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Review Article

Botanical Remedies for Managing Inflammatory Bowel Diseases

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ABSTRACT

Millions of people worldwide suffer from inflammatory bowel disease (IBD), a chronic inflammatory gastrointestinal disorder that includes Crohn's disease and ulcerative colitis. Although the exact cause is unknown, a complex interaction of immunological, environmental, and genetic variables is believed to be responsible. The incidence of IBD has increased over the past century, especially in industrialized countries, most likely as a result of environmental factors such as food, antibiotic usage, and urbanization that cause imbalances in the gut microbiota. Stress and smoking are also recognized to impact the course of disease; smoking worsens Crohn's disease while perhaps preventing ulcerative colitis. Aminosalicylates, corticosteroids, immunosuppressants, and biologics are examples of conventional allopathic therapies that try to lower inflammation, cause remission, and avoid relapses. Long-term adverse effects of these drugs, such as heightened susceptibility to infections and bone marrow suppression, are still a worry. Herbal remedies have gained popularity as adjunct therapy for IBD in recent years. Herbal plants with anti-inflammatory qualities and potential for symptom treatment include boswellia, aloe vera, and curcumin (found in turmeric). These natural medicines present a promising supplement to allopathic treatments, despite the lack of clinical data. This study highlights the need for integrated management techniques to maximize patient outcomes by looking at the pathophysiology, history, and environmental triggers of IBD and offering a thorough assessment of both conventional and herbal therapy options. The herbal medicinal plants have less side effects as compared to other therapies and as we know that medicinal property of plants are used worldwide. Nowadays novel approaches are also utilizing in herbal formulation to enhance boost bioavailability and effectiveness.

INTRODUCTION

Inflammatory bowel diseases is a chronic conditions characterized by inflammation of the gastrointestinal tract. IBD comprises two

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categories: Crohn's diseases and ulcerative colitis. It occurs in genetically predisposed individuals after an exaggerated immune response to a normal stimulus, such as food and intestinal flora. The symptoms of inflammatory bowel disease (IBD) is recurrent episodes of inflammation of the gastrointestinal tract induced by an inappropriate immune response to the gut microbiota. Diffuse inflammation of the intestinal mucosa is a symptom of ulcerative colitis (UC). Proctitis, the most common form of ulcerative colitis (UC), can also affect the sigmoid (proctosigmoiditis), the entire colon up to the cecum (pancolitis), or somewhere in between.[1] Transmural ulceration of any part of the gastrointestinal tract (GI) is a result of Crohn's disease (CD), but it most frequently affects the colon and terminal ileum. Both conditions are divided depending on their location and degree (mild, moderate, or severe). [1] irritable bowel syndrome (IBS) is a gastrointestinal ailment resulting in abdominal pain, constipation, diarrhea, and altered stool appearance. Gas pooling and symptoms related to impaired intestinal gas transit and evacuation tend to occur in IBS patients.[2]. Recent reports suggest that IBS patients experience abnormal intestinal fermentation and increased gas production.[3] Patients with IBD syndrome have the chance of bloating. Bloating can be defined as a sensation of gassiness or a sense of being distended, but on inspection, the abdomen is not distended in most cases.[1]While the girth may increase in a few individuals, only individuals with an obvious rise in their abdomen should be identified as having abdominal distention. Bloating generally occurs because of gas in the GIT, which can be referred to as a GI disorder. 15 to 35% of people experience bloating, which seems common. Bloating causes pain and discomfort, which can have a negative impact on people suffering from this. Bloating can also be defined as an inflated balloon in the abdomen part of the body. Studies show that half of individuals also have abdominal distention, which can be defined as a visible increase in abdominal size. In general, bloating symptoms may originate from the stomach, small intestine, constipation, eructation (the release of gas from the stomach or esophagus through the mouth), or belching (a way of pushing out excess air from your upper digestive tract). Flatulence is the continual sense of stomach distension and can be related to several illnesses such as irritable bowel syndrome (IBS), functional constipation. When gas accumulates in the intestine, it can cause pressure and distention, resulting in bloating.[5]Flatulence is thought to be caused by indigestion in the stomach. Gas generation in the abdominal cavity is caused by the stomach temperature level, food humidity, or inappropriate humidity in the digestive tract, and their interaction. In Iranian traditional medicine, flatulence is known as 'Nafkh,' 'Nafkhah,' or 'Rih.[5]

(1) The role of environmental factors regarding inflammatory bowel diseases:

