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Review Article

Inflamed journeys: Understanding ulcerative colitis

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ABSTRACT

Ulcerative Colitis is a kind of IBD (Inflammatory Bowel Disease). Recurrent episodes of gastrointestinal tract inflammation brought on by an aberrant immune response to gut microbiota is what defines IBD. India has the highest reported incidence of IBD (931 cases per 100,000 people) and UC (5.41 cases per 100,000 people) among the world's developing nations. It's interesting to note that whereas UC is more common in Northern India, CD is more common in Southern India. In a few years, India may have the highest overall burden of IBD due to its large population. Globally, the burden of disease will keep increasing because mortality is often low.The present study reveals historical background, epidemiology, and pathophysiology of Ulcerative Colitis, while also drawing comparisons between modern treatment modalities and traditional systems like Ayurveda. Ongoing research is focused on refining treatment strategies, with a tailored approach to enhance patient outcomes, minimize recurrence, and improve the quality of life for individuals dealing with Ulcerative Colitis.

1. Introduction

An autoimmune reaction that affects the colon is known as ulcerative colitis. Along with Crohn's disease, it is regarded as one of the main forms of inflammatory bowel disease. The immune system of the body targets healthy cells in the colon in ulcerative colitis, which causes inflammation and ulcers in the large intestine.

1.1. History of ulcerative colitis

Mathew Baillie's 1793 publication, "Morbid Anatomy of Some of the Most Important Parts of Human Body," provided proof that Ulcerative Colitis was becoming more common in the latter 18^{th} century. Instead of examining the origins, traits, course, and "outcomes" of the illness, this presentation focused on post-mortem pathological anatomical changes, organ by organ. In 1859, Sir Samuel Wilks was the first to explain the illness, stating that it was "simple Ulcerative Colitis." It was not distinguished from dysentery until recently. Up until around 20 years ago, Samuel Wilks determined this distinction while the patient was still alive; necropsies are increasingly frequently used to make this determination. In 1859, he coined the term Ulcerative Colitis to describe the inflammation of the colon and distal portion of the ileum. A 42-year-old lady who passed away following several months of fever and diarrhoea had a transmural ulcerative inflammation of the colon and terminal ileum, according to Wilks autopsy results. Later on, this was determined to be Chrons disease. Sir Arthur Hurst later corroborated Wilks' work. A case report by Wilks and Moxon from 1875 describes how young people who died of acute diarrhoea and an early incidence of ulcerative colitis developed ulcerations and inflammation over their whole colon. After then, Wilks and Moxon

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(1875), Allchin (1885), and Hale-White (1888) took careful note of the disease's histological and clinical characteristics. When Prince Charles had ulcerative colitis in 1745, he was able to control his symptoms by cutting out dairy and milk from his diet. After germ theory gained popularity, Londoner Sir William Hale White (1857–1949) published a comprehensive list of examples he had personally observed in 1888. These cases demonstrated cases of "ulcerative colitis" that could not be linked to any known etiology, including "tumours, dysentery, tuberculosis, typhoid, and similar factors". The phrase "ulcerative colitis" entered the wider medical lexicon as a result of this report. An important turning point in the knowledge of UC occurred in 1909. The London-based Royal Society of Medicine held a symposium in January of that year.

More than 300 ulcerative colitis (UC) cases from various London hospitals were presented at this meeting and thoroughly examined and discussed. Then, in March 1909, Herbert P. Hawkins' article, "Address on the natural history of ulcerative colitis and its bearing on treatment," appeared in the British Medical Journal. This talk, given in front of the Bristol Medico-Chirurgical Society, focused on how important it is to understand how the illness develops naturally. He used case studies to demonstrate the illness and advanced the idea that successful disease management required determining the "active bacterial agents" causing it. [1,2]

1.2. Ulcerative colitis

Ulcerative Colitis is a kind of IBD (Inflammatory Bowel Disease). Recurrent episodes of gastrointestinal tract inflammation brought on by an aberrant immune response to gut microbiota is what defines IBD. The two forms of idiopathic intestinal disease that make up IBD differ in where and how deeply they affect the colon wall.

