CDI, so optimizing their use can greatly reduce incidence [35]. Hospitals that have implemented robust antimicrobial stewardship programs have observed significant declines in healthcare-associated CDI rates [35]. Key stewardship principles for surgeons and perioperative clinicians include:

4.1 Limit broad-spectrum use

Avoid unnecessary broad-spectrum antibiotics, and tailor prophylactic and therapeutic antibiotics to the narrowest effective spectrum. Certain antibiotics carry especially high CDI risk—notably clindamycin, third-generation cephalosporins, fluoroquinolones, carbapenems and broad-spectrum penicillins [12, 19]. In one meta-analysis, third-generation cephalosporins were identified as the highest-risk class in hospitalized patients [36]. Surgeons should reserve these agents for clear indications and use alternative or narrower agents when possible.

4.2 Optimize prophylaxis duration

Perioperative prophylactic antibiotics should be given for the recommended short duration (usually a single preoperative dose, or <24 hours in most cases) [37, 38]. Prolonging prophylaxis "just in case" provides little benefit but substantially increases CDI risk [36]. Even a 2–3 days extension of broad prophylaxis can elevate the risk of CDI, as patients receiving >48 hours of coverage had significantly higher CDI rates than those de-escalated at 48 hours [36].

4.3 De-escalate and target therapy

In postoperative infections, obtain cultures and narrow therapy based on sensitivities. One study found that patients with bloodstream infections who were de-escalated from broad empiric therapy within 48 hours had markedly lower CDI rates than those kept on broad agents longer [36]. For surgical patients who do require antibiotics, regularly reassess the regimen and stop or step down therapy as soon as it is safe.

4.4 Avoid redundant antibiotics

Surgeons sometimes prescribe dual anaerobic coverage or unnecessary combinations (*e.g.*, metronidazole plus carbapenem)—these practices should be eliminated to reduce microbiome harm [39, 40]. Likewise, avoid treating non-infectious postoperative conditions with antibiotics. Each unnecessary antibiotic course can disrupt the gut flora for weeks, maintaining susceptibility to CDI for up to three months following exposure [19].

4.5 Minimize other modifiable risks

Restrict proton pump inhibitor use to clear indications in postoperative patients. Gastric acid suppression has been associated with increased CDI risk [12]. Despite ongoing debates over causation, it is prudent to deprescribe unneeded proton pump inhibitors in hospitalized patients, particularly those concurrently receiving antibiotics [41, 42]. Additionally, rigorous hand hygiene and environmental cleaning in surgical wards reduce spore transmission. *C. difficile* spores are not killed by alcohol-based hand rubs, therefore, handwashing with soap and water is required after caring for CDI patients [12].

4.6 Infection control in the operating rooms and wards

Operating rooms and surgical wards should enforce infection control protocols for CDI. If a patient is known to have pseudomembranous colitis, they should be placed on contact precautions. After surgery, thorough disinfection (using sporicidal agents like bleach on surfaces) is necessary, as spores can contaminate the environment and equipment. In pediatric surgical patients, antibiotic stewardship is equally important. Many children developing CDI have received multiple antibiotic courses [43, 44]. Pediatric stewardship programs focusing on limiting broad-spectrum cephalosporins and clindamycin have shown reduction in CDI rates in children [19].

5. Management and clinical outcomes

Management of pseudomembranous colitis requires timely initiation of effective therapy to eradicate *C. difficile* and supportive care to address fluid losses and inflammation. Treatment must also be tailored to disease severity—ranging from oral antibiotics in mild cases to urgent surgery in cases of life-threatening fulminant colitis. The comparative outcomes by treatment strategy are listed in Table 3 (Ref. [6, 17, 32, 45, 46]).

5.1 Medical therapy: antibiotics for *C. difficile*

5.1.1 Vancomycin and fidaxomicin

Oral vancomycin has long been the first-line therapy for CDI, and fidaxomicin (a newer narrow-spectrum macrocyclic antibiotic) is now recommended as an equal or superior first-line agent in many guidelines [7]. Both antibiotics achieve high concentrations in the colon and are poorly absorbed [47, 48]. For an initial episode of pseudomembranous colitis, a 10-day course of either vancomycin (125 mg four times daily) or fidaxomicin (200 mg twice daily) is indicated [7]. These treatments result in clinical cure rates of 80–90% in clinical trials [45].