(1.1) Smoking

Smoking tobacco cigarettes are associated with having increased incidence and recurrence rates with the case of peptic ulcer disease. It also has associations to IBD, CD as well as the development of carcinoma with esophagus, stomach, liver, pancreas, and colon.[6] Tobacco smoke is associated with an increased risk of penetrating intestinal diseases, strictures or fistulae and a requirement for resections made surgically. It also increases the likelihood of advanced and challenging to treat diseases.[6,7]The most indisputable example of the influence of the environment on IBD is tobacco use, particularly cigarette smoking. Smoking has a strikingly opposite effect on CD and UC, supporting the notion that distinct mechanisms underlie the pathogenesis of each form of IBD. Notably,

cigarette use is an important risk factor for CD, increasing the frequency of disease relapse and need for surgery, and discontinuation improves the disease course. Current smokers have a 2-fold increased risk of CD compared with people who have never used tobacco products. The risk of CD in former smokers lasts several years after smoking discontinuation.

(1.2) Drugs

Medication such as isotretinoin, antibiotics, nonsteroidal anti-inflammatory medications, oral contraceptives, mycophenolate mofetil, etanercept, ipilimumab, and rituximab has been associated with the development of inflammatory bowel disease (IBD).[8] NSAIDs and oral contraceptives are the two main pharmacological classes that have been extensively researched for potential epidemiological or cause-and-effect relationships with IBD.[8] Despite the lack of proof linking oral contraceptives to a specific cause, women who use them have a roughly twofold higher relative risk of CD than those who do not.

(1.3) Stress

In inflammatory bowel disease (IBD), psychological stress has long been associated with increased disease activity. More recently, well-designed studies have demonstrated that depression, chronic stress, and unfavorable life events raise the risk of relapse in individuals with quiescent IBD.[9]Patients with CD and UC often hold the assumption that stress may lead to IBD; however, stress is more likely to modify symptoms than to act as a stimulant. Studies of neuroimmune interactions in lab animals, animal models of colitis, and clinical observations all give evidence that stress can alter the course of IBD.

(1.4) Diet

Recent studies show that nutrition and food have a major part in the etiopathogenesis of the disease, although it is still unknown how exactly they function during the course of the illness.[10] Research demonstrated that among CD patients, 28% of those with active illness and 55% of those in remission were overweight or obese.[11] A higher risk of developing CD is associated with a low diet of fiber and a high consumption of monosaccharides and saturated fats.[12]

(2) History of inflammatory bowel diseases

In the year 1793, the first description of a life-threatening inflammatory bowel disease similar, some people believe, to the modern postulated UC, was made by Matthew Baillie. And subsequently, in 1859, a London doctor by the name of Samuel Wilks used the phrase “ulcerative colitis” to describe a patient suffering from a bowel disease which would have presumably been diagnosed as CD today. It was in 1907 that John Percy Lockhart-Mummery deployed an endoscope with a light bulb. His intention was to see the sigmoid colon, and it was then that he realised seven out of thirty six macroscopic patients with UC developed cancer.[13] It was agreed that diplostreptococci may play a causative role in the ulcerative condition, as demonstrated more recently by Jacob Arnold Bargen, who in 1920 was with the Mayo Clinic. He falsely reported diplostreptococci isolation from cases of rectal ulcer verbs in UC patients, and he showed that injection of this germ form colitis in rabbits.[14] In a relation with this pathology, Meyer and Gellhorn published the findings of their hypothesis in 1947 as well: - the reason of UC was a reduction of the mucous layer of enterocytes because of an increase in lysozyme enzymes, which were then drawing intestinal bacterial biological response. Furthermore, further etiological theories postulated for UC included neurosis, dietary intolerance to some foods, and sensitivity to pollen spores. Further, UC was

presumed to be triggered by the allergy to cow milk. [15,16,17] Roediger et al found that dietary ingestion of sulfur compounds causes ulcerative colitis in 1997. The proinflammatory effects are mediated by the intestinal microbiome, and it has been blamed for the recent increase in incidence of IBD in developing nations because of the worldwide spread of the Western-style diet-low in fruits, vegetables, whole wheat, and nuts; high in fats, sugars, and refined foods. [18] There are thousands of species of microorganisms that make up the human microbiome. It is estimated that each person has between 10 trillion and 100 trillion microbials. There is evidence that the commensal microbiota of the GI tract influences development of both immunological, physiological, and anatomical development of the host. In the last few decades our knowledge of the genetic factors of inflammatory bowel disease (IBD) has exploded. Hundreds

