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A COMPREHENSIVE OVERVIEW OF SURGICAL OPTIONS FOR ULCERATIVE COLITIS: A MULTIDISCIPLINARY APPROACH TO CURRENT THERAPIES, EMERGING TREATMENTS, AND FUTURE DIRECTIONS

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Abstract

Acute severe ulcerative colitis is a problem that needs to be treated in a hospital with strong medicine like intravenous steroids. One third of the people with acute severe ulcerative colitis do not get better with steroids and need special treatment to help them. Doctors can use infliximab or cyclosporine to help these people. They are equally good at helping. However doctors like to use infliximab because it is easier to use and they know more about it. But some people have already taken infliximab before so doctors are using cyclosporine often for them. If people with severe ulcerative colitis do not get better with this special treatment they may need to have surgery to remove their colon.

There is a lot of interest in finding ways to treat acute severe ulcerative colitis so that people do not need to have surgery. Some new medicines like Janus kinase inhibitors might be helpful when used with steroids from the start or as a treatment but 1more research is needed to know for sure if they work. Using a lot of infliximab to treat severe ulcerative colitis has had different results but it might be a good idea for people who are very sick and have a lot of disease in their body. In this article we talk about the ways to treat acute severe ulcerative colitis and some new ideas that might help people with acute severe ulcerative colitis, in the future.

Keywords: bowel disease; ulcerative colitis; infliximab; cyclosporine; acute severe colitis; Jak Inhibitors (JAKI)

I. Introduction

We know some things about Ulcerative colitis. That also known chronic disease. Ulcerative colitis a several causes inflammation called ulcers, in the inner lining of the large intestine, which includes the colon and the rectum—the end part of the colon. UC is one of the two main forms of chronic inflammatory disease of the gastrointestinal tract, called inflammatory bowel disease (IBD). The other form is called Crohn's disease. Normally, the large intestine absorbs water from stool and changes it from a liquid to a solid. In UC, the inflammation causes loss of the lining of the colon, leading to bleeding, production of pus, diarrhoea, and abdominal discomfort.

I.1. Causes of UC

The cause of UC is unknown though theories exist. People with UC have abnormalities of the immune system, but whether these problems are a cause or a result of the disease is still unclear. The immune system protects people from infection by identifying and destroying bacteria, viruses, and other potentially harmful foreign substances. With UC, the body's immune system is believed to react abnormally to bacteria in the digestive tract. UC sometimes runs in families and research studies have shown that certain gene abnormalities are found more often in people with UC. sensitivity to certain foods or food products does not cause UC, it may trigger symptoms in some people. UC is not caused by emotional distress, but the stress of living with UC may contribute to a worsening of symptoms. In addition, while sensitivity to certain foods or food products does not cause UC, it may trigger symptoms in some people.

I.2. Who are at high risk of getting UC

Ulcerative colitis is primarily found in people who are diagnosed with the disease. The increasing number of people getting UC in the past 50 years is particularly noteworthy. As stated by CDC, the occurrence of UC (the rate of new cases in each year) is between 2.2 and 14.3 cases per 100,000 inhabitants. The medium age at which most people are diagnosed with ulcerative colitis is 35-36 years, but the disease can be found in people of all age groups. The prevalence of ulcerative colitis is greater among men than women as men are more likely to be diagnosed with it in their 50s and 60s. Caucasians are more likely to get the ulcerative colitis background than the people of any other racial or ethnic group. This is the reason both ulcerative colitis and Crohn's disease tend to be more prevalent in the northern climate countries, urban areas, and developed countries. Most of these patterns are on the other hand gradually changing; for instance, the number of UC patients is rising in some developing countries such as China, India, and South America. In 2021, IBD was prevalent in 3.8 million individuals, with incident and death ASR of 4.4 and 0.5 respectively. The overall ASR incidence rate rose between 1990 and 2021 but in the same period ASR% of death, prevalence, and disability-adjusted life year rates declined. The age-standardized death rate (ASDR) barely increased in most regions and countries from 2019 to 2021. High-rate regions as Western Europe were still under fungible burdens. East Asian countries, notably China, were under the effect of a drastic surge in incidence. The ASRs are proportional to the incidence and death with an increase in the socio-demographic index. By 2035, the IBD incidence and death will ASR gradually decrease.

I.3. Responsible Genetic Factor

A group of scientists has made a finding that ulcerative colitis is likely to be inherited. Namely, during the family risk for getting IBD it is estimated to be between 1.5 percent and 28 percent for an affected person's first-degree relatives.7 On the one hand, genetics is surely a key factor, on the other hand, dietary habits, lack of exercise, or exposure to some pollutants could be environmental factors that trigger the onset, progression, and relapse of the disease. In this light, even though the family history has a credible influence on the increased risk of IBD, it actually remains impossible to accurately determine who, if any, the family members are that would develop ulcerative colitis.

People who have relatives diagnosed with ulcerative colitis do not have to take the test right away as a routine measure. If the family members on that side of the family get symptoms, they also need a check-up by a gastroenterologist. Some conditions come with symptoms that are almost indistinguishable from IBD, and as such getting a clear diagnosis is vital.

I.4. Symptoms of UC

The most common symptoms of UC are abdominal discomfort and blood or pus in diarrhoea. Other symptoms include:

- anaemia
- fatigue
- fever
- nausea
- weight loss
- loss of appetite
- rectal bleeding
- loss of body fluids and nutrients
- skin lesions
- growth failure in children

Most people diagnosed with UC have mild to moderate symptoms. About 10 percent have severe symptoms such as frequent fevers, bloody diarrhoea, nausea, and severe abdominal cramps.1 UC can also cause problems such as joint pain, eye irritation, kidney stones, liver disease, and osteoporosis. Scientists do not know why these problems occur, but they think these complications may be the result of inflammation triggered by the immune system. Some of these problems go away when UC is treated. Different varieties of ulcerative colitis and their associated symptoms. The manifestations of ulcerative colitis will be different based on the degree of inflammation and the position of the disease within the large intestine. Therefore, you must know which part of your large bowel is affected. Here are some of the most widely seen forms of ulcerative colitis •

I.4.1. Ulcerative proctitis

The inflammation and irritable bowel syndromes are confined solely to the rectum (usually no more than six inches of the rectum) and does not mean that the patient has a higher risk of increased cancer. The symptoms consist of rectal bleeding, urgency, and rectal pain. •

I.4.2. Left-sided colitis

This type of colitis is characterized by the continuous inflammation that starts at the rectum and goes up to the splenic flexure (a bend in the colon that is located near the spleen in the upper left abdomen). Proctosigmoiditis is also a kind of left-sided colitis. Symptoms of this condition are as follows: loss of appetite, weight loss, bloody diarrhoea, and pain on the left side of the abdomen.

I.4.3. Extensive Colitis

Affects the entire colon continuous inflammation that begins at the rectum and extends beyond the splenic flexure. Symptoms include loss of appetite, bloody diarrhoea, abdominal pain, and weight loss.

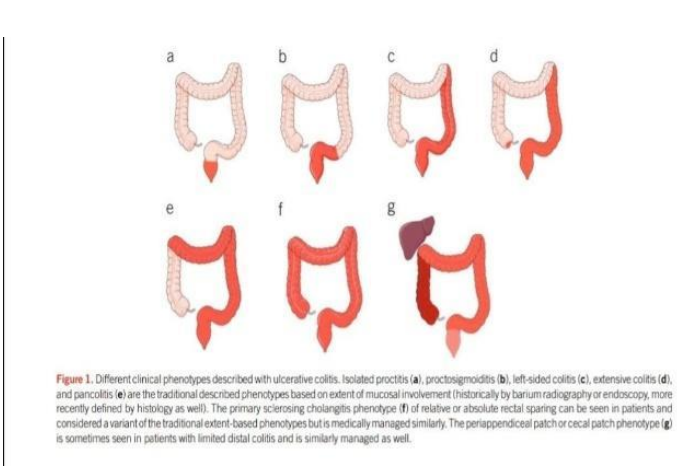


Figure 1. Different clinical phenotypes described with ulcerative colitis. Isolated proctitis (**a**), proctosigmoiditis (**b**), left-sided colitis (**c**), extensive colitis (**d**), and pancolitis (**e**) are the traditional described phenotypes based on extent of mucosal involvement (historically by barium radiography or endoscopy, more recently defined by histology as well). The primary sclerosing cholangitis phenotype (**f**) of relative or absolute rectal sparing can be seen in patients and considered a variant of the traditional extent-based phenotypes but is medically managed similarly. The periappendiceal patch or cecal patch phenotype (**g**) is sometimes seen in patients with limited distal colitis and is similarly managed as well.