Multiple trials have compared vancomycin *vs.* fidaxomicin: initial cure rates are generally equivalent [45], but fidaxomicin significantly reduces recurrence after treatment. A recent 2024 meta-analysis found that fidaxomicin reduced the 30–90 days recurrence risk by roughly 40–60% relative to vancomycin [45]. For example, the 40-day recurrence rate was ~19% with vancomycin versus ~10% with fidaxomicin (relative risk (RR) = 0.52) [45]. This is attributed to fidaxomicin's more selective eradication of *C. difficile* (sparing much of the normal flora) leading to less microbiome disruption [7].

In severe CDI, both drugs are effective, but some data suggest vancomycin may have a slight edge in fulminant cases. Despite this, fidaxomicin was associated with a lower all-cause mortality at 60 days compared to vancomycin (5.9% *vs.* 10.3%, RR 0.57) [45]. Current guidance favors fidaxomicin as a first-line treatment for non-fulminant CDI when available [7], while vancomycin remains a standard option and is often preferred in fulminant cases.

5.1.2 Metronidazole

Metronidazole was once a mainstay for mild-to-moderate CDI, but is no longer recommended as first-line treatment in adults [7]. Trials showed oral metronidazole had inferior cure rates (~70%) compared to vancomycin in severe CDI, and even in non-severe cases it was somewhat less effective [49]. It is now reserved for situations where vancomycin or fidaxomicin are not available, or may be used in combination (intravenous metronidazole added to oral vancomycin) for fulminant CDI [50]. In children, some guidelines still allow metronidazole for an initial mild episode, but pediatrics is shifting toward vancomycin as first-line therapy [5].

TABLE 3. Comparative outcomes by treatment strategy.

Treatment strategy	Key outcome findings
Vancomycin *vs.* Fidaxomicin	• Initial cure: Similar (85–90%) [45] • Recurrence: Fidaxomicin superior (10% *vs.* 19% with vancomycin, RR 0.52) [45] • Mortality: Fidaxomicin showed lower 60-day mortality (5.7% *vs.* 10%) [45] • Severe CDI: Vancomycin may have slight edge in fulminant cases [45]
Surgery *vs.* Medical Management for Fulminant CDI	• 25% of fulminant CDI patients require surgery [45] • Medical-only management in fulminant cases: up to 80% mortality • With surgery: 30% 30-day mortality [6] • Early surgery improves survival; operating before vasopressor-dependence has better outcomes [32] • Postoperative complications in 75% of survivors [32]
Loop Ileostomy + Lavage *vs.* Total Colectomy	• Loop ileostomy with lavage: 17.2% mortality *vs.* 39.7% with total colectomy ($p = 0.002$) [32] • NSQIP analysis: Partial colectomies similar mortality to total colectomy (~30%) [6] • Loop ileostomy preserves colon; potential for future reversal [32]
FMT *vs.* Antibiotics Alone	• Recurrent CDI: FMT success 80–90% [46] • Severe/Fulminant CDI: FMT program reduced mortality from 21.3% to 9.1% ($p = 0.015$) [46] • Colectomy rates dropped from 15.7% to 5.5% after FMT implementation [46] • Pediatric use: ~90% cure rates, comparable to adults [17]

CDI: Clostridioides difficile infection; FMT: Fecal microbiota transplantation; RR: Relative risk; NSQIP: National Surgical Quality Improvement Program.

5.1.3 Adjunctive support

All patients with pseudomembranous colitis need supportive care—aggressive fluid and electrolyte repletion (due to diarrheal losses), and avoidance of anti-motility drugs that could retain toxins. In moderate-to-severe cases, monitoring for complications like dehydration, renal failure or toxic megacolon is crucial. Emerging adjuncts include bezlotoxumab, a monoclonal antibody against toxin B that can halve the recurrence rate in high-risk patients [51].