Genomic studies: association can be conducted as sophisticated tools of DNA sequencing and genetic testing led to the finding of new SNPs. [20,21] There must be something more to the pathophysiology of Crohn's disease as there are other genes, such as PTPN2 and IL23R, which are also involved with autoimmune disease. Recent advances in the genetics of the disorder have explained the relevant pathophysiology of IBD. Hundreds of loci contribute to the total risk of conventional IBD, a group of polygenic diseases. There is good evidence from population-based research indicating genetic factors in the pathophysiology of IBD: relatives of UC and CD probands have an 8–10 fold increased risk of developing IBD, and—most importantly—twins are concordant. The strongest evidence for a genetic propensity to IBD, which is higher for CD than for UC, has come from studies in twins. Specifically, twin and family studies for IBD have demonstrated that in the presence of one sibling having CD, the risk for the child to develop CD is 26 times higher, whereas in UC, the risk is 9 times higher. This most recent and largest genetic association analysis reported 163 IBD loci; nearly 300 putative candidate genes were determined across more than 75,000 IBD patients and controls with teenage and adult onset using genome-wide association data. Of these 163 loci, 110 were associated with risk for both subtypes of IBD, whereas 30 were exclusive to CD and 23 were specific to UC. After a trans-ethnic investigation coming out with 38 novel loci for IBD, after examining over 20,000 people of European and non-European descent, the total number of known risk loci for IBD now goes up to 200, while proof of shared genetic risk cuts across cultures. [22,23]

(3) Symptoms of IBD

When compared to the adult population, the elderly patients show a narrower spectrum of illness diversity and severity. In this older group, the frequency of ileocolonic involvement with Crohn's disease (CD) and severe forms of ulcerative colitis (UC) is also less. Older patients with CD, however, are more likely to have colonic than ileal involvement. For older patients, the most common form of UC seen is one with a tight rectal disease, while left-sided disease predominates younger patients. There are neither brand new studies nor any controls looking at the proportions of B2 (stricturing) or B3 (penetrating) subtypes of CD in the older age groups which may present with complicated and uncomplicated disease at the time of diagnosis. The elderly cohorts demonstrating UC are more likely to become hospitalized at different stages of the disease predominantly at flares for the first time. There is evidence that the rates of hospitalization among women with inflammatory bowel disease (IBD) having the onset of CD in the adult years, and those who developed disease in advanced age is approximately the same. What is more, the older IBD patients are also at risk of higher mortality

rates during in-hospital petals and are also reported to have pro loged surgical recovery time which is larger than younger [24]

Diagnosis Approaches Of IBD-:

Clinical Diagnosis: Based on symptoms and family history.

Imaging: ENDOSCOPY (Colonoscopy, Sigmoidoscopy),MRI, CT SCAN , AND CAPSULE ENDOSCOPY.

Laboratory Test: Inflammatory Markers (CRP,ESR) Fecal calprotrctin, and serological tests(e.g PANCA,ASCA).

Histopathology: BIOPSY finding for UC VS CD [25, 26]

Complications: - The Complications related to IBD Are divided into category.

Intestinal

- Hemorrhage
- Strictures
- Colon perforation
- Anal fistulas
- Pelvic or perirectal abscesses
- Toxic megacolon
- Cholangiocarcinoma, colon cancer

Extra Intestinal

- Osteoporosis
- Deep vein thrombosis
- Anemia
- Gallstones
- Primary sclerosing cholangitis
- Aphthous ulcers
- Arthritis
- Iritis
- Pyoderma gangrenosum

Management Of Inflammatory Bowl Diseases

The main therapy for IBD includes immunomodulators, corticosteroid, and aminosalicylates drugs. Such treatment's including oral drugs with molecular weight distribution of low value and temperature that can be internally controlled as well as cost effective came into existence in the early decade of the 1950s. But, there are some people who develop side effects that make them unresponsive to the therapy and hence the need for new therapies arises. Biologics were developed and introduced in the 1990s with the intention of reducing relapses, surgical and hospitalization needs of patients and improving overall quality of life. Biologics are treatments based on monoclonal antibodies that are actually more specific and powerful than standard systemic drugs and have a higher level of stability. They also have three broad functional classes including therapeutic monoclonal antibodies, which target connective tissue inflamed during the sickness, via Integrins, anti-cytokine antibodies, and Tumor necrosis factor-alpha (TNF-α) blocking. Additionally, in the case of IBD, interfered Janus Kinase (JAK) are utilized as a substitute strategy. These small molecules are also involved in IBD pathophysiology and can manipulate cytokine signal transduction in autoimmune diseases. More precisely, available drugs for ulcerative colitis (UC) in these patients who do not respond to biological therapy or standard treatment include tofacitinib, upadacitinib and filgotinib. Small molecules possess some benefits.[27]

