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Fecal Calprotectin and Treatment Strategies in Ulcerative Colitis

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Abstract:

Ulcerative colitis (UC) is a long-term inflammatory condition that causes the intestinal mucosa to become inflamed over and over again. To treat it, you need a planned and goal-oriented approach. The major goal of therapy is to achieve long-term steroid-free remission while lowering the adverse effects and problems that come with treatment and the condition. Topical and oral 5-aminosalicylic acid (5-ASA) formulations are used for mild cases, whereas systemic corticosteroids, immunomodulators, and biologic therapies are utilized for moderate to severe cases. Consequently, these treatment strategies are dictated by the severity, scope, and clinical trajectory of the disease. Patients with refractory illness may need advanced therapies, such as calcineurin inhibitors (like cyclosporine or tacrolimus) or biologic medications (like infliximab), which are antibodies that fight tumor necrosis factor-alpha. Surgery is still needed for problems like toxic megacolon, perforation, or severe refractory hemorrhage. To get the best outcomes from this kind of treatment, intestinal inflammation has to be precisely measured.

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Faecal calprotectin has emerged as a reliable, non-invasive biomarker for intestinal inflammation in recent years. It is very useful in medicine for keeping track of illnesses, predicting relapses, and evaluating how well treatments are working. Integrating it into clinical practice allows for quick changes to treatment and better disease activity evaluation. Including fecal calprotectin in clinical decision-making makes it feasible to have a more personalized and proactive treatment plan. This makes it possible to use faecal calprotectin-guided approaches that cut down on the necessity for invasive procedures like colonoscopy. Because of this, faecal calprotectin is very important for better managing UC, improving the results of the disease, and making treatment decisions that follow approved global standards.

Keywords: Ulcerative colitis, calprotectin, inflammatory bowel disease, azathioprine.

Introduction:

Ulcerative colitis (UC) is a long-term inflammatory illness that causes the intestinal mucosa to become irritated again and over again. The inflammatory process often begins in the rectum and progresses continuously proximally across the colon. Patients with severe sickness may have endoscopic symptoms of "backwash ileitis," but the terminal ileum is often preserved. A tailored approach to diagnosis and therapy is essential because of the considerable variability in sickness progression and severity across individuals. Clinical practice guidelines are developed in this setting based on the most robust scientific evidence to identify optimal strategies for illness management. [(1)] There are 48 potential disease states that need to be considered before making evidence-based recommendations. This shows how complicated ulcerative colitis is and how many different clinical outcomes are conceivable. Some examples of these include remission, mild, moderate, or severe disease activity, asymptomatic states following an initial flare-up, rising disease severity, chronic symptoms, and repeated episodes. Furthermore, accounting for significant patient-specific factors including as age, comorbidities, surgical history, pharmaceutical intolerances, lifestyle, personal preferences, and extraintestinal symptoms may provide over a thousand potential treatment strategies. [(2)]

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Instead, then forcing everyone to use the same technique, professional recommendations are supposed to be followed in a flexible way, with suggested treatment options. Several national and international entities, including the European Crohn's and Colitis Organization (ECCO), the American College of Gastroenterology (ACG), and the Deutsche Gesellschaft für Verdauungs- und Stoffwechselkrankheiten (DGVS), have formulated consensus recommendations that constitute the foundation of modern clinical practice and are supported by expert opinion. The Oxford Centre for Evidence-Based Medicine has looked at these suggestions and given them levels of evidence (EL) and recommendation grades (RG). EL 1 is the lowest level of evidence, while RG A is the highest recommendation. In the ACG and DGVS grading system, recommendations are scored from A (highest) to D (lowest). The primary objective of medical therapy for ulcerative colitis (UC) under this structured therapeutic framework is to swiftly achieve steroid-free remission while mitigating complications associated with the condition and its treatment. Current guidelines for UC still support a pyramidal step-up plan, even if there is still a lot of debate over whether a "top-down" approach, which involves using extremely strong drugs early on to prevent complications, is the best way to go.