5.1.4 Outcomes of medical therapy

With prompt antibiotic treatment, most patients improve within 48–72 hours. Successful initial cure is expected in >80%. The major outcome concern is recurrent infection, which occurs in about 20–25% of cases after a first episode even with appropriate therapy [7]. Recurrent CDI can be challenging: after one recurrence, the risk of further recurrences increases (up to 40–60% after two or more episodes) [7]. In terms of mortality, uncomplicated CDI has a low attributable mortality in modern series (<2% in postoperative cases) [3]. However, if CDI progresses to fulminant colitis or occurs in a frail host, mortality can be significant. Reassuringly, in children, CDI is rarely fatal [13], highlighting that outcomes in otherwise healthy children are generally good with therapy.

5.2 Surgical management: when and what to operate

Surgery becomes necessary in pseudomembranous colitis when the disease is fulminant or refractory to medical therapy. Fulminant CDI is defined by hypotension, shock, ileus or megacolon [52, 53]. Approximately 1 in 4 patients with fulminant CDI will require surgical intervention despite maximal medical therapy [36]. The classical surgical procedure is a subtotal colectomy with end ileostomy, removing the diseased colon as a source of toxin and sepsis.

Colectomy for fulminant CDI is life-saving but carries a high risk—in modern cohorts, 30-day mortality after emergency colectomy for fulminant CDI remains around 30% [6]. This high mortality reflects the critical condition of patients who come to surgery (often with multi-organ failure). However, without surgery, mortality approaches 80–100% once fulminant toxic megacolon with perforation has developed [32]. Early surgical consultation is therefore imperative at the first signs of fulminant colitis [36]. Indications for surgery include diffuse peritonitis, colon dilation >8 cm (toxic megacolon), or clinical deterioration despite 24 to 48 hours of maximal medical therapy [32].

5.2.1 Subtotal colectomy *vs.* diverting ileostomy

In the last decade, an alternative surgical approach has emerged—a diverting loop ileostomy with colonic lavage, sometimes called the "loop ileostomy and vancomycin flush" procedure [54, 55]. This involves leaving the colon in place but diverting fecal stream at the ileum, and intraoperatively irrigating the colon with polyethylene glycol solution, then instilling vancomycin flushes via the ileostomy postoperatively [32]. A retrospective study of 98 fulminant CDI patients found mortality ~17% in those treated with loop ileostomy *vs.* ~40% in those undergoing total colectomy [55]. In a National Surgical Quality Improvement Program (NSQIP) analysis, partial colectomies had no worse mortality than total colectomy (~30% in both groups) [56], suggesting that in some fulminant cases a limited resection might suffice.

Current guidelines still consider subtotal colectomy with ileostomy the standard for fulminant colitis with shock [32], especially if there is colon perforation or necrosis [32]. But in experienced centers, loop ileostomy with lavage is an acceptable alternative for appropriate candidates [32]. Surgeons should individualize the approach based on patient stability and disease extent—the key is to intervene early with whichever procedure can be performed safely.

5.2.2 Surgery in pediatric CDI

It is worth noting that surgery for *C. difficile* is exceedingly uncommon in children. Pediatric fulminant colitis is rare, and few cases require colectomy—many centers have never had to perform CDI-related colectomy in a young child [13]. When fulminant colitis does occur in a child (usually an immunocompromised patient), the same principles of early surgery apply, though data are limited to case reports.

5.2.3 Fecal microbiota transplantation (FMT)

FMT has revolutionized the management of recurrent CDI and is now being explored for severe cases as well. FMT involves instilling processed stool from a healthy donor into the patient's colon, aiming to restore a balanced microbiome. For recurrent CDI (two or more recurrences), FMT yields ~80–90% cure rates, often succeeding when antibiotics fail [46].

In the context of fulminant CDI, recent evidence indicates FMT can be a life-saving adjunct. A large single-center study implementing an early FMT program for refractory severe/fulminant CDI found that hospital mortality dropped significantly, from 10.2% to 4.4% overall (p = 0.02) [46]. Among patients with fulminant CDI, mortality decreased from 21.3% to 9.1% (p = 0.015) [46]. Additionally, the need for colectomy was reduced by two-thirds [46]. These outcomes suggest that, in centers with expertise, FMT can serve as an alternative or bridge to surgery in fulminant cases. The FDA approved a standardized oral microbiota product in 2022 for prevention of recurrent CDI. In pediatric patients, FMT is increasingly used for recurrent CDI and has shown similar success rates and safety as in adults [57, 58].