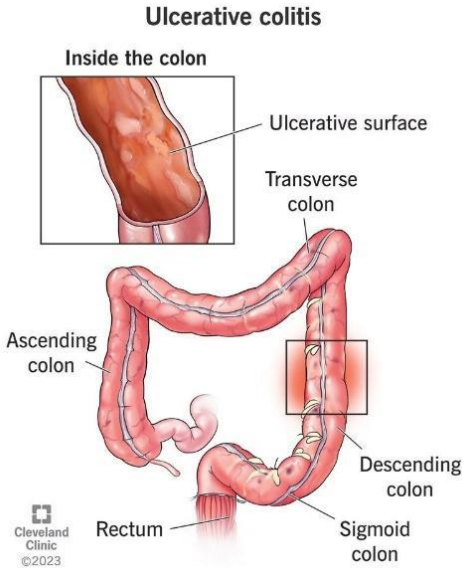
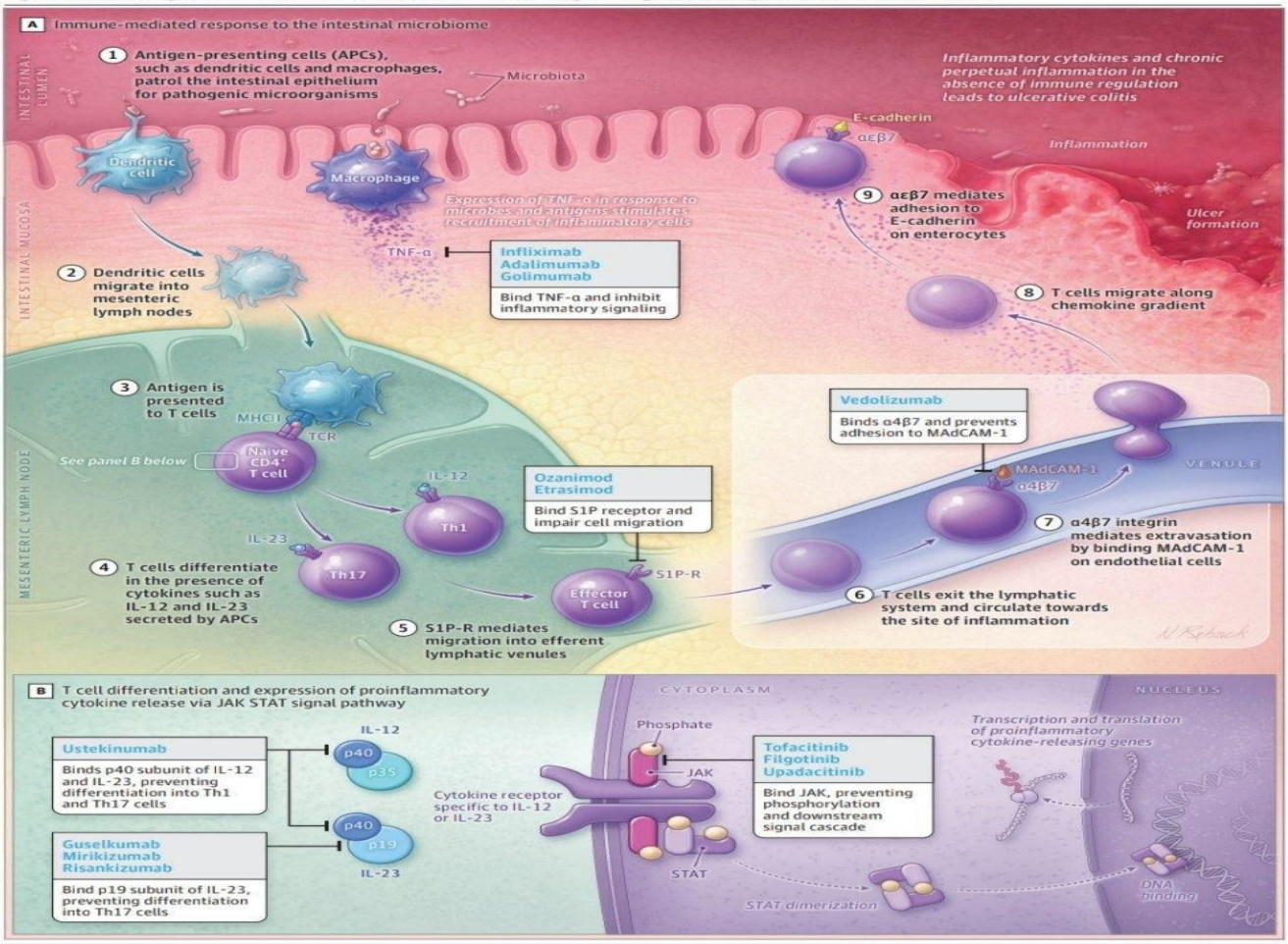


Figure- ulcerative colitis

II. Pathophysiology

UC, as a recurrent and refractory digestive disease, is closely associated with colorectal cancer, but the exact aetiology has not yet been determined (13). UC is greater customary in developed international locations than in growing countries, however the incidence in creating nations is getting greater and higher. The gut microbiota, mucosal barrier damage, and immune response regulation are affected by nutritional imbalance and dietary factors. While UC can occur in people of any age, it usually develops between the ages of 15 and 30and less frequently between the ages of 60 and 80. The disease affects men and women equally. People with a family member or first-degree relative with an IBD are at higher risk for developing UC, as are Caucasians and people of Jewish descent. The pathogenesis of UC is driven by gene-environment interactions leading to an abnormal immune system response to the intestinal microbiome. More than 200 genetic loci have been associated with UC. Environmental exposures may predispose an individual to UC by influencing the diversity and composition of the 1 trillion bacteria, viruses, and fungi that form the intestinal microbiome.

Figure 1. The Pathogenesis of Ulcerative Colitis With the Immunological Targets of Biologics and Oral Small Molecules



IL indicates interleukin; JAK, janus kinase; MAdCAM-1, mucosal vascular addressin cell adhesion molecule 1; MHCII, Major Histocompatibility Complex Class II; S1P, sphingosine-1-phosphate; STAT, signal transducer and activator of transcription; TCR, T-cell receptor; and TNF, tumor necrosis factor.

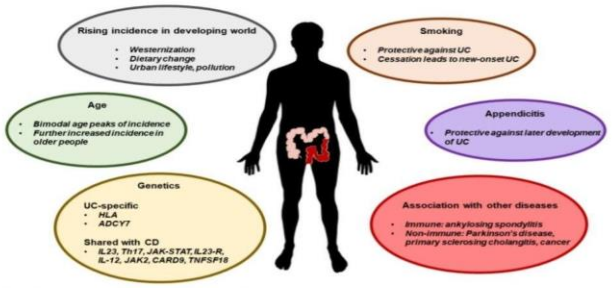


Figure 1. **General factors associated with increased susceptibility of UC.** CD, Crohn's disease; UC, ulcerative colitis.

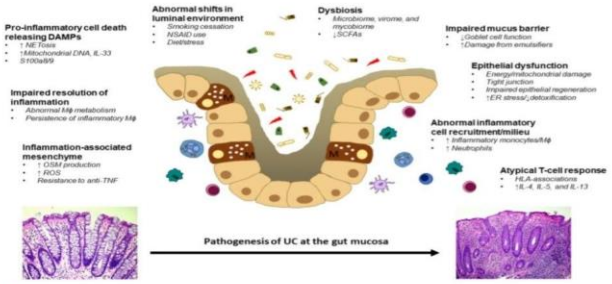


Figure 2. **Molecular mechanisms involved in the development of mucosal inflammation in UC.** DAMPs, damage-associated molecular patterns; ER, endoplasmic reticulum; HLA, human leukocyte antigen; IL, interleukin; Mϕ, macrophage; NSAID, non-steroidal anti-inflammatory drug; OSM, oncostatin M; ROS, reactive oxygen species; SCFA, short-chain fatty acid; TNF, tumour necrosis factor; UC, ulcerative colitis.

III. Diagnosis &Testing of uc

Health care personnel do a colonoscopy and collect tissue samples, called biopsies, from the inside of the colon for the diagnosis of ulcerative colitis. The possibilities of other forms of inflammatory bowel disease, such as Crohn's disease, can be ruled out with the help of additional tests like stool tests, MRI, and CT scans. Inclusion of one or more of the below tests and processes would be on recommendations as part of the course of treatment for someone with ulcerative colitis.

3.1. Laboratory tests:

3.1.1. Blood tests: The doctor might recommend blood tests to look for anaemia or indications of infections. Anaemia is a medical condition that results in the body not having the required amount of red blood cells to carry oxygen to the organs. In some cases, they also look for signs of inflammation.

3.1.2. Stool studies: The presence of white blood cells or some proteins in stool can be helpful in the diagnosis of ulcerative colitis. A stool sample can also rule out other problems such as bacterial, viral or parasitic infections.

3.2. Endoscopic processes

3.2.1. Colonoscopy: Using a slender and flexible light with a camera at the end, the healthcare professional is allowed to check the entire intestine. They take tissue samples during the therapy for testing in the laboratory. This procedure is called a biopsy. One of these tissue samples is required for the identification.

3.2.2. Flexible sigmoidoscopy: As with colonoscopy, flexible sigmoidoscopy features a thin, flexible, and lighted tube with a camera attached to it but the tube is shorter. This test is designed for the rectum and sigmoid colon, the final part of the colon. When the colon is highly inflamed, this test may be done instead of a full colonoscopy.