1. Crohn's disease
2. Ulcerative Colitis

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. Both conditions are categorized based on severity (mild, moderate, or severe) and location. CD is also categorized based on presentation-inflammatory, structuring, or penetrating. Aside from the GI tract, both Crohn disease and ulcerative colitis have numerous manifestations outside the intestines. While most patients can distinguish between the two disorders, around 10% of patients have such similar symptoms that initial differentiation is challenging. [3]

The lining of the colon and the rectum become inflamed when someone has ulcerative colitis. Ulcerative colitis is an autoimmune disease in which the large intestine's inner lining becomes inflamed and ulcerated due to aberrant immune system responses. Although ulcerative colitis can strike anyone at any age, those between the ages of 15 and 30 are more prone to get it. The rectum is always affected by inflammation (proctitis), and in certain situations, it spreads continuously proximally to affect the entire colon (pancolitis). Long-term pancolitis can cause the colon to shrink, and after the inflammation is over, "pseudopolyps"—normal or hypertrophied remnant mucosa—develop in the atrophy areas. The deeper layers of the intestinal wall are unaffected by the inflammatory process, which is restricted to the mucosa. The crypts and lamina propria are invaded by both acute and chronic inflammatory cells. Abscesses in crypts are common. Mucus is lost by goblet cells, and in chronic situations, glands deform. Colon cancer may arise as a result of dysplasia, which is characterized by nuclear atypia, an accelerated rate of mitosis, and a piling of cells within crypts. Crohn's disease and Ulcerative Colitis was mentioned in (Figure 1) [2]

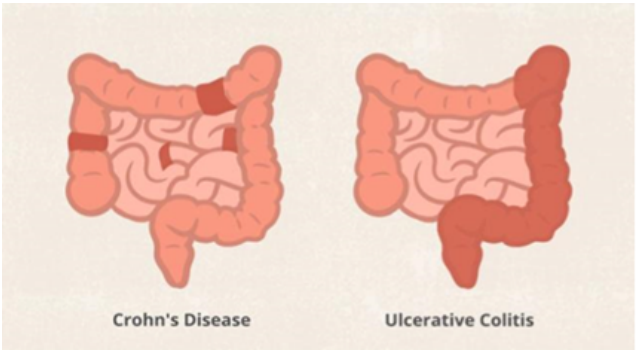

Figure 1: Crohn's disease and ulcerative colitis
Source: https://www.everydayhealth.com/crohnsdisease/symptoms/key-difference-between-crohns-disease-ulcerative-colitis/

1.3. Types of ulcerative colitis

Ulcerative colitis is frequently categorized by location by medical professionals. Each type's symptoms frequently overlap. Ulcerative colitis types include.

1.3.1. Ulcerative proctitis

Approximately one-third of patients with UC have this kind of UC. The rectum, the final segment of the large intestine, is the only area where intestinal inflammation occurs in ulcerative proctitis. Less than 6 inches of rectum are often affected by this condition. There is no connection between ulcerative proctitis and an increased risk of cancer as mentioned in (Figure 2).

1.3.2. Symptoms

It includes pain in your rectum, bleeding in your rectum, and a sudden need to go to poop.

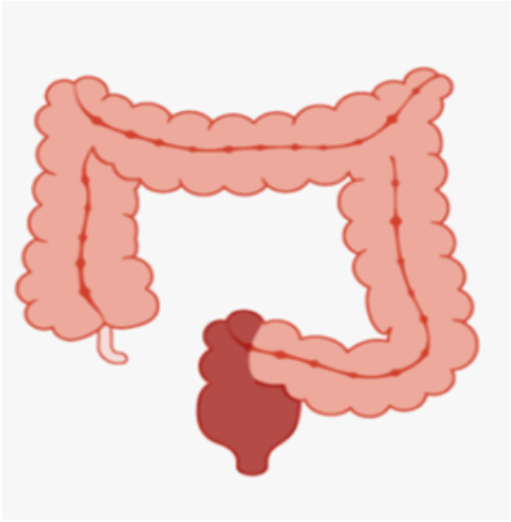

Figure 2: Ulcerative proctitis
Source: https://images.app.goo.gl/K6GsKRG3uBgew7mb7]

1.3.3. Left-sided colitis

From your rectum to the splenic flexure of your colon, which is a bend close to your spleen, this type of UC can induce inflammation as shown in (Figure 2). Proctosigmoiditis, which affects the rectum and sigmoid colon—the lower portion of the colon above the rectum—is one of these conditions.