Therapy Type	Drug Examples	Usage	Mechanism	Efficacy	Limitation

Aminosalicylates[28]	Mesalamine, sulfasalazine	moderate to mild UC used in the moderate to mild ulcerative colitis .	prevents the synthesis of inflammatory mediators by the intestinal epithelium.	useful for bringing on and keeping UC in remission. Because of the deeper and more extensive bowel involvement, it is less effective in CD.	Minimal effectiveness in CD; insufficient for moderate-to-severe UC.
Corticosteroids[29]	Prednisone, Budesonide	management of mild to severe episodes of IBD during the short term.	reduces inflammation by immune system suppression.	It works well for short-term flare-ups of IBD, but its adverse effects make it unsuitable for long-term maintenance.	systemic adverse effects, such as infection, hypertension, and osteoporosis. Unsuitable for prolonged usage.
Immunomodulators[30]	Azathioprine, Methotrexate	In IBD cases which are steroid-dependent or steroid-refractory, ongoing therapy is used to achieve remission.	lowers the immunological response by preventing immune cells from synthesizing DNA.	Good for maintaining remission over the long term, especially for patients who are on steroids. In CD, methotrexate is often used.	It might take months to start working; there is a chance of infection, liver damage, and myelosuppression.
Anti-TNF-ALPHA Therapies [31]	Infiximab Adalimumab golimumab	Steroid-dependent or steroid-refractory IBD patients require continuous treatment to reach remission.	inhibit a key pro-inflammatory cytokine, TNF-α.	incredibly successful in causing and sustaining remission, encouraging mucosal repair, and lowering the need for surgery in both CD and UC.	The production of antibodies may decrease efficacy and raise the danger of infections (like TB) and cancers (like lymphoma).
Anti-Integrin Therapies[32]	Vedolizumab Natalizumab	IBD which is moderate to severe when anti-TNF is ineffective or inappropriate.	helps decrease inflammation by blocking integrins, which in turn reduces leukocyte migration to the stomach.	More effective than anti-TNF drugs in both UC and CD, but with a reduced systemic risk. It's safer to use Vedolizuma.	A rare but significant risk of progressive multifocal leukoencephalopathy (PML) is linked to natalizumab. Vedolizumab acts more slowly

Anti-IL-12/23[33]	Ustekinumab	CD ranging from moderate to severe, and more recently UC.	targets the cytokines IL-12 and IL-23, which are implicated in inflammation and the immunological response ++in IBD.	helpful for UC and CD patients who have not responded to previous biologics. Inducing remission is quick and efficient, especially for individuals who have not responded to previous treatments.	Although often lower than anti-TNF medications, there is a risk of infections and cancer.
JAK Inhibitors[31]	Tofacitinib	oral treatment for UC that is mild to severe.	inhibits the JAK1 and JAK3 enzymes, preventing inflammatory intracellular signaling.	Remission is brought on quickly and effectively, especially for individuals who have not responded to previous treatments.	possibility of severe adverse consequences, including cancer, infections, and blood clots. limited usage in certain demographics.
Emerging Therapies[34]	Gene therapy, stem cell treatment, and fecal microbiota transplantation (FMT)	Experimental for both CD and UC. FMT attempts to restore the state of balance of the gut microbiota. Gene therapy and stem cell therapy aim to change the immune system.	inhibits both JAK1 and JAK3 enzymes, preventing inflammatory intracellular signaling.	Remission is brought on quickly and effectively, especially for individuals who have not responded to previous treatments.	It is still experimental, and its long-term safety and effectiveness are uncertain.
Surgical Interventions[35,36]	Strictureplasty, Resections, Colectomy (for UC)	Surgery for CD usually tackles problems like strictures or fistulas, but surgery for UC may be curative.	removal of the infected bowel part surgically	Colectomy may be curative for UC. Although CD can reoccur in other bowel segments, surgery is frequently necessary to treat its consequences.	Surgery for CD has a recurrence risk and is not a cure. In UC, surgery is usually reserved for patients who are recalcitrant or have problems

List Of Medicinal Herbal Plants Used in The Treatment of Inflammatory Bowel Diseases.