3.3. Imaging procedures

3.3.1. X-ray: If dyspeptic symptoms are very severe, a standard X-ray of the abdomen is done firstly to exclude the possibility of dilated colon. Dilation of the colon can have serious complications, such as toxic megacolon and perforation.

3.3.2. CT scan: A CT scan of either the abdomen or pelvis may be performed to assist in checking the inflammation. It may also be carried out to rule out the presence of an abscess or other sources of pain.

3.3.3. CT enterography and magnetic resonance (MR) enterography: A health professional may suggest one of these noninvasive tests, to rule out the inflammation in the small intestine. These tests are more sensitive to detect inflammation than conventional imaging tests. MR enterography is a radiation-free alternative toother diagnostic imaging modalities.

Table 1. Investigations for ulcerative colitis

Investigation	Type of investigation	Common findings in UC	Notes
Blood tests	Full blood count Urea and electrolytes C-reactive protein Vitamin D and bone profile Haematinics Liver biochemistry	Anaemia, thrombocytosis, low vitamin D and raised inflammatory markers	Consider pre-immunosuppressant/biologic screen in those likely to need escalation to immunosuppressant/biologic therapy (including TPMT, viral serology, quantiferon) Primary sclerosing cholangitis can be associated with UC and can present with deranged liver biochemistry
Stool cultures	*Clostridioides difficile* toxin assay MC&S	Should be negative if UC, but infections such as *C difficile* can co-exist	Take a thorough history, including travel history to rule out other causes Recent antibiotic use may be associated with *C difficile*
Faecal calprotectin	Indicates migration of neutrophils to the lumen via the intestinal mucosa	A level of 50–100 µg/g has a high negative predictive value of 98–99% in the diagnosis of IBD	Can be used as a method of monitoring treatment response
Endoscopy	In acute setting, flexible sigmoidoscopy Ileocolonoscopy is recommended in all patients to delineate disease extent, severity of inflammation and to exclude Crohn's disease; also for surveillance	Erythema, oedema, loss of vascular pattern, blood and ulcers/erosions	Mayo score and Ulcerative Colitis Endoscopic Index of Severity (UCEIS) score are used to assess severity of ulcerative colitis endoscopically
Histology	Recommend at least two biopsies from each bowel segment for histological assessment	No histological features are diagnostic of UC, but basal plasmacytosis, crypt atrophy/distortion and villous surface irregularity are suggestive of UC	The presence of granulomas is more suggestive of Crohn's disease
Imaging	Abdominal X-ray	Thumbprinting, lead-piping, oedema and toxic megacolon	
Imaging	Cross sectional imaging: CT/MRI	Bowel wall oedema and inflammatory pseudopolyps	Small bowel imaging can help differentiate between UC and Crohn's disease in challenging cases

CT = computed tomography; IBD = irritable bowel disease; MC&S = microscopy, culture and sensitivity; MRI = magnetic resonance imaging; TPMT = thiopurine methyltransferase; UC = ulcerative colitis.

IV. Endoscopy target for disease clearance in UC

The goal of treatment for Ulcerative Colitis or UC is to clear up the disease. Mucosal healing, which is also called MH is a goal for treatment. It is recommended for patients with Inflammatory Bowel Disease or IBD. It is included in the STRIDE-2 plan for long-term treatment of UC. This is because mucosal healing is related to a favourable course of the disease. There are scores that doctors use to measure the severity of UC. The Mayo Endoscopic Score or MES is the commonly used score in clinical practice and trials. It is easy to use and gives a visual representation of the degree of inflammation in UC. The score ranges from 0 to 3. Another score that is used is the Ulcerative Colitis Endoscopic Index of Severity or UCEIS. UCEIS is also established in the STRIDE-2 plan. It includes three descriptors: pattern, bleeding and erosions and ulcers. UCEIS is more accurate in describing the severity of the lesions. How well patients respond to treatment. This is because it considers the size and depth of ulcers. Both MES and UCEIS describe the severely affected area of the colon but they do not describe the extent and location of the inflammation. Mucosal healing is often defined as an MES of 0 or 1. However complete endoscopic healing, which is an MES of 0 is associated with outcomes. Therefore doctors should consider concepts, such as endoscopic improvement and endoscopic remission. Endoscopic improvement is an MES of 0 or 1 and endoscopic remission is an MES of 0 or a UCEIS of 0. Several studies have shown that patients with an MES of 0 have outcomes than those with an MES of 1. In two studies patients with an MES of 1 had a rate of relapse than those with an MES of 0. A recent meta-analysis found that patients with an MES of 1 had a risk of relapse than those with an MES of 0. Another meta-analysis found that achieving remission, which is an MES of 0 had a 52% lower risk of clinical relapse. A recent study compared patients with UC who did not have disease clearance and those who did. Disease clearance was defined as clinical, endoscopic and histological remission. The study found that patients who achieved disease clearance had a risk of hospitalization and surgery. This shows that achieving disease clearance is associated with outcomes. In summary an MES of 0 should be the goal of treatment for UC. This is because it predicts an outcome. Endoscopy is a tool for demonstrating mucosal inflammation and defining the extent of the disease. The Mayo Endoscopic Subscore provides a measure of endoscopic severity throughout the colon. Colonoscopy with intubation of the ileum is recommended for patients with suspected IBD. UC typically involves the rectum. Extends proximally in a continuous circumferential pattern. It can be classified as proctitis sided colitis or extensive colitis. The severity of UC can be measured by the mucosal changes. Mild disease is associated with erythema, vascular congestion and partial loss of the vascular pattern. Moderate disease shows loss of the vascular pattern, adherent blood, erosions and mucosal friability. Severe disease is characterized by bleeding and ulceration. In UC ulcers occur within inflamed mucosa whereas in Crohn's disease the adjacent mucosa may appear normal. There are some variants and challenges in diagnosing UC. Recognized variants include sparing, particularly in patients with primary sclerosing cholangitis and peri-appendiceal patchy inflammation. Some patients, those, with extensive colitis exhibit backwash ileitis, which can make it difficult to distinguish from Crohn's disease. Magnetic resonance enterography can help diagnose backwash ileitis. It shows valve gaping and terminal ileal dilatation whereas increased bowel wall thickness is more suggestive of Crohn's disease.

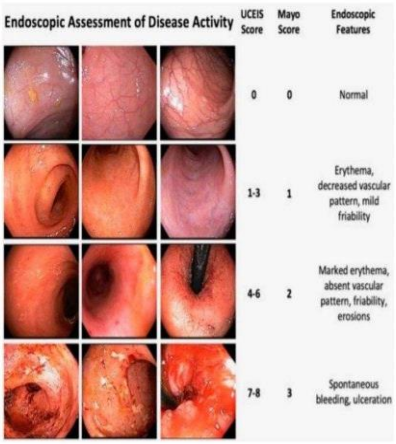


Figure 2. Sample endoscopic images of Ulcerative Colitis using the Mayo endoscopic subscore (66) and the ulcerative colitis endoscopic index of severity (59). UCEIS, Ulcerative colitis Endoscopic Index of Severity.

V. Histopathology for Accurate Classification

Biopsy method and the histopathological analysis are both crucial for the precise classification and the separation of UC from other types of colitis. No less than a pair of biopsy specimens must be obtained from each of the six examined places including the terminal ileum, ascending, transverse, descending, and sigmoid colon, and rectum. The selected samples must be macroscopically normal mucosa and be fixed right after collection in buffered formalin .The typical histological deviations seen in the case of UC are crypt architectural distortion, mucosal atrophy, and a diffuse transmucosal inflammatory infiltrate with basal plasmacytosis, where cryptitis and crypt abscesses are the features of active disease .There are not all characteristics in stage 1 disease, which highlights the function of basal plasmacytosis as a marker that appears early and is different from others [1].;In histological terms, crypt architectural distortion can be seen in the absence of early disease and is reported in just 20% of patients who experience symptoms a fortnight before diagnosis. By contrast, basal plasmacytosis appears earlier and has high predictive value for UC, helping to distinguish it from infectious colitis, in which crypt architecture is usually preserved.

VI. Biomarkers of UC

The existing research work being carried out on Ulcerative Colitis (UC)being biomarkers which currently deal mainly with the conversion of the invasive procedures from colonoscopies to the modern non-invasive, molecular-based device handling of the disease activity and response to treatment prediction are impacting massively on this field.

6.1. Confirmed Clinical Biomarkers

Even if numerous new markers continue emerging, the ones listed in the most recent clinical guidelines, like the ones the American Gastroenterological Association (AGA) have put out, stress the following three primarily non-invasive means of monitoring:

6.1.1. Fecal Calprotectin (FCP): the most common marker of mucosal inflammation, neutrophil-derived protein. Typically, the active inflammation in patients that are in symptomatic remission is ruled out through a FCP level <150 Î¼g/g.