5.2.4 Prognosis and long-term outcomes

With appropriate treatment, the majority of postoperative CDI patients recover fully. However, the illness can prolong hospitalization substantially. The NSQIP data indicated an average increase in hospital stay of 4–6 days in surgical patients who developed CDI [3]. CDI during a surgical episode also correlates with higher readmission and reoperation rates [3]. One study showed CDI was associated with a 10-fold higher odds of unplanned readmission after surgery [59].

Recurrence is the predominant unfavorable event subsequent to first treatment. Each recurrence should be addressed promptly, often with fidaxomicin if it has not been previously

administered, or consider fecal microbiota transplantation after numerous recurrences. The mortality rate in pseudomembranous colitis is predominantly influenced by cases of fulminant illness. Survivors with fulminant CDI who have had surgery have a challenging recovery, frequently involving an ICU admission and the temporary necessity of an ileostomy.

Pediatric patients generally have excellent outcomes; recurrence rates in children are similar (20–30%), but with proper treatment, long-term recovery is expected and children usually regain normal growth and health [5]. Deaths are extremely rare in pediatric CDI, in contrast to the significant mortality seen in older adults [13].

Furthermore, while current approaches effectively address postoperative CDI, exploring personalized microbiome assessments prior to elective abdominal surgery could represent a novel strategy. Preoperative identification of microbiome vulnerabilities using rapid sequencing techniques might allow tailored probiotic supplementation or selective antibiotic prophylaxis to minimize microbiota disruption and prevent CDI [60, 61]. Additionally, integrating predictive analytics and artificial intelligence to stratify surgical patients by individual CDI risk could facilitate targeted monitoring and early intervention. Another innovative suggestion is the exploration of microbiome-enhancing dietary modifications in perioperative protocols, potentially providing a non-pharmacological preventive measure [62, 63]. Such microbiome-oriented strategies, combined with existing infection control practices, could substantially enhance CDI prevention, representing a critical evolution from current standardized guidelines.

5.2.5 Limitations of the review

One limitation of this review is its reliance primarily on previously published studies and clinical guidelines, resulting in limited original insights or novel clinical data. The discussion is broad and comprehensive but lacks detailed subgroup analyses that could further clarify CDI management nuances, particularly in specific high-risk populations such as immunocompromised individuals or those with IBD. Additionally, the rapid pace of emerging microbiome research means some recent developments, such as the newly approved live biotherapeutic products like Rebyota, are only briefly addressed and may require future updates. Furthermore, the review does not extensively explore health-economic aspects or patient-reported outcomes, leaving opportunities for further studies to better assess the real-world effectiveness and cost-benefit balance of various prevention and treatment strategies.

6. Conclusions

Postoperative pseudomembranous colitis due to *C. difficile* is a potentially serious complication in both adult and pediatric surgical patients. Surgeons should maintain awareness that even routine perioperative antibiotics can precipitate life-threatening colitis. Early recognition—facilitated by understanding risk factors such as recent antibiotic use, advanced age, and comorbid conditions—and prompt diagnostic testing are critical to initiating life-saving therapy. Advances in diagnostics (like multi-step stool assays) have improved our ability to confirm CDI quickly, even as differentiating colonization from infection remains a challenge in some cases.

A strong emphasis on antibiotic stewardship in the perioperative period is essential to prevent CDI; judicious use of prophylactic antibiotics and early de-escalation of therapy can significantly reduce incidence. The management of pseudomembranous colitis has evolved, with fidaxomicin and fecal microbiota transplantation emerging as valuable tools that improve outcomes by reducing recurrences and even lowering mortality in severe cases. Most postoperative CDI can be managed medically with oral vancomycin or fidaxomicin, leading to cure in the majority of patients. However, in fulminant colitis unresponsive to medical therapy, prompt surgical intervention is vital and can be life-saving, albeit the associated significant risk. Novel surgical approaches that preserve the colon show promise in improving survival and quality of life for these patients.