Medicinal Plants	Biological Source	Family	Parts used	Active Compound	Mechanism of action	Efficacy	Side Effects
Turmeric [37,38,39,40]	*Curcuma Longa*	Zingiberaceae	Rhizome	Curcumin or curcuminoid	reduces inflammation by blocking pro-inflammatory cytokines (TNF-α, IL-1, and IL-6) as well as NF-κB and COX-2.	shown to decrease inflammation and symptoms in individuals with mild to moderate ulcerative colitis.	liver injury,Allergic reaction,kidney damage,lower bloodpressure,lowered blood sugar
Aloe vera [41,42,43,44]	*Aloe barabadensis,Aloe Spicata,Aloe perryi,Aloe ferox*	liliaceae	Leaf Gel	Aloin,	has anti-inflammatory and antioxidant properties; it modulates the immunological response to minimize intestinal inflammation.	could decrease inflammation and clinical symptoms in UC patients.	Belly pain, kidney damage, electrolyte disbalance
Frankincense tree [45,46,]	***Boswellia serrata***	Burseraceae	resin	Boswellic acids	Inhibits 5lipoxygenase reducing,leukotriene formation and inflammation in the gut.	Good for UC and CD patients in terms of lowering inflammation and keeping remission.	Nausea , acid reflux , diarrohea
Cannabis [47,48,49, 50]	*Cannabis sativa*	*Cannabaceae*	Leaves, Flowers	Cannabinoids (THC, CBD)	reduces inflammation and abdominal discomfort in CD by modifying the endocannabinoid system.	helps CD patients with their symptoms, such as lowering diarrhea and stomach discomfort.	headache, sleepiness, nausea, and dizziness.

Plantago ovata [51]	*Psyllium husk*	Asteraceae	seed	Fiber, Mucilage	reduces the frequency of bowel movements and discomfort in individuals with IBD by forming a gel-like material in the intestines.	helps UC patients have more frequent and consistent bowel movements; it may also help keep remission going.	Some patients experience gas and bloating.
Green Chiretta[52]	***Andrographis paniculata***	Acanthaceae	Aerial part	Andrographolide, Flavonoids	reduces intestinal permeability and inhibits TNF-α, IL-6, and IL-1β, making it anti-inflammatory and immunomodulatory.	In mild to severe instances, it has been demonstrated to decrease inflammation and UC symptoms on par with mesalamine.	Tiredness and the occasional headache are often readily tolerated.
Licorice[53].	***Glycyrrhiza glabra***	fabaceae	root	Glycyrrhizin, Flavonoids	anti-inflammatory, regulates gut immunological activity, and prevents the synthesis of pro-inflammatory cytokines.	contains antioxidant properties and may help lessen intestinal irritation and inflammation in UC and CD.	Long-term use may causes the hypokalemia and hypertension.
Ginger[54] .	***Zingiber officinale***	Zingiberaceae	rhizome	Gingerols, Shogaols	reduces inflammation by blocking NF-κB and TNF-α, which also affects	has the potential to lessen intestinal inflammation and	Causes moderate gas, heartburn, or other gastrointestinal discomfort.

					the gut's immunological response.	the intensity of IBD symptoms in individuals.	

Recent studies on IBD

Cells that renew the colon's lining could be affected by excess sugar present in the diet in a mouse model of inflammatory bowel disease (IBD), according to a new study by University of Pittsburgh scientists. The prevalence of IBD is rising around the world, and it is rising the fastest in cultures with industrialized, urban lifestyles, which typically have diets high in sugar . Too much sugar is not good for a variety of reasons, and the new study published in *Cellular and Molecular Gastroenterology and Hepatology*adds to that evidence by showing how sugar may be harmful for diet[55] In 1990, the global age-standardized incidence rate (ASIR) of IBD was 4.22 per 100,000, but by 2021, it had risen to 4.45 per 100,000. From 1990 to 2021, the age-standardized mortality rate (ASMR) dropped from 0.60 per 100,000 to 0.52 per 100,000. The age-standardized DALYs rate also dropped, from 21.55 per 100,000 in 1990 to 18.07 per 100,000 in 2021. Comparisons by gender revealed very little variation in the burden of illness. The World Bank upper-middle income area (EAPCs, 1.25) and the World Bank high-income region (EAPCs, 1.00) saw the most increases in IBD-associated ASIR and ASMR, respectively. East Asia saw the most growth in ASIR regionally (EAPCs, 2.89). In 2021, the Netherlands had the highest ASMR (2.21 per 100,000), while China had the largest growth in ASIR (EAPCs, 2.93).[56]

CONCLUSION: -Using both alternative and orthodox medical systems may give a fuller picture of addressing IBD. Moreover, herbal medicines may be of extra help in alleviating symptoms and inflammation but orthodox drugs are very important to induce and maintain remission especially in the case of moderate to severe diseases. In order to enhance the outcomes and the functioning and well-being of patients suffering with IBD, customized treatment protocols, constant monitoring and ongoing evolution of both conventional and alternative medicine are needed.

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