6.1.2. Fecal Lactoferrin: An iron-binding glycoprotein found in neutrophils, used similarly to calprotectin to identify active Inflammatory Bowel Disease (IBD).C-Reactive Protein (CRP) is a serum substance synthesized by the liver during acute-phase response. Although it is a nonspecific indicator, its utilization in monitoring control of flare and effect of treatment is very efficient; particularly it gives reliable results in severe cases.

6.2. The Newest Molecular and Genetic Biomarkers

The notable research articles published recently speak of groundbreaking candidates for specific UC activity.

6.2.1. MicroRNAs (miRNAs): The study reflected in PMC shows the most significant hallmarks of diagnostics, namely miR-21, miR-126, and miR-146b-5p. For instance, the increase in the level of miR-126 in the active UC tissues associated with healthy controls was the 18-fold increase.

6.2.2. Gene Signatures:

Lipid Metabolism Genes: The novel diagnostic markers such as ABCD3 (AUC = 0.9185) and NR3C2 (AUC = 0.9025) are identified using machine learning.

6.2.3. Hub Genes: The case studies published in Scientific Reports report that CXCL1, CYP2R1, LPCAT1, and NEU4 are the significant activity markers associated with immune cell infiltration.

6.2.4. PARP8: Cited as a potential biomarker correlating positively to the presence of neutrophils and M1 macrophages.

6.3. *Prognostic and Investigative Markers*

6.3.1. CAR and NAR Ratios: The cattle study CRP-to-Albumin Ratio (CAR) and the Neutrophil-to-Albumin Ratio (NAR) although the same are different are discovered as the earliest signs of treatment effectiveness, particularly for biologics besides infliximab.

6.3.2. Anti-integrin αvβ6 Antibodies: These antibodies are present in 92% of patients with UC (compared to 5.2% of healthy controls) indicating very high specificity to the diagnosis and monitoring of the disease.

6.3.3. Fecal Metabolites: Certain compounds such as L-tryptophan and succinic acid are found in much larger amounts during UC exacerbation, however, lithocholic acid is joined with remission.

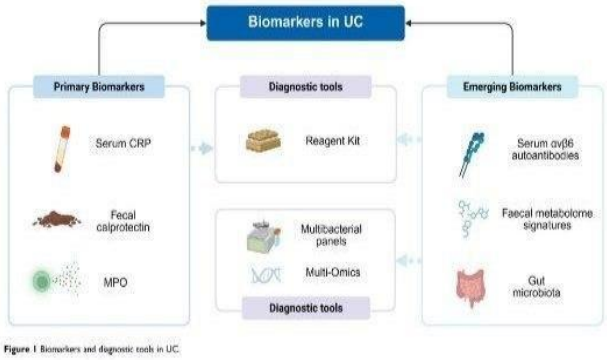


Figure 1 Biomarkers and diagnostic tools in UC.

Table 1 summarizes representative biomarkers categorized by type, target, clinical relevance, and diagnostic accuracy parameters.

While traditional biomarkers remain effective for activity monitoring, multi-omics and microbiome-based models promise greater specificity and long-term prognostic power, providing a stronger foundation for personalized therapy.

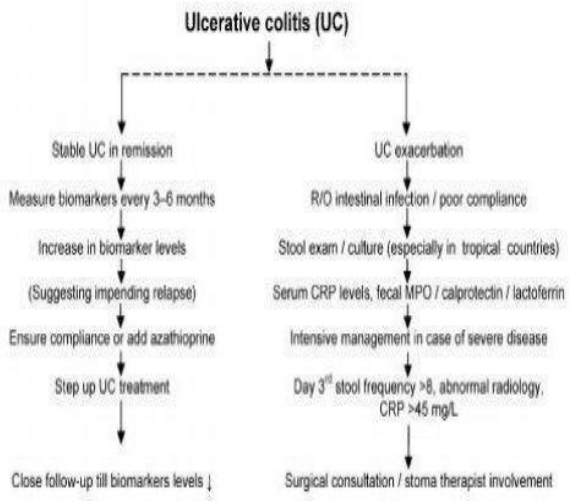


Figure 1: Algorithm for clinical use of biomarkers in ulcerative colitis

VII. Treatment Of UC

Treatment for UC depends on the severity of the disease and its symptoms. Each person experiences UC differently, so treatment is adjusted for each individual. Medication Therapy While no medication cures UC, many can reduce symptoms. The goals of medication therapy are to induce and maintain remission periods when the symptoms go away for months or even years—and to improve quality of life. Many people with UC require medication therapy indefinitely, unless they have their colon and rectum surgically removed. The type of medication prescribed depends on the severity of the UC.

7.1. Aminosalicylates: Aminosalicylates, medications that contain 5-aminosalicyclic acid (5-ASA), help control inflammation. One medication, sulfasalazine (Azulfidine), is a combination of sulfapyridine and 5-ASA.

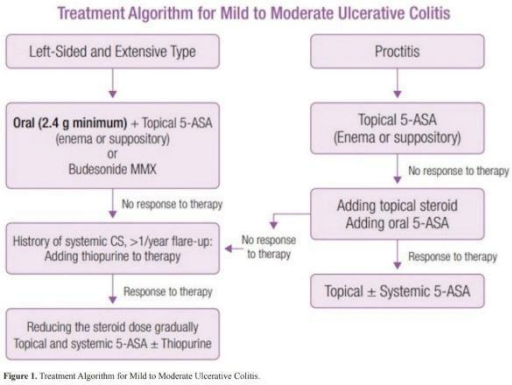


Figure 1. Treatment Algorithm for Mild to Moderate Ulcerative Colitis.

The sulfapyridine component carries the anti-inflammatory 5-ASA to the intestine. Sulfapyridine may lead to side effects such as nausea, vomiting, heartburn, diarrhoea, and headache. Other 5-ASA agents, such as olsalazine (Dipentum), mesalamine (Asacol, Canasa, Lialda, Rowasa), and balsalazide (Colazal), cause fewer side effects, and can be used by people who cannot take sulfasalazine. Depending on which parts of the colon and rectum are affected by UC, 5-ASAs can be given orally; through a rectal suppository, a small plug of medication inserted in the rectum; or through an enema—liquid medication put into the rectum. Unless the UC symptoms are severe, people are usually first treated with aminosalicylates. These medications are also used when symptoms return after a period of remission.

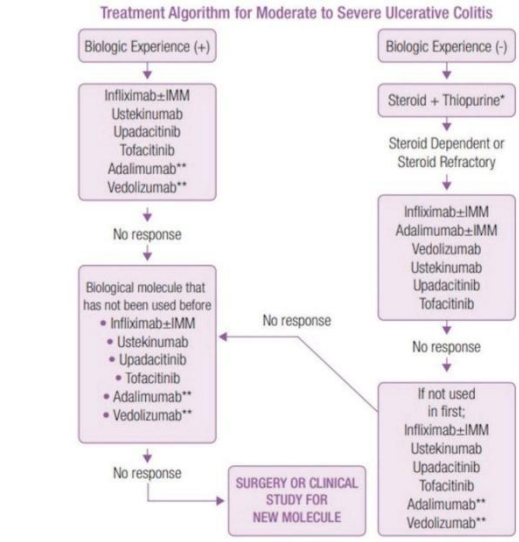


Figure 2. Treatment Algorithm for Moderate to Severe Ulcerative Colitis.

7.2. Infliximab: (Remicade) is one of the agents that fall under the category of anti-tumor necrosis factor (anti-TNF) that are recommended for use in patients with UC who fail or cannot use the other medications or 5-ASAs. Patients on Infliximab must also be given immunomodulators, which are used for the prevention of the allergic reactions. This immunosuppressive drugs main action is to inhibit a type of protein called TNF from binding with its receptor, which prevents further intestinal inflammation. The drug is administered intravenously, meaning it is directly infused into the bloodstream through a catheter-that is connected to a vein in the arm- and it is done every 6 to 8 weeks at either the hospital or outpatient settings. Some may have the side effects of toxicity and becoming more susceptible to infections, especially tuberculosis. Different medicines can be taken to control the symptoms of anxiety or pain, to defer other problems like diarrhoea, or to get rid of the infection.

7.3. Immunomodulatory Azathioprine, immunomodulators such as (Imuran, Azasan) 6-mercaptopurine (6-MP) (Purinethol), and cyclosporine (Neoral, Sandimmun, Sandimmune), respectively, are agents that have the function of the immune system. They are used for patients who do not respond to cocosteroids- AZAs. Immunomodulators are prescribed per os, but they have slow onset of action and can take 3-6 months to become effective. Complications such as nausea, vomiting, fatigue, pancreatitis, hepatitis, a decreased WBC count, and an increased risk of infection are observed in the people taking these drugs are monitored. Cyclosporine, is used only for extremely sick UC cases, because the frequent side effect of it is toxicity, meaning it can cause long-term effects on the body. In addition, medication has the side effect of serial toxicity which indicates that it causes terrible consequences in the human body through time. Protein called TNF from binding with its receptor, which prevents further intestinal inflammation. The drug is administered intravenously, meaning it is directly infused into the bloodstream through a catheter-that is connected to a vein in the arm- and it is done every 6 to 8 weeks at either the hospital or outpatient settings. Some may have the side effects of toxicity and becoming more susceptible to infections, especially tuberculosis. Different medicines can be taken to control the symptoms of anxiety or pain, to defer other problems like diarrhoea, or to get rid of the infection.

