# A REVIEW OF ULCERATIVE COLLITUS

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## ABSTRACT

Ulcerative colitis (UC) is one of the most common inflammatory bowel diseases Characterised by continuous inflammation of inner layer of the large intestine. It is a subcategory of inflammatory bowel disease, afflicts 1-2 million people in the United States, and many more worldwide. Doctors usually start with medicine called 5-aminosalicylate acid for people with mild to moderate symptoms. If this medicine does not work well enough, Management aims to achieve rapid resolution of symptoms, mucosal healing and improvement in a patient's quality of life. Ulcerative colitis (UC) is a biologically complex condition characterized by chronic, relapsing inflammation of the gastrointestinal tract. An Integrative Therapeutic approach encompassing homeopathic, ayurvedic , and allopathic interventions, complemented by nutritional modulation and structured physical activity, plays a pivotal role in the prevention and holistic management ulcerative colitis. In moderate-to-severe UC, corticosteroids are usually used in induction of remission with or without another class of medications such as thiopurines or biologics including anti-tumor necrosis factor, anti-integrins, or Janus kinase inhibitors for main

## *INTRODUCTION*

The term ulcerative colitis (UC) was initially used in the middle of the nineteenth century. One The rectum is frequently affected by this idiopathic, chronic inflammatory disease of the colonic mucosa, which can also spread in a continuous and proximal manner to incorporate additional colonic regions. Ulcerative colitis (UC) is a chronic illness characterized by recurrent, uncontrollable colon inflammation (1). Inflammation always affects the rectum, extending from the distant from the colonic segments that are proximal. UC is becoming more commonplace worldwide, and its patients frequently suffer from crippling symptoms like diarrhea, abdominal pain, rectal bleeding, extra-intestinal manifestations, and a higher risk of colorectal cancer. Ulcerative colitis (UC) is a long-lasting disease that goes through cycles of improvement and worsening. It can develop from mild inflammation that doesn't show symptoms to severe inflammation of the colon. This can lead to frequent bloody stools, problems with bowel movements, possible permanent scarring and damage to the tissue, overall body symptoms, and sometimes the need for surgery (2-6). Ulcerative colitis (UC) extends beyond being a simple gastrointestinal disorder; it significantly affects patients' daily lives, emotional health, and their overall well-being. Those living with UC frequently face a complex range of symptoms, including abdominal discomfort, diarrhea, severe fatigue, and unintended weight loss. The disease's episodic nature, marked by cycles of flare-ups and remissions, creates additional challenges by instilling a sense of unpredictability and concern for both the individuals afflicted and their medical care teams. This uncertainty often impacts treatment decisions and the psychological state of patients managing UC. (13,14) The global incidence and prevalence of UC have been increasing, particularly in developing countries, due to changes in diet, lifestyle, and environmental exposures. The precise mechanism of the disease is not clearly known

but there are hereditary factors that are included in the risk of developing the disease with epithelial barrier defects and environmental factors. Currently, a number of genetic and environmental factors that raise the risk of developing UC are Established. (17)

## KEYWORDS

Ulcerative colitis, Bowls Imflammatory Disease, Management of Treatment

## ULCERATIVE COLITIS

Ulcerative Colitis (UC) is a chronic inflammatory condition belonging to the group of disorders known as **Inflammatory Bowel Diseases (IBD)**. IBD encompasses diseases that arise from an **abnormal immune reaction against the gut microbiota**, resulting in **recurrent inflammation of the gastrointestinal tract**.