In this model, corticosteroids and immunomodulators are used to make therapy more successful, although 5-aminosalicylic acid (5-ASA) is still the major drug used. If medication doesn't work or the person is really sick, they may need rescue therapies such calcineurin inhibitors (like tacrolimus and cyclosporine A [CsA]), infliximab (IFX), or surgery. [2–3] Faecal calprotectin has emerged as the paramount noninvasive biomarker in ulcerative colitis for assessing disease activity and refining treatment strategies. It is necessary to assess the level of inflammation, evaluate therapy efficacy, and predict clinical recurrence. Calprotectin is a 36-kDa protein that belongs to the S100 family of proteins that bind calcium. There is a central hinge region, two EF-hand motifs (helix-loop-helix structures), and distinct N- and C-terminal domains. "S100" is the name for its ability to dissolve in 100% ammonium sulphate at a neutral Ph. [4] Calprotectin serves several biological roles, including competitive inhibition of zinc-dependent enzymes, antibacterial biostatic effects by zinc ion chelation, induction of apoptosis in malignant cells, and regulation of inflammatory responses. These traits show that it may be used as a sensitive

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diagnostic for intestinal inflammation. [5] Faecal calprotectin is a valuable clinical instrument for monitoring disease activity, assessing treatment efficacy, differentiating functional disorders such as irritable bowel syndrome (IBS) from inflammatory bowel disease (IBD), predicting disease recurrence, and potentially reducing the necessity for invasive endoscopic interventions. [6] Therefore, an essential component of modern, personalized, and biomarker-driven ulcerative colitis management strategies is its integration into clinical decision-making.

Management of ulcerative colitis:

People with ulcerative colitis (UC) typically suffer stomach discomfort and diarrhea that has blood or mucus in it, no matter whether they have just been diagnosed or are having flare-ups again. In extreme cases, systemic symptoms such as tachycardia, hypoglycemia, diarrhea, and anemia may manifest. Before starting medical treatment, it is very important to rule out other possible causes of colitis or enteritis. These could be infections (like cytomegalovirus [CMV] or Clostridium difficile), drug toxicity (like antibiotics or nonsteroidal anti-inflammatory drugs [NSAIDs]), mesenteric ischaemia, or intestinal cancer. Before raising the dose of medicine, it is extremely essential to rule out opportunistic infections like CMV, especially in those who are on immunosuppressive therapy and not responding to corticosteroids.[7]

For UC, a comprehensive but minimal diagnostic evaluation is essential due to the absence of a definitive gold standard for diagnosis. This includes a full clinical exam with a focus on signs outside the intestines, as well as a full blood count, an erythrocyte sedimentation rate (ESR), a C-reactive protein (CRP), a stool microbiological investigation, an abdominal ultrasound, and an endoscopic evaluation with mucosal biopsies. In emergencies, it is best to wait and conduct the endoscopic and histological tests again if the diagnosis is still not obvious. When deciding on treatment, you should think about the patient's preferences, past medication history, possible side effects, disease activity, course, frequency of relapses, extraintestinal manifestations, and how far the disease has spread (proctitis, left-sided colitis, or extensive colitis).[7]

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The Montreal categorisation system is a common way to group illnesses by their activity and severity. This makes it easier to compare diseases and plan therapy. Remission (S0) is characterized by no more than three stools per day without blood and no urgency; mild disease (S1) is characterized by occasional bloody stools (≤4/day) with normal vital signs and inflammatory markers; moderate disease (S2) is characterized by four to six bloody stools per day without systemic features; and severe disease (S3) is characterized by more than six bloody stools per day with systemic signs such as fever (>37.5°C), tachycardia (>90 beats per minute), haemoglobin <10.5 g/dL, or ESR >30 mm/h. [8] There are three levels of illness: proctitis (E1), which only affects the rectum; left-sided colitis (E2), which involves the sigmoid and descending colon; and extensive colitis (E3), which is also called pancolitis. This combined assessment of disease severity and distribution is necessary to find the best treatment options and predict how the disease willprogress.[7-8] Faecal calprotectin has emerged as a valuable noninvasive biomarker that corroborates endoscopic, laboratory, and clinical findings within this diagnostic and classification framework. It is very important to tell the difference between inflammatory bowel disease (IBD) and functional disorders like irritable bowel syndrome (IBS) since it has a significant negative predictive value that is particularly useful for ruling out IBD in those who have symptoms. Faecal calprotectin also has a high sensitivity for finding patients who require a colonoscopy or other tests to find out what's wrong.[5]