Table 2 Comparison of UC Treatment Strategies

Treatment Category	Representative Agents	Indication	Mechanism of Action	Efficacy Highlights	Key Adverse Events
Aminosalicylates	Mesalazine, Sulfasalazine	Mild to moderate UC	Anti-inflammatory via inhibition of prostaglandin & leukotriene synthesis	Induction of remission in >50% of mild cases	Headache, nausea, nephrotoxicity (rare)
Corticosteroids	Prednisolone, Budesonide	Moderate to severe flares	Broad immunosuppression via glucocorticoid receptor activation	Rapid symptom control in acute exacerbation	Weight gain, osteoporosis, hyperglycemia
Immunomodulators	Azathioprine, 6-MP	Steroid-sparing maintenance	Purine analog; inhibits DNA synthesis & lymphocyte proliferation	Remission maintenance in ~40% of cases	Leukopenia, hepatotoxicity, infection risk
Anti-TNF Biologics	Infliximab, Adalimumab	Moderate to severe UC, anti-TNF naive	Neutralizes TNF-alpha, reducing inflammation	Up to 60% response at induction; ~30% remission	Infusion reactions, infection, malignancy risk
Anti-Integrin Agents	Vedolizumab	Anti-TNF failures or intolerant patients	Blocks α4β7 integrin; gut-selective lymphocyte trafficking	Sustained clinical remission up to 45% at 52 weeks	Nasopharyngitis, headache, rare PML (progressive multifocal leukoencephalopathy)
Anti-Interleukin Agents	Ustekinumab, Mirikizumab	Moderate to severe UC	Inhibits IL-12/23 or IL-23 pathways (Th1/Th17 modulation)	Clinical remission ~20–30%; improving durability	URTI, injection site reactions, rare hypersensitivity
JAK Inhibitors	Tofacitinib, Upadacitinib	Moderate to severe UC, rapid control	Blocks JAK-STAT cytokine signaling	Clinical remission up to 40–50% at 8–12 weeks	Herpes zoster, lipid elevation, thromboembolism risk
S1P Modulators	Ozanimod	Moderate UC	Sequesters lymphocytes via S1P1 receptor	~37% remission at week 10 (True North trial)	Bradycardia, liver enzyme elevation
FMT (Fecal Microbiota Transplant)	Capsule/oral/ donor infusion	Refractory or relapsing mild to moderate UC (investigational)	Microbiota restoration; immune modulation	Remission rates very (15–57%); durability variable	GI symptoms, donor-dependent serious AEs (rare)
Emerging Therapies	CRISPR, MSCs, RNAi	Investigational	Genetic or cellular modulation of inflammation	Early-phase promising signals	Long-term safety unknown; immunogenicity

7.4. Corticosteroid: Corticosteroids, prednisolone, methylprednisolone, and hydrocortisone are medications that can also decrease inflammation. They are offered to patients enduring more acute manifestations and those who are not responsive to 5-ASAs. Also referred to as steroids, corticosteroids might be used in various ways; they can be taken by mouth, or infused intravenously, or delivered through an enema, rectal foam, or a suppository depending on what parts of the colon and rectum are influenced by UC. Among the side effects are weight gain, acne, facial hair, hypertension, diabetes, mood swings, impaired bone mass, and a rise in the risk of infection. Today people use these drugs only occasionally because side effects are often harsh; because of this, steroids are not ordinarily recommended for long-term use. Steroids are generally prescribed for a time frame and then stopped once the colon is healed. The remaining medications used for UC are prescribed for persistent manifestation control.

Table. Therapies for Ulcerative Colitis (UC) From Randomized Clinical Trials (continued)

Drug class	Mechanism of action	Use	Dose and administration	Efficacy at primary end point from randomized trials	Adverse effects
Intravenous or subcutaneous					
Infliximab	Human chimeric monoclonal IgG1 antibody against TNF-α	Induction and maintenance in moderate to severe UC.	Dose of 5 mg/kg IV. Induction wk 0, 2, and 6 and then every 8 wk for maintenance. SC formulation for maintenance: 120 mg every other week.	ACT 1 and 2 induction trials (N = 364 and N = 364). Clinical response at wk 8: 69.4% with infliximab (5 mg/kg at 0, 2, and 6 wk) vs 37.2% with placebo, $P < .001$.[60] ACT 1 maintenance trial. Clinical response at wk 54: 45.5% with infliximab (5 mg/kg every 8 wk) vs 19.8% with placebo, $P < .001$.[60]	Injection site or infusion reactions (eg, infusion reactions to infliximab reported in 5%-23%).[61] Increased risk of infections, reactivation of latent tuberculosis, psoriasiform skin changes, drug-induced lupus. Avoid in advanced heart disease due to risk of decompensation and in demyelinating diseases.
Adalimumab	Human monoclonal IgG1 antibody against TNF-α	Induction and maintenance in moderate to severe UC.	Dose of 40 mg SC. Induction SC 160 mg at wk 0, 80 mg at wk 2, 40 mg at wk 4, and 40 mg every other week for maintenance.	ULTRA 2 trial (N = 494). Clinical remission at wk 8: 16.5% with adalimumab (160 mg at wk 0, 80 mg at wk 2) vs 9.3% with placebo, $P = .019$.[62] Clinical remission at wk 52: 17.3% with adalimumab (40 mg every other week) vs 8.5% with placebo, $P = .004$.[62]	
Golimumab	Human monoclonal IgG1 antibody against TNF-α	Induction and maintenance in moderate to severe UC.	Induction first dose: 200 mg SC at wk 0, 100 mg at wk 2. Maintenance SC: 50 mg every 4 wk (weight <80 kg) or 100 mg every 4 wk (weight ≥80 kg).	PURSUIT-SC induction trial (N = 774). Clinical response wk 6: 51% (200 mg SC at wk 0 and 100 mg) with golimumab vs 30.3% with placebo, $P = .001$.[63] PURSUIT-M maintenance trial (N = 464). Clinical response at wk 54: 49.7% with golimumab (100 mg every 4 wk) vs 31.2% with placebo, $P < .001$.[64]	
Vedolizumab	Human monoclonal anti-integrin α4β7	Induction and maintenance in moderate to severe UC.	Dose: 300 mg IV. Induction wk 0, 2, and 6 and then IV 300 mg every 8 wk for maintenance. SC formulation for maintenance: 108 mg every other week.	GEMINI I trial (N = 374). Clinical response at wk 6: 47.1% with vedolizumab (300 mg at wk 0, 2, and 6) vs 25.5% with placebo, $P < .001$. Clinical remission at wk 52: 41.8% with vedolizumab (300 mg every 8 wk) vs 15.9% with placebo, $P < .001$.[65]	Vedolizumab adverse events compared with placebo (9 RCTs of UC and Crohn disease (N = 4268): RR, 1.01 (95% CI, 0.96-1.07).[66] Adverse effects reported in trials: nasopharyngitis, upper respiratory tract infection, arthralgia.[66]
Ustekinumab	Human monoclonal antibody against the p40 subunit, shared by IL-12/23	Induction and maintenance in moderate to severe UC.	Induction, IV first dose: 260 mg (weight ≤55 kg), 390 mg (weight, 55-85 kg), or 520 mg (weight >85 kg). Second dose SC at wk 8, 90 mg. Standard maintenance: 90 mg SC every 8 wk.	UNIFI trial (N = 961). Clinical remission at wk 8: 15.6% with ustekinumab (IV 130 mg or 6 mg/kg at wk 0) vs 5.3% with placebo, $P < .001$. Clinical remission at wk 44: 43.8% with ustekinumab (90 mg SC every 8 wk) vs 24.0% with placebo, $P < .001$.[67]	Serious adverse events (6 RCTs of UC and Crohn disease, N = 2574) of ustekinumab (27.5 per 100 patient-years) not different from placebo (21.23 per 100).[68] Adverse effects reported in trials: headaches, nausea, injection site reaction, joint pain. Increased risk of infections.
Mirikizumab	Human monoclonal antibody IgG4 against the p19 subunit of IL-23	Induction and maintenance in moderate to severe UC.	Induction: IV 300 mg every 4 wk (3 doses) followed by 200 mg SC every 4 wk for maintenance.	LUCENT 1 induction trial (N = 1162). Clinical remission at wk 12: 24.2% with mirikizumab (300 mg every 4 wk x 3 doses) vs 13.3% with placebo, $P < .001$.[69] LUCENT 2 maintenance trial (N = 544). Clinical remission at wk 40: 49.9% with mirikizumab (200 mg every 4 wk) vs 25.1% with placebo, $P < .001$.[69]	Incidence of adverse events during trials similar in mirikizumab and placebo groups. Common adverse events: upper respiratory tract infections, rash and injection site reactions.[69] In a randomized trial, opportunistic infections and cancer observed in a small number of patients taking mirikizumab (n = 23).[69]

7.5. Biological therapies: The human-based and animal-based therapies produced from protein living organisms are. These medications are anti-antibodies that block some proteins in the body from causing inflammation. These medicines are now available in an injectable format or as an intra-venous infusion (through veins). In the list of biopharmaceuticals, one can also find the biosimilar drugs. These are the biosimilars whose aim is to be almost exact copies of other previously approved biologic therapies that are known as the originator drug or reference product. They are identical in safety and efficacy as well as the original medications they are taken the same way.