The term IBD primarily refers to **two idiopathic intestinal disorders** that vary in their **site of involvement and the depth of inflammation within the intestinal wall**:

1. **Ulcerative Colitis (UC)**
2. **Crohn's Disease**

In Ulcerative Colitis, inflammation is typically **confined to the mucosal layer of the colon**, distinguishing it from Crohn's disease, which can affect any segment of the gastrointestinal tract and involve deeper tissue layers. While most patients can distinguish between the two disorders, around 10% of patients have such similar symptoms that initial differentiation is challenging. Aside from the GI tract, both Crohn disease and ulcerative colitis have numerous manifestations outside the intestines. CD is also categorized based on presentation inflammatory, structuring, or penetrating. Both conditions are categorized based on severity (mild, moderate, or severe) and location. Crohn disease (CD) causes full-thickness ulceration in any part of the gastrointestinal tract (GI), typically affecting the end of the small intestine and colon. (4-7)

## PATHOPHYSIOLOGY

The pathophysiology of ulcerative colitis involves issues with the epithelial barrier, immune response, leukocyte recruitment, and colon microflora. The epithelial barrier has problems with colonic mucin and possibly tight junctions, which leads to increased absorption of luminal antigens. The lamina propria of the mucosa has more activated and mature dendritic cells, including many toll-like receptors (TLR), specifically TLR2 and TLR4. There also seems to be an unusual T-helper (Th) cell response in patients with ulcerative colitis, particularly Th2, which targets epithelial cells in a cytotoxic manner. Other immune-related factors relevant to ulcerative colitis include tumor necrosis factor-alpha (TNF-alpha), interleukin 13, and natural killer T-cells. Levels of IgM, IgA, and IgG rise in inflammatory bowel disease, but patients with ulcerative colitis show a disproportionate increase in IgG1 antibodies. Leukocyte recruitment faces two main issues. There is an increased release of the chemoattractant CXCL8 in ulcerative colitis, which helps recruit leukocytes to the mucosa from the bloodstream. Also, mucosal cellular adhesion molecule-1 (Mad-CAM1) is more prevalent on the endothelium of mucosal blood vessels, promoting leukocyte adhesion and movement into mucosal tissue. Research indicates that enteric microflora plays a crucial role in the inflammation and severity of the disease. Ulcerative colitis may also stem from an imbalance between enteric microflora and the host's mucosal immunity, leading to an abnormal response to non-pathogenic bacteria. (18-19)

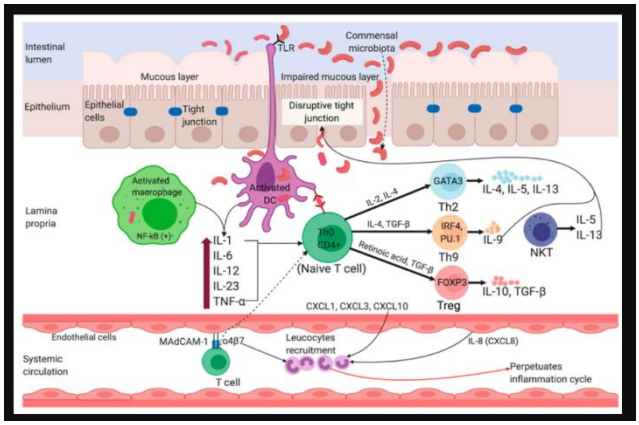


Figure 1 Pathogenesis of inflammatory bowel disease

Source: Ralston S, Penman I, Strachan M, HobsonR, Editors. Davidson's Principles of Medicine. 23rd ed. International edition; 2018, P-815

## TYPES OF ULCERATIVE COLITIS

### 1. Ulcerative Proctitis

Ulcerative proctitis is the **localized and mildest variant** of Ulcerative Colitis (UC), in which the **inflammatory response is restricted to the rectal mucosa**. Patients frequently present with **rectal bleeding, a sense of urgency, and tenesmus**, which refers to a constant feeling of incomplete evacuation. Although limited in extent, this form can **severely disrupt daily activities and psychological well-being** of affected individuals [11].

**2. Distal or Left-Sided Colitis**

Left-sided colitis, also called **distal colitis**, involves inflammation extending **from the rectum through the sigmoid and descending colon**. Common manifestations include **persistent diarrhea with blood, abdominal discomfort, and progressive weight reduction**. This form of UC generally exhibits **moderate severity** and often requires **escalation of treatment intensity** compared to proctitis, sometimes involving **systemic corticosteroids or immunomodulators** [7,12].