Crucially, fecal calprotectin serves as a dynamic marker of intestinal inflammation, enabling continuous monitoring of disease activity and assessment of therapy effectiveness. A valuable instrument for guiding treatment modification within a tailored care plan, fluctuations in its levels are indicative of mucosal healing or disease recurrence. However, elevated levels may also be seen in other inflammatory conditions, including colorectal cancer and infectious enteritis, since fecal calprotectin indicates inflammation rather than disease specificity. [6]

Proctitis/distal colitis:

Topical therapy is the primary treatment for ulcerative colitis (UC), proctitis, and distal colitis. It is important to take care of these problems in a methodical and step-by-step

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way, particularly when the sickness is moderate to severe. Topical 5-aminosalicylic acid (5-ASA), like mesalazine suppositories (1 g/day), is the first therapy that doctors offer. It works in 31–80% of patients, but it only works in 7–11% of patients who get a placebo. There is no observable dose-response relationship when topical mesalazine doses above 1 g/day. You may use 5-ASA foam enemas, but suppositories are usually better since they are easier to use and transport the medicine directly to the rectum.(9)

Topical corticosteroids, such budesonide (2–8 mg/day) and viscocortisone (100 mg/day), are commonly used as extra or alternative treatments since they don't work as well as mesalazine. If patients don't respond to topical therapy and oral mesalazine (2–6 g/day for induction), it is recommended to increase the oral drug as a second-line treatment. If symptoms linger longer than two to four weeks, it is very important to carefully check how well the medication is working. It is essential to exclude viral etiologies of colitis and use endoscopic evaluation to confirm the presence of chronic inflammatory proctitis prior to escalating therapy. It might be hard to figure out what's wrong and treat overlapping conditions like infectious colitis or irritable bowel syndrome. Cytomegalovirus (CMV) infection must be excluded, particularly in refractory cases. Traditional staining methods may provide false negatives, thus immunohistochemistry labeling of viral proteins in mucosal biopsies is still the best way to discover CMV. It is much harder to understand what the positive quantitative CMV PCR findings mean since they don't always show the difference between an actual pathogenic role and an incidental viral presence in inflamed mucosa. It is important to treat proctitis that doesn't go away even with the greatest topical and oral therapies as a symptom of more serious or extensivecolitis.(10)

Faecal calprotectin (FC) has emerged as a prominent noninvasive biomarker in inflammatory bowel disease within this therapeutic and diagnostic framework, holding particular clinical relevance in ulcerative colitis (UC). It is a reliable indicator of mucosal inflammation due to its robust connection with clinical burden and endoscopic disease activity. FC is useful for more than just diagnosing diseases. It is especially useful for patients who have persistent abdominal symptoms of unknown cause Normal FC levels indicate a conservative, noninvasive therapeutic approach, but elevated levels clearly imply underlying organic disease, necessitating further diagnostic procedures, such as

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endoscopy.[11] High levels of faecal calprotectin are linked to active inflammation of the mucosa and may be able to predict a recurrence of the disease while therapy is being evaluated, especially if they are greater than normal clinical levels. This is a great reason to swiftly raise the level of treatment in order to repair the mucosa. So, FC is important for helping a personalized management plan decide whether to raise or lower medication. [12] However, because to the possible effects of confounding variables such using nonsteroidal anti-inflammatory drugs, having gastrointestinal problems, or having cancer on FC levels, it is important to be very cautious when interpreting the results. Also, FC's ability to diagnose changes depending on how bad the disease is, even if it is connected to endoscopic disease activity in UC. FC may identify rectal illness and signify localized mucosal inflammation in ulcerative proctitis; however, its sensitivity may be limited owing to generally lower levels in comparison to more widespread colitis. This limitation is elucidated by the presence of more solidified stool, which may lead to an unequal distribution among fecal samples, the restricted inflammatory mucosal surface area, and the decreased secretion of calprotectin into the intestinal lumen. [13]

Importantly, in patients with isolated proctitis, conventional blood-based inflammatory markers such as leukocyte count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) do not reliably correlate with endoscopic findings. Therefore, fecal calprotectin remains the most useful noninvasive biomarker in this subgroup, despite its limitations, underscoring its vital importance in the current biomarker-guided management of ulcerative colitis. [14]

Left-sided UC:

The ECCO (EL1b, RG B) and ACG (EL A) say that the best way to treat moderate to severe active left-sided ulcerative colitis is to mix topical aminosalicylates with oral mesalazine at doses of more than 2 g/day. A dose-response relationship has been corroborated by the association of higher dosages of oral mesalazine with expedited clinical improvement and a quicker resolution of rectal bleeding compared to lower doses (9 days vs. 16 days, $P < 0.05$). Nonetheless, clinical investigations indicated no statistically significant differences in overall remission rates across treatment groups (20.2% vs. 17.7%,

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$p > 0.05$). Examples of treatment techniques that employ a step-up escalation paradigm include topical corticosteroids, oral 5-ASA, and topical mesalazine. If rectal bleeding doesn't stop after 10 to 14 days of combination therapy, it is best to go on to systemic corticosteroids. The usual initial dose of corticosteroids is 40 to 60 mg taken by mouth once a day. There is no statistically significant difference between starting doses of 40 mg and 60 mg. Standard tapering methods usually start with 40 mg of prednisolone every day for seven days, then go down to 30 mg for one week, 20 mg for one month, and finally 5 mg every week. This is done since there is no strong evidence that one method is better than another. physicians in the United States have been more careful about introducing corticosteroids than physicians in Europe because they are worried about systemic adverse effects. Consequently, newer oral corticosteroids with reduced systemic absorption and customized colonic release have been developed, such as beclomethasone dipropionate and budesonide. In severe cases of left-sided colitis, hospitalization and intensive systemic therapy are often required. [10,15] [Figure 1]

In ulcerative colitis, accurate assessment of disease activity is essential for guiding therapy and optimizing outcomes. Faecal calprotectin (FC) has a better diagnostic performance because of the higher degree of inflammatory mucosa, the increased infiltration of neutrophils, and the subsequent release of calprotectin into the colonic lumen. This is especially true for left-sided disease, where FC is strongly correlated with endoscopic activity. Consequently, there exists a proportional relationship between the extent of mucosal inflammation and FC levels. Furthermore, FC reliably differentiates between active and inactive disease across various inflammatory stages, validating its use as a surrogate marker for mucosal healing and enabling continuing monitoring, as well as supporting biomarker-guided therapeutic adjustments. FC is the most therapeutically helpful noninvasive biomarker, particularly in patients with left-sided or severe colonic involvement, while standard blood markers such C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) display poor accuracy in reflecting endoscopic severity.[15]

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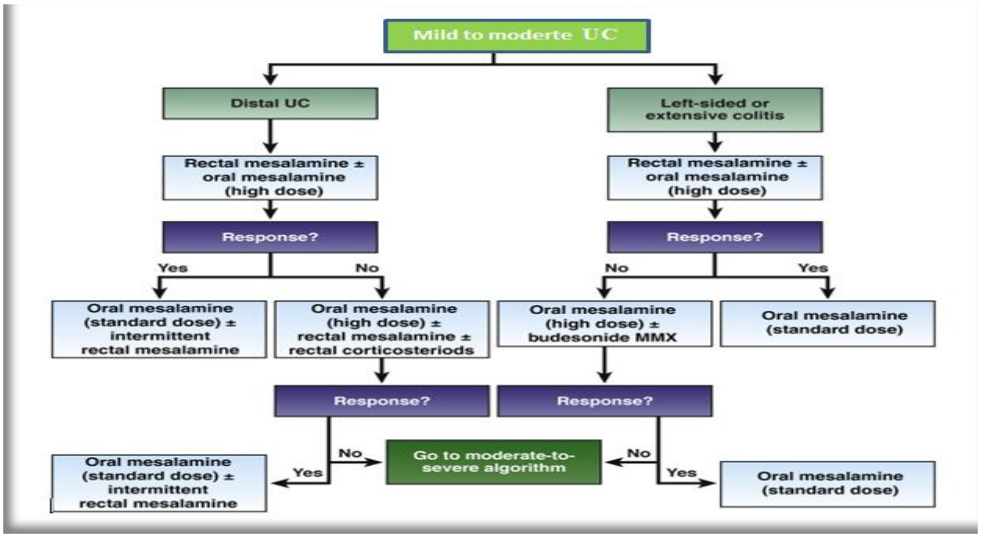