Table. Therapies for Ulcerative Colitis (UC) From Randomized Clinical Trials (continued)

Drug class	Mechanism of action	Use	Dose and administration	Efficacy at primary end point from randomized trials	Adverse effects
Thiopurines Azathioprine Mercaptopurine	Inhibition of purine synthesis	Maintenance of remission in steroid-dependent UC or in combination with TNF inhibitors to decrease immunogenicity.	1.5-2.5 mg/kg/d for azathioprine 0.75-1.5 mg/kg/d for mercaptopurine Dose adjustment for low thiopurine methyltransferase levels or neutropenia.	Steroid-dependent UC (N = 72). Clinical and endoscopic remission at 6 mo and steroid discontinuation: 53% with azathioprine (2 mg/kg/d) vs 21% with 5-ASA (3.2 g/d), $P = .006$.[50]	Adverse events (5 studies, N = 257) of azathioprine or mercaptorpurine (9%), placebo (2%): RR, 2.82 (95% CI, 0.99-8.01).[51] Adverse events include leukopenia, hepatotoxicity, gastric intolerance, and pancreatitis. Increased risk of lymphoma: 0.50 events per 1000 person-years with azathioprine; aHR, 2.18 (95% CI, 1.38-3.45)[52] and nonmelanoma skin cancer (11 studies; RR, 1.88 [95% CI, 1.48-2.38]).[53]
Tofacitinib	Inhibition of JAK1, JAK2, JAK3, and TYK-2	Induction and maintenance in moderate to severe UC.	Induction dose: 10 mg twice daily orally for 8 wk. Maintenance dose: 5 mg twice daily orally.	OCTAVE 1 and 2 induction trials (N = 598 and N = 541). Clinical remission at wk 8: 18.5% with tofacitinib (10 mg twice daily) vs 8.2% with placebo, $P = .007$ (OCTAVE 1); 16.6% vs 3.6%, $P < .001$ (OCTAVE 2).[54] OCTAVE maintenance trial (N = 593). Clinical remission at wk 52: 34.3% with tofacitinib (5 mg twice daily) vs 11.1% with placebo, $P < .001$.[54]	JAK inhibitors for IBD or other immune-mediated diseases compared with placebo (82 studies, N = 66 159): incidence of serious adverse effects: 9.88 per 100 person-years. Increased risk of herpes zoster (RR, 1.57 [95% CI, 1.04-2.37]).[55] Safety considerations: cytopenia, creatinine phosphokinase level increase, increased risk of vein thrombosis. Monitor cholesterol levels.
Filgotinib	JAK1 inhibitor	Induction and maintenance in moderate to severe UC.	Induction dose and maintenance dose: 200 mg/d.	SELECTION trial (N = 659). Clinical remission at wk 10: 26.1% with filgotinib (200 mg) vs 15.3% with placebo, $P = .016$.[56] Clinical remission at wk 58: 37.2% with filgotinib (200 mg) vs 13.5% with placebo, $P < .001$.[56]	
Upadacitinib	JAK1 inhibitor	Induction and maintenance in moderate to severe UC.	Induction dose: 45 mg/d for 8 wk. Maintenance dose: 15-30 mg/d.	U-ACHIEVE induction trial (N = 474). Clinical remission at wk 8: 26% with upadacitinib (45 mg) vs 5% with placebo, $P < .0001$).[57] U-ACHIEVE maintenance (N = 451). Clinical remission at wk 52: 52% with upadacitinib (30 mg) vs 12% with placebo, $P < .001$.[57]	
Ozanimod	S1P receptor modulator	Induction and maintenance in moderate to severe UC.	Dose titration at induction: days 1-4: 0.23 mg/d orally; days 5-7: 0.46 mg/d; day 8 and thereafter: 0.92 mg/d. Maintenance dose: 0.92 mg/d orally.	TRUE NORTH induction trial (N = 1012). Clinical remission at wk 10: 18.4% with ozanimod (1 mg) vs 6.0% with placebo, $P < .001$.[58] TRUE NORTH maintenance (N = 457). Clinical remission at wk 52: 37.0% with ozanimod (1 mg) vs 18.5% with placebo, $P < .001$.[58]	S1P modulator for IBD or other immune-mediated diseases compared with placebo (26 studies, N = 9604): herpes zoster infection (risk ratio, 1.75 [95% CI, 1.09-2.80]) and bradycardia (risk ratio, 2.64 [95% CI, 1.77-3.96]).[59]

7.6. Janus kinase inhibitors (JAK Inhibitors): These pills that are now on the market have to be taken as they are not absorbed directly into the blood but instead are broken down in the gastrointestinal tract after ingestion and then transported to the bloodstream via the intestinal wall. Since these molecules are found in a very
small size, they get easily dissolved in the blood and can reach any body part even the immune system. In contrast to some of the other oral drugs, these agents are not only faster to act but also can lead to and sustain remission. Their main indication is ulcerative colitis.

Table. Therapies for Ulcerative Colitis (UC) From Randomized Clinical Trials

Drug class	Mechanism of action	Use	Dose and administration	Efficacy at primary end point from randomized trials	Adverse effects
Topical therapy					
Aminosalicylates (eg, mesalamine, mesalazine) Suppositories Foam Enema	Unknown	Induction and maintenance of remission in mild to moderate UC. Suppositories treat up to the upper rectum. Foam formulation can reach the sigmoid colon. Enemas can reach the descending colon.	Topical therapy is recommended at ≥1 g dose/d at least 2-8 wk for induction and the same dose for maintenance.	Mild to moderate UC proctitis (N = 129). Endoscopic remission rates at wk 4: 81.5% with mesalazine (1 g/d) suppositories vs 29.7% with placebo, $P < .001$.[42]	Adverse effects in clinical trial did not differ from placebo.[42]
Steroids Suppositories Foam Enema	Multiple signal transduction pathways	Induction of remission in mild to moderate UC.	Budesonide, hydrocortisone, prednisolone, and beclomethasone preparations available. Used for induction once daily for 2-8 wk in patients with distal UC involvement.	Mild to moderate UC proctitis or proctosigmoiditis (N = 546). Clinical remission at wk 6: 41.2% with budesonide foam (2 mg twice daily) vs 24% with placebo, $P < .001$.[43]	Adverse effects in clinical trials did not differ from 5-ASA or placebo.[43]
Oral therapy					
Aminosalicylates Sulfasalazine (5-ASA bonded to sulfapyridine) Mesalamine	Unknown	Induction and maintenance of remission in mild to moderate UC.	Mesalamine: dose range, 2 g/d to 4.8 g/d orally. Once daily dosing of mesalamine is not different from split dose.[44]	PINCE trial (N = 127). Clinical remission at wk 8: 64% with oral 5-ASA (2 g twice daily) and topical 5-ASA (1 g/d) vs 43% oral 5-ASA alone, $P = .03$.[45] ASCEND III trial (N = 772). High vs low dose of 5-ASA. Clinical remission at wk 6: 43% (4.8 g/d) vs 35% (2.4 g/d), $P = .04$.[46]	Serious adverse events of mesalamine (1%) compared with placebo (2%) were not different (3 studies, N = 826): RR, 0.60 (95% CI, 0.19-1.84).[47] Sulfasalazine: needs laboratory monitoring (blood count, liver and kidney function) and folate supplementation (1 mg/d). Rare adverse effects to aminosalicylates (<1%): hypersensitivity reactions, interstitial nephritis, agranulocytosis, drug-induced lupus, myocarditis, hepatitis.
Oral corticosteroids Budesonide Prednisone Prednisolone	Multiple signal transduction pathways	Budesonide MMX: induction of remission in mild to moderate UC. Prednisone: induction of remission in moderate to severe UC.	Budesonide MMX, 9 mg /d for 8 wk. Prednisone, 1 mg/kg/d, maximum of 40-60 mg/d single dose in the morning tapering dose by 5 mg every week.	CORE I trial (N = 509). Combined clinical and endoscopic remission at wk 8: 17.9% with budesonide MMX (9 mg/d) vs 7.4% with placebo, $P = .014$.[48]	Serious adverse events of budesonide (27 per 1000) compared with placebo were not different (31 per 1000) (2 studies, N = 513): RR, 0.88 (95% CI, 0.33-2.4).[48] Reduction in cortisol levels in budesonide, 10 mg (15 per 1000) lower than 40 mg of prednisolone (758 per 1000): RR, 0.02 (95% CI, 0.00-0.33).[49] Short-term use of prednisone: mood changes, insomnia, weight gain, increase glucose levels, increased risk of infections. Long-term use of prednisone: osteoporosis, peptic ulcer, diabetes, Cushing syndrome, adrenal insufficiency hypertension, cataracts.