Outcomes for postoperative pseudomembranous colitis are improving thanks to heightened vigilance and new therapies. Still, the condition carries substantial morbidity—increased length of stay, higher readmission rates, and in severe cases, notable mortality and an impact on postoperative recovery. Multidisciplinary care is key: surgeons, infectious disease specialists, and critical care teams must coordinate to optimize treatment. In pediatric cases, outcomes are generally favorable, but careful diagnostic consideration is needed due to high background rates of *C. difficile* colonization in young children.

In conclusion, postoperative pseudomembranous colitis exemplifies the delicate balance between necessary surgical antibiotic use and unintended infectious consequences. By focusing on prevention through stewardship, utilizing accurate diagnostics, and applying effective medical or surgical therapies tailored to disease severity, surgeons can dramatically mitigate the impact of this complication. Ongoing research into microbiome-based treatments and optimal surgical techniques holds promise for further reducing the burden of this disease in surgical populations. With vigilance and evidence-based management, most patients, adults and children alike, can overcome *C. difficile* colitis and successfully continue on their recovery from surgery.

7. Recommendations

To reduce the risk of postoperative CDI, surgical teams should prioritize meticulous antibiotic stewardship and stringent infection control measures. Antibiotic prophylaxis should be carefully tailored, favoring narrow-spectrum agents administered at minimal effective durations, ideally limited to a single preoperative dose or less than 24 hours postoperatively. Avoiding prolonged broad-spectrum antibiotics and redundant anaerobic coverage significantly minimizes microbiome disruption and subsequent CDI risk. Furthermore, routine reassessment of antibiotic therapy is crucial, allowing timely de-escalation based on clinical response and microbiological data. Proton pump inhibitors, commonly used for stress ulcer prophylaxis, should be prescribed judiciously due to their association with increased CDI risk, and unnecessary usage must be actively curtailed. Rigorous hand hygiene, particularly handwashing with soap and water, remains essential as alcohol-based sanitizers are ineffective against *C. difficile* spores. Environ-

mental cleaning protocols utilizing sporicidal agents should be strictly enforced in operating rooms and surgical wards. In addition, early suspicion and prompt diagnosis with multistep stool testing algorithms can facilitate timely initiation of therapy, thereby improving patient outcomes and preventing nosocomial spread. The diagram for diagnostic workup and management pathway for postoperative pseudomembranous colitis is illustrated in Fig. 1.

Furthermore, future research should focus on identifying individualized microbiome markers predictive of CDI risk to enable personalized prophylactic interventions. Prospective trials evaluating preoperative microbiota modulation via targeted probiotics or dietary strategies could illuminate new preventive measures. Clinical trials indicate that Rebyota is safe and effective, even in patients with IBD [64]. Additionally, probiotics such as *Saccharomyces boulardii* and *Lactobacillus species* have been explored for CDI prevention. While some studies suggest potential benefits, the evidence remains mixed, and further research is needed to establish their efficacy and optimal use [64]. Additionally, studies integrating artificial intelligence and advanced analytics to develop predictive algorithms for CDI in surgical patients may enhance early identification and tailored management, ultimately reducing morbidity, mortality and healthcare resource utilization associated with postoperative CDI.

ABBREVIATIONS

CDI, *Clostridioides difficile* infection; EIA, Enzyme immunoassay; FMT, Fecal microbiota transplantation; GDH, Glutamate dehydrogenase; IBD, Inflammatory bowel disease; ICU, Intensive care unit; NAAT, Nucleic acid amplification test; NPV, Negative predictive value; PCR, Polymerase chain reaction; PPI, Proton pump inhibitor; ASA, American Society of Anesthesiologists; OR, Odds ratio; GI, Gastrointestinal; HA-CDI, Healthcare-Associated Clostridioides difficile Infection; RR, Relative risk; NSQIP, National Surgical Quality Improvement Program.

AVAILABILITY OF DATA AND MATERIALS

Not applicable.

AUTHOR CONTRIBUTIONS

CHL—Writing. TLL—Data curation. CYH—Literature review and proof reading. CHH—Supervision and study design. All authors read and approved the final manuscript.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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