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**3. Extensive Colitis (Pancolitis)**

Pancolitis, or **extensive colitis**, represents a **diffuse inflammatory process affecting the entire colon**. The clinical picture is typically dominated by **frequent loose stools, severe abdominal pain, pronounced fatigue, and significant weight loss** [13]. Because of its wide distribution, this variant carries a **greater likelihood of complications**, including **colorectal neoplasia**. Management often necessitates **early use of biologic therapies** and **long-term monitoring** for disease progression [8,9].

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**4. Acute or Fulminant Colitis**

Fulminant colitis is an **acute, severe, and rapidly progressing** form of UC that can become **life-threatening** if not promptly treated. It involves **extensive mucosal damage throughout the colon**, accompanied by **fever, dehydration, abdominal swelling**, and occasionally, **toxic megacolon**. Due to its critical nature, patients require **immediate hospital admission**, **intravenous corticosteroids**, and **urgent surgical consultation** if medical therapy fails [10].

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**5. Pediatric-Onset Ulcerative Colitis**

Pediatric-onset UC is identified when the disease manifests **before the age of 18 years**. Children and adolescents often display **more extensive colonic involvement** than adults, along with **growth retardation, delayed puberty, and nutritional deficiencies**. The disease also poses **emotional and developmental challenges**, making a **multidisciplinary management approach**—including pediatric gastroenterologists, nutritionists, and mental health professionals—essential for optimal outcomes and long-term quality of life (8-12).

# CAUSES AND RISK FACTORS

- **Immune system malfunction:** In UC, the immune system mistakenly attacks the lining of the colon, causing chronic inflammation.
- **Genetic factors:** Having a family history of UC or other inflammatory bowel diseases increases risk.
- **Environmental triggers:** Western diets, pollution, and certain lifestyle habits may contribute to the disease.
- **Age and ethnicity:** It commonly appears between ages **15–30** and again after **60**, and is more frequent among people of **Ashkenazi Jewish descent**. (20)

# COMMON SYMPTOMS

UC symptoms can vary in intensity and often appear in cycles of flare-ups and remission:

- **Abdominal pain and cramping.**
- **Chronic diarrhea**, often urgent or frequent.
- **Rectal bleeding and mucus in stool.**
- **Weight loss and fatigue** due to poor nutrient absorption.
- **Fever and anemia** from ongoing inflammation and blood loss. (20)

# TREATMENT OF ULCERATIVE COLITIS:

1) Allopathy
2) Ayurvedic

3) Homeopathy
4) Exercise and Diet for prevention

# TREATMENT IN ALLOPATHY

The nature of the condition presents a framework for its management. It is possible for patients to have mild, moderate or severe disease. The classification of the clinical severity distinguishes the subsequent medical and pharmacological management. The goals of management are to induce remission and then maintain remission with as few corticosteroids as possible. Historically, the Truelove and Witts criteria have been used to classify mild, moderate, severe, and fulfilling ulcerative colitis . A patient with mild clinical disease has less than four stools per day with or without blood and there are no signs of systemic toxicity. Common clinical symptoms include mild crampy abdominal pain and tenesmus. A patient with moderate-severe disease will have abdominal pain, frequent loose bloody stools (generally >4 per day), and has mild anemia that does not require blood transfusions. They have only minimal signs of systemic toxicity, such as low grade fever. Patients with fulfilling disease will have >6 loose bloody stools daily with severe abdominal cramps, and signs of systemic toxicity, such as fever or tachycardia. They will likely have other signs of inflammation such as anemia or an elevated ESR or CRP. The Truelove and Witts criteria classification of a patient also provide appropriate assessment of the colon. At diagnosis, it is helpful to know what portion of the colon is inflamed, as recommended by the Truelove and Witts criteria, as this can affect future medical and therapeutic management. Generally, patients with left sided or extensive disease will require medical treatment and therapy for a longer duration than patients with proctitis symptoms. (21-22)

## Mild to Moderate Ulcerative Colitis (UC)

- **Frequency:** <4–6 bowel movements/day
- **Bleeding:** Mild–moderate rectal bleeding
- **Systemic signs:** No fever or tachycardia
- **Lab findings:** Normal inflammatory markers and no anemia

**Endoscopic Definition**

- Mucosal **erythema**, **decreased/absent vascular pattern**, **friability**, and **erosions**.