Figure (1): The Management guidelines for mild to moderate ulcerative colitis

Extensive ulcerative colitis

Oral sulfasalazine, with the dose slowly increased to 4–6 g/day, or a mix of oral and topical mesalazine should be the first line of therapy for mild to moderately severe extensive ulcerative colitis. About 20% of patients who use oral 5-ASA formulations go into remission. People who don't get well after 10 to 14 days of treatment or who are already on the right maintenance drug may need another round of oral steroids. For patients who are reliant on steroids or who can't take steroids, the recommended doses of azathioprine and 6-mercaptopurine for starting and maintaining remission are 2.5 mg/kg and 1.5 mg/kg, respectively. (16)

Severe ulcerative colitis

People with severe ulcerative colitis have more than six bloody stools a day and other symptoms throughout their body, such fever, a rapid pulse, and anemia. Patients must be hospitalized for extensive treatment and monitoring due to potentially fatal complications such as toxic megacolon and perforation. Intravenous steroids, such hydrocortisone 400 mg per day or methylprednisolone 60 mg per day, are the main treatment option for helping people get well. If people don't get well after obtaining enough oral treatment with prednisolone and 5-ASA, they may try the tumor necrosis factor (TNF)

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inhibitor IFX at a dose of 5 mg/kg.[17]

For individuals with severe ulcerative colitis requiring intravenous corticosteroids, the colectomy rate may reach 29%. Therefore, the patient must be referred to the colorectal surgeon upon admission. Gastroenterologists and surgeons must work together on a daily basis to minimize unnecessary delays in surgery. If a patient's illness does not get better or becomes worse after three days of steroid treatment, a colectomy may be an option. If the symptoms don't go better after seven days of taking steroids, the treatment is useless and might create problems with recovery following surgery that could have been avoided. To gauge the response to intravenous steroids, it is recommended to measure stool frequency, CRP levels, and conduct abdominal radiography on the third day. When pharmacological treatment has proven ineffective, proctocolectomy or CsA rescue therapy should be considered. [18]

To avert prompt surgical intervention in instances with corticoid-resistant malignancy, we delineate the application of calcineurin inhibitors (CsA, tacrolimus) and infliximab (IFX) as a second-line therapy. [18] Giving CsA intravenously at a dose of 4 mg/kg every day works nicely. This may be an option for those who can't take corticosteroids because they have conditions like steroid psychosis. As soon as remission induction works, immunosuppressants such azathioprine (2.5 mg/kg daily) must be begun. It is recommended to switch to oral tacrolimus medicine while gradually reducing the dosage of cyclosporine A (CsA) over three to six months. Patients may not show any benefit for up to three months while using azathioprine and 6-mercaptopurine together. It is not recommended to use tacrolimus and cyclosporine A together for maintenance treatment. After intravenous administration, 50 to 80 percent of patients receiving CsA as a rescue treatment report significant short-term enhancement. Studies examining long-term consequences indicated that 58%–88% of these individuals had a colectomy within seven years after the therapy.

The fact that CsA has a short half-life is a big plus for rescue treatment compared to IFX. It goes away in a few hours, but IFX may last for weeks if it doesn't work. The recommended dose of the calcineurin inhibitor tacrolimus to achieve trough levels of 10–15 ng/mL is 0.1–0.2 mg/kg daily or 0.01–0.02 mg/kg intravenously. Retrospective uncontrolled investigations show that trough levels between 4 and 8 ng/mL are better and

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have fewer side effects. Patients on triple immunosuppressive therapy are at an elevated risk for opportunistic infections due to *Pneumocystis jiroveci* (DGVS EL B); hence, chemoprophylaxis is recommended. [19]

If the patient can't take the 160/800 mg dose of trimethoprim-sulfamethoxazole every two weeks, they may try inhaling 300 mg of pentamidine once a month instead. The ECCO and the most current ACG recommendations say that IFX may help lower the need for colectomy. In clinical practice, it is often regarded as a secondary alternative due to its extended half-life relative to CsA. The first dosage of infliximab is 5 mg/kg administered by an IV. Infusions are expected to happen every eight weeks following weeks two and six. In a randomized controlled study, 45 patients with severe corticoid-refractory colitis were administered either continuous intravenous betamethasone or infliximab (IFX). Seven out of twenty-four patients were considerably less than fourteen out of twenty-one ($P = 0.017$; odds ratio 4.9; 95%). [20]