TABLE 1 Current clinical treatment for ulcerative colitis.

Treatment	Class	Example	Indication	Ref.
Modern medical treatment	Aminosalicylic acid preparation	Salazine, mesalazine, metronidazole, dexamethasone, prednisone and hydrocortisone	They can effectively improve the gut microbiota environment of patients and prevent the secretion of inflammatory cells, so as to effectively inhibit inflammation.	(48–50)
	Glucocorticoid hormones	Dexamethasone, prednisone, methylprednrone and Budesonide	They have a strong anti-inflammatory effect, has a good effect on UC, but easy to cause dependence.	(51–53)
	Immunosuppressive agents and biologics	Immunosuppressants: azathioprine, mortemycophenol, 6-mercaptopurine, cyclosporine; Biological agents: Infliximab, adalimumab; JAK inhibitor	The first choice for acute severe ulcerative colitis, overinduction and maintenance of inflammatory relief, the combination of the two is better than the single drug effect.	(54–57)
	Microbial preparation	Antibacterial drugs, probiotics, prebiotics and fecal transplants of healthy people	It can inhibit pathogenic bacteria, strengthen intestinal mucosal barrier and regulate immune response.	(58–60)
	Surgical treatment	Subtotal resection of colon, temporary ileostomy	The surgery is designed to restore the patient's health by removing the burden on the colon, but the risk of cancer remains and requires constant testing of the rectum.	(61–63)

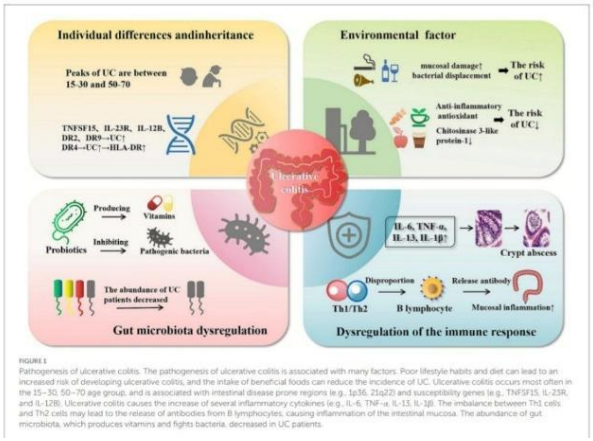


FIGURE 1
Pathogenesis of ulcerative colitis. The pathogenesis of ulcerative colitis is associated with many factors. Poor lifestyle habits and diet can lead to an increased risk of developing ulcerative colitis, and the intake of beneficial foods can reduce the incidence of UC. Ulcerative colitis occurs most often in the 15–30, 50–70 age group, and is associated with intestinal disease prone regions (e.g., 1p36, 21q22) and susceptibility genes (e.g., TNFSF15, IL-23R, and IL-12B). Ulcerative colitis causes the increase of several inflammatory cytokines (e.g., IL-6, TNF-α, IL-13, IL-1β). The imbalance between Th1 cells and Th2 cells may lead to the release of antibodies from B lymphocytes, causing inflammation of the intestinal mucosa. The abundance of gut microbiota, which produces vitamins and fights bacteria, decreased in UC patients.

VIII. Non pharmaceutical therapy

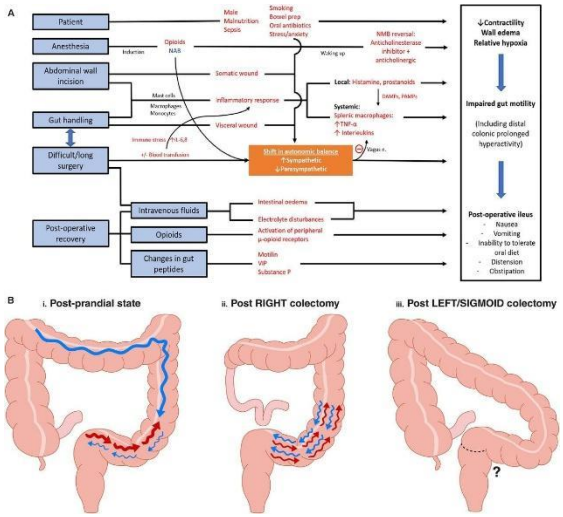
Surgical intervention, being the major problem for the inflammatory bowel disease, has become less important with the development of a new generation of medical therapies. A significant portion of the people who are diagnosed with inflammatory bowel disease will, before long, need to have surgery. However, some of these symptoms may also suggest the need for an operation before the actual diagnosis has been made. Emergency surgery is needed for the cases of toxic megacolon, colonic perforation and refractory hemorrhage while planned operations are for medically refractory disease, medication intolerance, endoscopically unresectable dysplasia, and colorectal cancer. The most common technique is the restoration of the bowel through proctocolectomy and the implantation of an ileal pouch to the rectum with an anal anastomosis (IPAA) because this procedure frees from permanent stoma. To avoid complications such as anastomotic leak, pelvic sepsis, and pouch dysfunction surgery is performed in a staged manner. Those patients with a poor nutritional state or aggressive disease often are candidates for the three-stage protocol: subtotal colectomy with end ileostomy initially, then, completion proctectomy with IPAA and diverting loop ileostomy, finally, closure of the ileostomy. The involvement of complications following colectomy in around 1/3 patients is introduced. Initial issues are brought up as infection and ileus, and later on are presented as pouchitis and faecal incontinence. Despite improvements in technique, about 10% of pouches need to be removed or diverted permanently within a period of 10 years. Instead of operation, it is the long-term healing of the mucosa that has become a central target in UC. The STRIDE-II guidelines advocate the utilization of either sigmoidoscopy or colonoscopy to monitor mucosal healing in the context of a treat-to-target strategy, which joins medical management to objective endoscopic outcomes.

IX. Future therapy

The health-related and social issues of ulcerative colitis stem from its wide range of causes and powerful super viruses as a result tissue viruses. Ulcerative colitis also has a complicated etiology and pathogenesis, which includes genetic disposition, the state of the immune system, the environmental factors, the presence of infectious agents, as well as the imbalance between intestinal flora, which among other elements trigger the disease. Notably, ulcerative colitis has a fairly complex pathos that is not comprehended by health experts yet. It entails the involvement of cytokines that are released from macrophages, the main immune system that gets affected, and the possibility that the dysfunction of these cells is one of the core pathogenesis of the disease. Besides, at present, the most effective instrument in UC diagnosis is the colonoscopy but it is constrained by its being invasive. Medical imaging and other diagnostic tools that will be advanced more include ultrasound, magnetic resonance, calprotectin, and CRP which can actually substitute some tests. As mentioned previously, new targeted therapies that are progressing quickly, such as hormones, cytokines, and immunomodulators, are also coming up with more solutions for UC treatment. Investigating novel bile acid receptor pathways as therapeutic strategies in ulcerative colitis is under progress. Farnesoid X receptor (FXR) is one of the main targets because it is responsible for regulation of mucosal immunity and intestinal barrier function. Agents that activate FXR (farnesoid X receptor) like obeticholic and cilofexor are considered as the techniques to restore epithelial homeostasis through the process of inflammation similar to what drugs, such as multitargets, might do.

X. Prognosis and complications

More often than not, it is the postoperative ileus that these patients face and other conditions like ischuria and wound infections also do accompany them. A similar adverse effect resulting from the inflammatory colitis is the toxic megacolon, which exhibits the presence of small and insignificant colon perforations. A sigmoid perforation was reported by a study as well as colovesicular and colocutaneous fistula diseases in a 43-year-old man, who had a history of UC and the patient underwent repeated surgeries. A deeper look into the circumstances revealed that he had diverticulitis along with UC as the cause of his condition. Only a few instances of perforated diverticulitis associated with UC could be found in medical literature, so the diagnosis is often more challenging. Furthermore, the study showed that the IBD prone people also have high cardiovascular diseases. High-risk postoperative complications have been observed among older patients and those who have more than one underlying illness.