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**First-line therapy:**
**Mesalamines (5-ASA)** — for **induction of remission.**

**Formulations: Oral mesalamine, Suppository, Liquid enema.**

| UC Type | Mesalamine Formulation | Dose & Notes |
|---|---|---|
| **Proctitis (rectum)** | Suppository | 1 g/day at bedtime (retain 1–3 hrs) |
| **Left-sided UC (up to splenic flexure)** | Oral 2–3 g/day + Enema 4 g/day | Enema at bedtime, retain ~8 hrs |
| **Extensive UC (beyond splenic flexure)** | Oral 2–3 g/day + Enema 4 g/day *or* Suppository 1 g/day | Combination therapy recommended |

**Efficacy**

- Clinical response in **40–70%** within **14 days**
- Full remission may take **up to 8 weeks (23-26)**

## Moderate-Severe Ulcerative Colitis

Clinically, Moderate-Severe Ulcerative Colitis (UC) is defined as 4–6 bowel movements per day with moderate-severe rectal bleeding (no constitutional symptoms or signs). Endoscopically, moderate-severe UC is defined as marked mucosal erythema, absent vascularization, friability, granularity, spontaneous bleeding, and ulcerations. At the time of this writing, currently, approved agents for induction and maintenance of remission for moderate-severe UC include the biologics infliximab, adalimumab, golimumab, vedolizumab, and us tekinumab, as well as the small

molecule Janus kinase (JAK) inhibitor tofacitinib. Generally, for all patients starting these agents and immunomodulators, appropriate pre-initiation safety labs (and vaccinations) should be completed, although these may not be feasibly completed with an acute presentation or because follow-up care was deferred due to continued healthcare maintenance needs for the patient.

Therapeutic Class Mechanism of Action Formulation

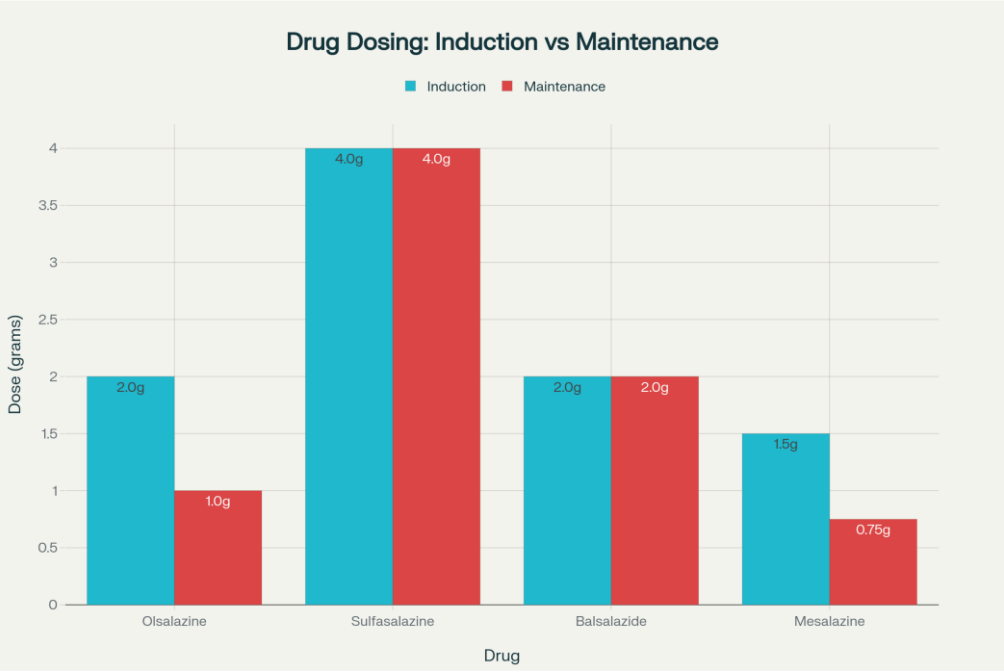
Anti-TNF agents Monoclonal antibodies directed against TNF-alpha, an inflammatory cytokine Infliximab Intravenous Infusion Adalimumab Subcutaneous Golimumab Subcutaneous Anti-integrin agents Monoclonal antibody directed against 47 cell surface glycoprotein on B and T lymphocytes Vedolizumab Intravenous Infusion Anti-IL agents Monoclonal antibody directed against p40 subunit of IL-12 and IL-23 Ustekinumab Intravenous, (1st dose) Subcutaneous JAK inhibitors Small molecule janus kinase 1 and 3 inhibitor.(27-32)