A confidence interval (CI) of 1.4–17 indicates that the patient had a colectomy within three months of starting infliximab (IFX) therapy. In recent research assessing infliximab (IFX) as a rescue treatment for tacrolimus-refractory ulcerative colitis (UC), six out of twenty-four patients, almost 25% of the total, demonstrated improvement after IFX administration. The efficacy of infliximab (IFX) rescue treatment for steroid-refractory severe prolonged colitis remains uncertain due to the small sample size and considerable diversity in colectomy rates seen in current case studies. In order for remission to happen, patients on infliximab (IFX) must complete a six-month IFX maintenance program. At this time, adalimumab, a completely humanized TNF alpha inhibitor, cannot be used for the treatment of ulcerative colitis. [21]

Leukocytopheresis, which involves the targeted physical separation of activated immune cells participating in the inflammatory process, is an alternative therapy for active ulcerative colitis. This method is now being investigation, although its effectiveness remains contentious. Research from Japan indicates that leukocytapheresis induces remission with fewer adverse effects compared to corticosteroid therapy. A recent study including an international cohort revealed no significant differences in clinical outcomes between the apheresis and sham therapy groups. [20]

There is little evidence indicating that methotrexate (MTX) may induce remission.

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The only randomized placebo-controlled trial demonstrated no benefit for ulcerative colitis. In a trial including 34 patients with steroid-dependent UC, researchers investigated the effects of 6-mercapto-purine, oral MTX, and 5-ASA in conjunction with prednisolone. 58.3% of patients in the MTX group did not achieve remission, while 35% of patients in the 5-ASA group did not achieve remission ($P > 0.5$). The modest impact shown in all of the studies might have been caused by giving little quantities of MTX (15 mg/kg per week) via mouth. MTX may be used in certain cases, starting at 25 mg/week and going down to 15 mg/week when the disease goes away, although recommendations do not currently support its use. MTX may cause serious adverse effects such lung damage, bone marrow depression, and liver toxicity. These side effects need to be carefully managed. The possibility for teratogenic consequences underscores the need for rigorous contraception. Taking 5 mg of folic acid by mouth the morning after taking MTX is a safe and efficient way to lessen its negative effects. [22] [Figure2]

Faecal calprotectin (FC) levels increase in direct correlation with the severity of endoscopic disease, as assessed by the Mayo endoscopic subscore in this intensive therapeutic regimen, indicating a strong relationship with mucosal inflammatory activity. High FC levels indicate that there is a lot of inflammation and damage to the mucosa. There is a link between FC and how bad the sickness is, but in severe ulcerative colitis, the level of inflammation is the major thing that makes it go up, and how bad the illness is is the second thing. The dependability of FC as a surrogate marker for endoscopic activity and its utility in biomarker-guided management of severe ulcerative colitis are substantiated by the observation that markedly elevated FC levels predominantly indicate severe mucosal damage and significant neutrophilic infiltration, rather than the anatomical distribution of the disease. [23]

Things to think about while giving adjuvant treatment to those with severe ulcerative colitis. Antibiotic medication may only be considered after an accurate diagnosis of an infection. It is not completely apparent how well tobramycin and metronidazole work together to treat severe ulcerative colitis. Patients need to get enteral nutrition if they can take it and there is no ileus or subileus, since bowel rest has no effect on the progression of acute colitis.Enteral feeding significantly lowers the probability of complications (9% vs.35%).

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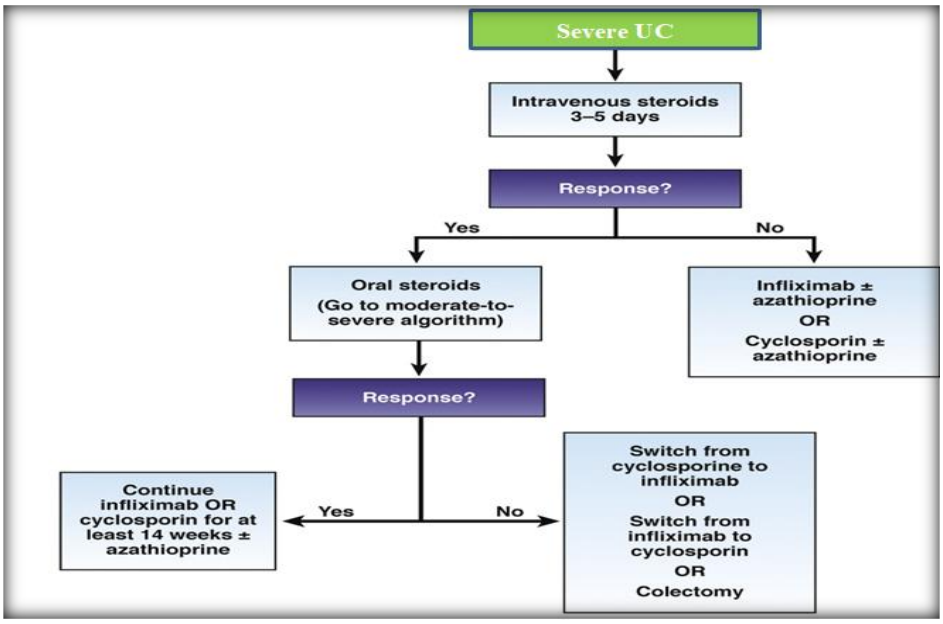