XI. Surgery intervention

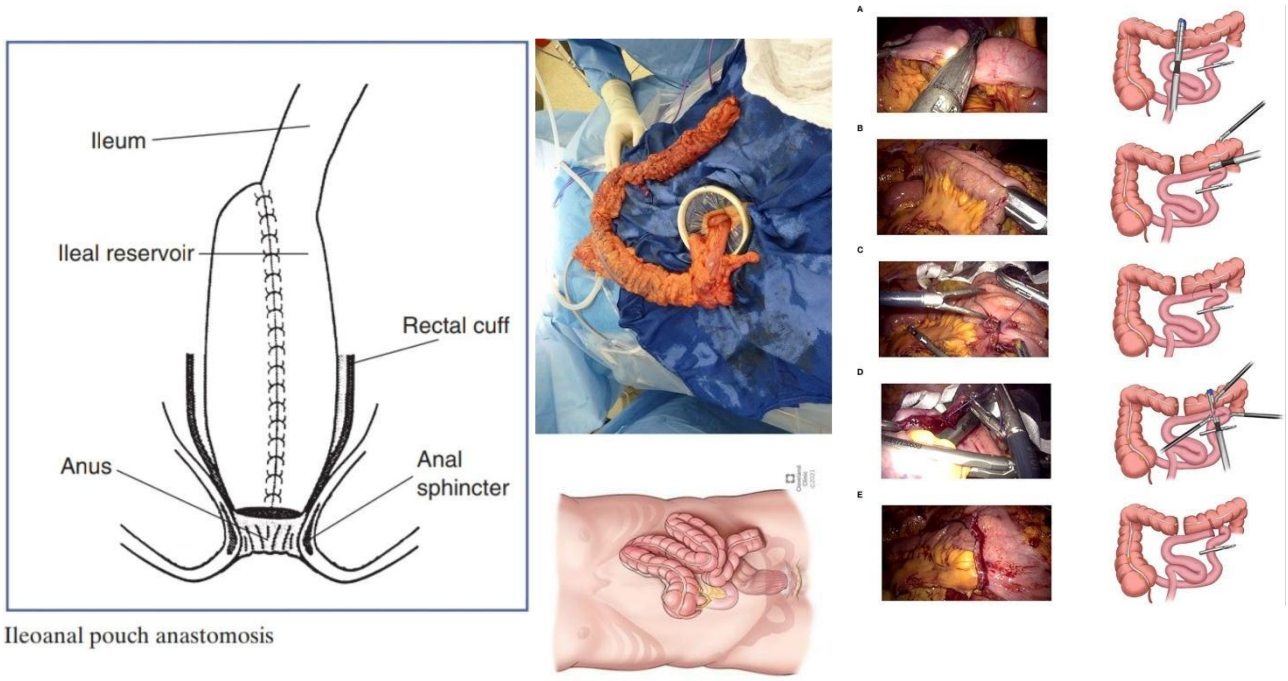
The medication or exercise habits of the patient are the two forms of treatment for the first stages of UC which is medication or good lifestyle practices. Eating chop suey in the cafeteria and having a bad sleep at night are the two causes which encourage a short-lived stress response. According to the survey, as much as 20-30% of the patients with severe ulcerative colitis have either inadequate reaction to medication or have an acute colitis onset that requires surgery. Emergency patients had the surgical indexes complicated with massive gastrointestinal bleeding, intestinal mucosa perforation, complicated with toxic paralysis, megacolon and ineffective medical treatment. The patients undergoing elective surgery were indicated to be complicated with intestinal carcinoma. The target of surgical intervention is to restore the patient's health through the elimination of the load on the colon. The major operations other than the removal of the rectal stump are subtotal colectomy and brief ileostomy. The surgeons' actions are aimed at restoring health and thus help with the patients by giving the burden of the colon. Apart from the rectal stump that has to be removed, the most significant surgical methods are subtotal colectomy and temporary ileostomy. The study has shown that patients with UC who have undergone a long-term illness are more prone to develop colorectal cancer. Many ulcerative colitis individuals have a good reaction to medical treatment and may not even have to go through the operation. Nevertheless, by the end of 30 years, almost one-third of those with ulcerative colitis will have to undergo surgery at some time in their lives. In some cases, the operation is necessary for the treatment of multiple complications. Among these are arterial bleeding of the intestinal ulcer, intestinal perforation (hole), and a toxic megacolon. Surgery also can be discussed if all of the colon and the rectum (a proctocolectomy) are removed when the medicines are not controlling the disease well anymore or when there are precancerous changes in the colon. Ulcerative colitis is an illness that deals with the immune system, and the more frequent signs that occurred after surgery, like joint pain or skin disorders, may be on the list and start again, even if the colon is taken out.



A proctocolectomy is followed by one of the following operations:

11.1. Ileoanal pouch anastomosis, also called "pouch surgery," makes it possible for people with UC to have normal bowel movements, because it preserves part of the anus. For this operation, the surgeon preserves the outer muscles of the rectum during the proctocolectomy. The ileum—the lower end of the small intestine—is then pulled through the remaining rectum and joined to the anus, creating a pouch. Waste is stored in the pouch and passes through the anus in the usual manner. Bowel movements may be frequent and watery than before the procedure. Inflammation of the pouch, called pouchitis, is a possible complication and can lead to symptoms such as increased diarrhea, rectal bleeding, and loss of bowel control. Pouch surgery is the first type of surgery considered for UC because it avoids a long-term ileostomy

11.2. *Ileostomy* is an operation that attaches the ileum to an opening made in the abdomen, called a stoma. The stoma is about the size of a quarter and is usually located in the lower right part of the abdomen near the beltline. An ostomy pouch is then attached to the stoma and worn outside the body to collect stool. The pouch needs to be emptied several times a day. An ileostomy performed for UC is usually permanent. A specially trained nurse will teach the person how to clean, care for, and change the ostomy pouch and how to protect the skin around the stoma. The type of surgery recommended will be based on the severity of the disease and the person's needs, expectations, and lifestyle. People faced with this decision should get as much information as possible by talking with their doctors; enterostomal therapists, nurses who work with colon surgery patients; other health care professionals; and people who have had colon surgery. Patient advocacy organizations can provide information about support groups and other resources.



Ileoanal pouch anastomosis

XII. Follow up & Monitoring

The best way to create patient understanding and engagement in their care is to be clear and straight-to-the-point. Clinical parameters should be evaluated every 3 months during the period of ongoing clinical activity. Following the cessation of symptoms clinical assessments should be conducted every 6-12 months. Colonoscopies should be done every 1-5 years for suspected flares and to make sure of colorectal dysplasia with the use of dye-spray chromoendoscopy preferred. Unquestionable each time in the case of acute severe ulcerative colitis (as per Truelove and Witts), the essential part is intravenous steroids. For those unable to respond to intravenous steroids, a day 3 decision is essential, using some of the different indices as a guideline. Otherwise, either ciclosporin or infliximab may become the rescue medicine, it is worth noting that even trials have not observed any efficacy difference between the two options. Noteworthy, at the time of considering rescue therapy, it is crucial to look at surgical options too.



XIII. Diet, and Nutrition For UC

Dietary changes may help reduce UC symptoms.
A recommended diet will depend on the person's symptoms, medications, and reactions to food. General dietary tips that may alleviate symptoms include:
• eating smaller meals more often
• avoiding carbonated drinks
• eating bland foods
• avoiding high-fibre foods such as corn and nuts
For people with UC who do not absorb enough nutrients, vitamin and nutritional supplements may be recommended.

XIV. Conclusion

The end of the article is about the management of UC that is a particular stage across its remission induction to maintenance and acute treat-can be done only by whole individual-made method related to the severity of the disease and the patient's reaction to a particular therapy. In the case of mild to moderate UC, oral mesalazine at higher doses and colonic-released budesonide have shown efficacy, and combined therapies are the best option compared to monotherapies. For patients who show inadequate response, the additional topical steroids and the use of systemic corticosteroids, on the other hand, are the possible alternatives, while the use of thiopurines is not recommended because they have a longer period of onset. In the case of chronic treatments, the use of the same type of mesalazine formulations is crucial in reducing the risk of relapses, while the use of thiopurines is an option only for patients who are intolerable to mesalazine. In moderate to severe UC, the introduction of biologic therapies such as anti-TNF agents, vedolizumab, ustekinumab, and oral small molecules such as Janus kinase inhibitors has resulted in disease control for patients who are steroid refractory or dependent. For acute severe UC, prompt initiation of corticosteroids and multidisciplinary management, particularly in collaboration with IBD surgeons, remains crucial, with salvage options such as infliximab and cyclosporine available for patients who do not respond. Overall, effective treatment requires close monitoring and an individualized approach to achieve optimal clinical and endoscopic responses and improve the quality of life for patients with UC.

Summary of UC

Points to Remember

• Ulcerative colitis (UC) is a chronic, or long-lasting, disease that causes inflammation and sores, called ulcers, in the inner lining of the colon and rectum.

• Ulcerative colitis is one of the two main forms of chronic inflammatory disease of the gastrointestinal tract, called inflammatory bowel disease (IBD).

• The most common symptoms of UC are abdominal discomfort and bloody diarrhoea.

• Flexible sigmoidoscopy and colonoscopy are the most accurate methods for diagnosing UC and ruling out other possible conditions, such as Crohn's disease, diverticular disease, or cancer.

• Treatment for UC depends on the severity of the disease and its symptoms. Each person experiences UC differently, so treatment is adjusted for each individual. Treatment may include medication therapy, hospitalization, or surgery to remove the rectum and part or all of the colon.

• Most people with UC never develop colon cancer, but two factors that increase the risk are the duration of the disease and how much of the colon is affected.

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