Olsalazine (Dipentum): 250 mg tablets, 5-ASA dimer linked by azo-bond, delivered to colon.Dose: 2–3 g induction, 1 g maintenance.

Sulfasalazine (Azulfidine/Salazopyrin): 500 mg tablets (200 mg 5-ASA), 5-ASA linked tosulfapyridine by azo-bond, delivered to colon. Dose: 4–6 g (0.8–1.6 g 5-ASA) induction andmaintenance.

Balsalazide (Colazal/Colazide): 750 mg tablets (267 mg 5-ASA), 5-ASA linked to 4-aminobenzoyl-beta-alanine by azo bond, delivered to colon. Dose: 2–6.75 g (0.7–2.4 g 5-ASA) induction and maintenance.

Mesalazine (various brands including Asacol, Salofalk, Pentasa, Lialda, Mezavant): multiple formulations with coatings like Eudragit-S, Eudragit-L, Multi Matrix system targeting ileum, colon, sigmoid, rectum, and small bowel. Dose varies between 1.5–6.75 g induction and0.75–4.5 g maintenance depending on product. This organizes critical drug information for treatment phases in inflammatory bowel disease. Flowchart of drugs for induction and maintenance of remission in inflammatory bowel disease




# TREATMENT IN AYURVEDIC

Inflammatory gastrointestinal disorders are widespread, and exploring the anti-inflammatory benefits of Ayurvedic herbs and dietary practices may offer safer, long-term alternatives to conventional treatments. However, current scientific evidence remains limited, as few peer-reviewed studies have evaluated the effectiveness of Ayurvedic nutrition and herbal therapies for adult GI inflammation. This review focuses solely on Ayurvedic dietary and herbal approaches, excluding aspects like mental health, physical activity, or dosha-based treatments.

Ayurveda is a traditional system of medicine developed in India more than 3,000 years ago, which emphasizes the balance of mind, body, and spirit. Ayurveda emphasizes longevity and prevention of disease using the body's natural healing capacity [33]. In Ayurveda, disease mechanisms and inflammation are believed to be a consequence of imbalance of the life forces, or doshas. There are three doshas: vata, pitta, and kapha, and a specific ratio of each one of them is present in the body, which varies from individual to individual [34]. The imbalance of doshas can cause specific health disorders, for which Ayurveda focuses on changing diet (Ahar) and food (Anna) to restore or re-

establish the balance of an individual's dosha. In Ayurveda, foods are not specifically good or bad, but all foods are seen to have either an aggravating or pacifying effect on every dosha category depending on their distinct morphological characteristics and physiological impacts [35,36]. A few examples of universal suggestions for healthy eating habits are: consuming freshly cooked hot meals, using herbs and spices to harmonize foods in a meal, refraining from opposing mixtures like milk with bananas, as well as consumption with a relaxed, peaceful, and attentive mind [33].