Figure (2): The Management guidelines for severe ulcerative colitis

Outcome prediction and surgical intervention:

A recent European population-based study found that people with ulcerative colitis (UC) had an 8.7% chance of needing a colectomy over the course of 10 years. There is still not a lot of strong prognostic evidence that is specific to UC, even though a lot of the literature that is now being published has focused on signs of poor pharmacological response. A younger age at diagnosis, early corticosteroid usage, ileal involvement, absence of mucosal healing, and smoking have been associated with an elevated chance of catastrophic surgical outcomes or disability in Crohn's disease; analogous prediction models in ulcerative colitis are less advanced. [24]

According to population-based studies, primary sclerosing cholangitis, as evidenced by the Norwegian IBSEN cohort over a 10-year follow-up, an elevated erythrocyte sedimentation rate (ESR ≥ 30 mm/h), and severe disease at initial presentation are all associated with a heightened risk of colectomy in ulcerative colitis. Conversely, smoking and an older age at disease onset (≥ 50 years) have been associated with a reduced probability of colectomy. In a prospective cohort of 49 hospitalized patients with severe ulcerative colitis (UC), colectomy was required in 85% of instances where patients

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exhibited elevated C-reactive protein (CRP > 0.45 mg/L) on day 3 of treatment, a history of frequent diarrhea (3–8 episodes/day), and sustained high stool frequency (≥ 8/day) despite corticosteroid and/or cyclosporine A administration. These findings underscore the need for the early identification of prognostic indicators to facilitate timely surgical intervention in refractory disease trajectories. [25,26].

Emergency surgery is needed for life-threatening conditions include intestinal perforation, refractory toxic megacolon, and recurring severe colorectal hemorrhage. In these very serious situations, reconstructive surgery is not an option. Instead, subtotal colectomy with ileostomy and rectum preservation is recommended. When dysplasia or colon cancer is present, structuring disease might produce partial or complete intestinal obstruction, or chronic continuous colitis may not respond to immunosuppressive therapy, elective surgery may be indicated. Total proctocolectomy with ileal pouch–anal anastomosis (IPAA) is the most common elective surgical treatment for UC. It may be able to cure the disease. However, this approach is associated with significant postoperative complications, impacting around 20% of patients and resulting in intra-abdominal abscesses, fistula formation, infections, and diminished fertility and sexual dysfunction. Ileorectal anastomosis could be a temporary choice in certain cases, particularly for young women who have never given birth. However, this method needs strict long-term monitoring since the saved rectal segment has a higher chance of illness recurrence or cancer. [27,28]

Faecal calprotectin (FC) has become an essential biomarker for mucosal inflammation, offering considerable predictive value in ulcerative colitis within this prognostic and therapeutic context. High levels of FC are linked to severe illness, which is linked to an increased inflammatory load in the colonic mucosa and worse clinical outcomes, such as a higher likelihood of needing surgery. It is also suggested that higher FC levels may predict treatment outcomes since they are linked to a poorer response to both first- and second-line medicines.In addition, particular FC cutoff values are quite good at finding people who are likely to become worse with their condition. [29]

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Also, high FC levels, which show how bad the underlying mucosal inflammation is, are strongly linked to the failure of intravenous corticosteroid treatment and a higher chance of needing a colectomy. FC is a useful technique for early risk assessment since it helps locate those who are at high risk and might benefit from immediate surgery or rescue medicines. In the end, our biomarker-guided method improves the clinical results for ulcerative colitis by making management strategies better and cutting down on delays in getting the right medicine. [12]

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