Herbs and spices also have a crucial role to play in Ayurvedic dietary guidelines, and they serve not only to add taste but also to balance the food by their capacity to counteract inflammation, regulate gut microbiome, and defend against infection. Ginger, for instance, will not just balance the dense characteristics of a food like pork, but is also utilized to assist in the treatment of diseases like indigestion, vomiting, cold, and cough [33]. The herbs slippery elm and licorice, and a well-known polyherbal medicinal preparation (common name: triphala), were observed to possess strong prebiotic characteristics and thus a deep influence on the structure of the gut microbiota [37]. Ayurveda makes extensive medicinal plants for the management of disease. Guggulutikthaka gritha is also an herbal drug employed as a remedy for long-standing inflammatory diseases, and it has been proven to be effective in its capacity to regulate hyperglycemia, hyperlipidemia, and inflammation in dyslipidemic rats [38]. Furthermore, some GI inflammatory diseases, such as peptic ulcer disease, are primarily triggered by an imbalance in the offensive and protective components of the gut mucosal epithelium and are treated with herbs such as Acacia arabica in order to restore the equilibrium [39,40]. Based on the numerous side effects arising from the use of conventional drugs , most medicinal plants have provided an alternative, natural method of healing GI inflammatory diseases [40].

**Herbal and Formulation-Based Treatment**

**a. Kutaja (Holarrhena antidysenterica)**

- Acts as a natural anti-diarrheal and anti-inflammatory agent.
- Active constituents: Conessine and conessidine possess antimicrobial and astringent properties.
- Clinical studies show improvement in stool frequency and reduction in mucosal bleeding.[41]

**b. Bilva (Aegle marmelos)**

- Useful in chronic diarrhea and dysentery.
- Acts as *Grahini* (absorbent) and *Pitta-shamak* (reduces inflammation).
- Contains tannins and marmelosin, which provide mucosal protection.[42]

**c. Nagakesara (Mesua ferrea)**

- Reduces bleeding due to its *Rakta-stambhaka* (hemostatic) property.
- Also exhibits anti-inflammatory and antioxidant activities.[43]

**d. Yashtimadhu (Glycyrrhiza glabra)**

- Possesses potent anti-ulcer and mucoprotective effects.
- Glycyrrhizin enhances mucosal healing and reduces inflammation in UC.[44]

**e. Amalaki (Emblica officinalis) and Haritaki (Terminalia chebula)**

- Used in *Triphala churna*, beneficial for gut cleansing and antioxidant protection.
- Regular use promotes bowel regularity and reduces oxidative stress.[45]

**3. Panchakarma Therapy**

Ayurveda recommends **Panchakarma detoxification** for long-term UC management:

- **Virechana (therapeutic purgation)** with *Trivrit lehya* for Pitta elimination.
- **Basti (medicated enema)** using *Pitta-shamak* decoctions like *Bilvadi taila* or *Mustadi kwatha*.
- **Takra dhara (buttermilk pouring therapy)** helps soothe intestinal mucosa.[46]

**4. Dietary Management (Pathya–Apathya)**

- **Pathya (Recommended):**
  - Buttermilk (*Takra*), rice gruel (*Peya*), pomegranate juice, and boiled vegetables.
  - Curd, ghee, and cow's milk are soothing and Pitta-pacifying.[47]
- **Apathya (Avoid):**
  - Spicy, sour, fried, and fermented foods.
  - Caffeine, alcohol, and stress-inducing habits.[48]

## Limitations

Ayurvedic medicine can be a powerful tool in managing inflammatory conditions because many aspects of Ayurveda aim to heal inflammation. However, conducting clinically based research on proper Ayurvedic medicine can be difficult due to numerous reasons. Many of the clinical trials explored in this study did not include treatment delivered by an Ayurvedic doctor. Moreover, most of the treatments in the studies used a *one-size-fits-all* formula, whereas actual Ayurveda focuses on individualized care based on patients' symptoms and body type. Also, the studies did not showcase a comprehensive Ayurvedic diagnosis and treatment that encompasses lifestyle, food quantity and composition, and spiritual practice changes, but rather studied the effects of herb extracts on clinical symptoms or modulation of inflammatory markers.

## Anti-inflammatory Effects

The body's inflammation is a critical driver of the progression of IBD. A number of researches employed inflammatory markers to provide evidence on the progression of IBD. TNF-α and IL-6 are primary proinflammatory cytokines that are more significantly related to CD. The researches proved a range of Ayurvedic treatment and their ability to decrease these inflammatory biomarkers. Herbs like Sesbania grandiflora showed a reduction in quantified levels of TNF-α by 3.97% and IL-6 by 15.89%\in acetic acid-induced UC mice [38]. In another study, the methanol fraction of Cordia dichotoma bark's use effectively reduced the production of both TNF-α and PGE2. These groups also had good healing and reduced infiltration by neutrophils and other inflammatory cells [51]. In addition, one study showed how the hemoglobin rose and inflammatory markers such as ESR decreased with the use of polyherbal ayurvedic preparations [39]. A polyherbal preparation of some strong Ayurvedic plants such as Eryngium foetidum (Burma dhaniya), Manilkara zapota (Sapota), and Murraya koenigii (curry leaves) brought down signs of inflammation by lowering myeloperoxidase (MPO) activity through its antioxidant constituents such as alkaloids and flavonoids [37]. Terminalia arjuna hydro-alcoholic extract administration alleviated the disease process in 2,4,6-trinitrobenzene sulfonic acid (TNBS)-induced colitis by inhibiting expression of pro-inflammatory cytokines and chemokines such as MPO, malondialdehyde, and nitric oxide, inhibiting oxidative stress, and enhancing plasma zinc concentration and composition of gut microbiota [52].

Curcumin, which exists in turmeric, is an anti-inflammatory natural product with nontoxicity offering an alternative for modulating inflammatory disorders. Two studies investigated the impact of curcumin as a possible treatment for IBD. In another study, pretreatment with curcumin caused significant suppression of inflammatory mediators such as Interferon-gamma (IFN-g), IL-12, p40 mRNA expression, and iNOS mRNA induction with minimal induction of IL-4 mRNA in TNBS-treated mice [50]. Curcuminoids have been shown in one study to cause improvement in symptoms like decreased frequency of urgent defecation, better patients' self-reported well-being, and decreased clinical activity of UC [49].

# TREATMENT IN HOMEOPATHY

Ulcerative colitis (UC) is a relapsing and remitting chronic inflammatory disorder of the colon, primarily affecting the mucosal layer. Its etiology involves genetic predisposition, immune dysregulation, environmental triggers, and gut microbiota imbalance (Ungaro et al., 2017). Conventional pharmacologic treatment includes aminosalicylates, corticosteroids, immunosuppressants, and biologic agents (Harbord et al., 2017). However, due to adverse effects and incomplete remission rates, many patients turn to complementary approaches like homeopathy (Rahman et al., 2020).

Homeopathy, developed by Samuel Hahnemann in the 18th century, is based on the principles of **"similia similibus curentur"** (like cures like) and **potentization** of substances. It focuses on individualized treatment based on the patient's totality of symptoms (Bell et al., 2013). In chronic conditions like UC, homeopathy is often used as adjunct therapy.

| Remedy | Characteristic Symptoms | Indication |
|---|---|---|
| **Aloe socotrina** | Urgent, involuntary stool with rectal burning; mucus and blood present | Frequent, small, painful stools with weakness |
| **Arsenicum album** | Burning pain, restlessness, anxiety, thirst for small quantities | Severe diarrhea with prostration |
| **Mercurius corrosivus** | Bloody, slimy stools with tenesmus and rectal pain | Hemorrhagic inflammation |
| **Phosphorus** | Bright red bleeding, craving for cold drinks, easy fatigue | Profuse bleeding and prostration |
| **Kali bichromicum** | Tenacious mucus, rectal pain, localized ulceration | Chronic mucus discharge |
| **Sulphur** | Burning, itching anus, offensive stool | Chronic or relapsing UC |
| **Natrum muriaticum** | Periodic symptoms, emotional suppression, rectal bleeding | Stress-related exacerbation |

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