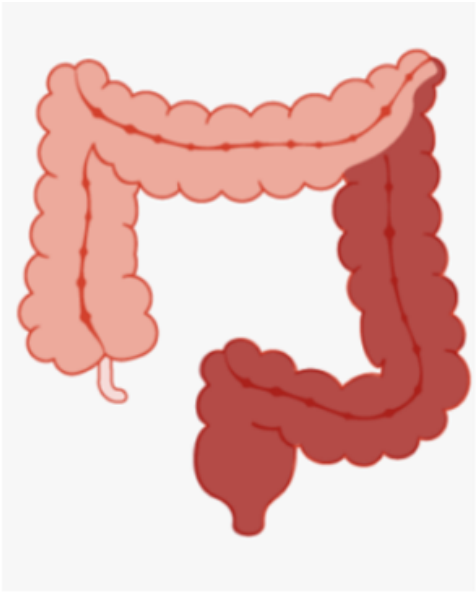

Figure 3: Left sided colitis
Source: https://images.app.goo.gl/K6GsKRG3uBgew7mb7

1.4. Symptoms

Loss of appetite, weight loss, pain in belly or bloody diarrhea.

1.5. Extensive colitis (Pancolitis)

This type of UC affects entire colon. Inflammation starts at rectum and goes beyond splenic flexure shown in (Figure 4).

Figure 4: Extensive colitis (Pancolitis)
Source: https://images.app.goo.gl/K6GsKRG3uBgew7mb7]

1.5.1. Symptoms

It might include bloody diarrhea, pain in belly, lack of appetite, weight loss, fever and cramp.

Ulcerative Colitis symptoms often get worse over time. In beginning you may notice signs of mild UC including:

1. Diarrhoea (may or may not be bloody)
2. Increased bowel movements or episodes of diarrhoea (four or fewer episodes daily)
3. Urgent bowel movements (sudden need to poop)
4. Tenesmus(feeling like you have to poop but being unable to)
5. Mild abdominal (belly) cramping or tenderness

Later you may also have symptoms of moderate to severe UC, including:

1. Frequent bowel movements or episodes of diarrhoea (four or more episodes daily)

2. Blood, mucus or pus in your stool
3. Severe belly cramping
4. Fatigue (extreme tiredness)
5. Sudden weight loss
6. Nausea[4]

1.6. Causes of ulcerative colitis

Numerous elements that are yet poorly understood contribute to ulcerative colitis. Ulcerative colitis is caused by an aberrant immune response, genetics, microbiology, and environmental factors. According to research, the body's immune system and a virus or bacterium in the colon may combine to cause ulcerative colitis. Temporary inflammation is a natural immunological reaction to fight an infection or disease. After you are well and no longer ill, the inflammation will then go away. In people with ulcerative colitis, inflammation continues long after the immune system should have completed its task. White blood cells are still being sent by the body into the intestinal lining, where they cause ulcers and persistent inflammation.[5]

1.7. Epidemiology

Globally, the prevalence of IBD is significantly increasing. Despite being high in western nations, the incidence of UC and CD may be plateauing. For instance, the frequency of both UC and CD in Canada was estimated to be around 15 cases per 100,000 people in the 2000s; however, recent population-based data on incidence from three Canadian provinces has revealed a decline in the prevalence of UC and CD in adults. Although developing nations have a far lower incidence of UC than developed jurisdictions, new evidence shows that the incidence is rising in developing jurisdictions. India has the highest reported incidence of IBD (931 cases per 100,000 people) and UC (5.41 cases per 100,000 people) among the world's developing nations. It's interesting to note that whereas UC is more common in Northern India, CD is more common in Southern India. In a few years, India may have the highest overall burden of IBD due to its large population. Globally, the burden of disease will keep increasing because mortality is often low. All ages have been reported to experience UC. The highest incidence of pediatric IBD (occurring in children under 16) is seen in Scandinavia and Canada; in Norway, there are 10.6 cases per 100,000 people, in Sweden, there are 12.8 cases per 100,000 people, and in Canada, there are 9.68 cases per 100,000 people.

Nonetheless, the study found that the prevalence of very early onset IBD, which affects children aged 0 to 5, rose by 7.2% annually. According to reports, the prevalence of UC is the same for both sexes from childhood to maturity. Ethnicity may not be a significant factor in the epidemiology of IBD, although further research is necessary. Children of individuals who moved from low-incidence to high-incidence locations, for instance, have been found to have the same incidence as those who moved to the new location. Ethnicity may not be a significant factor in UC, as seen by the rising frequency in emerging nations and the similar phenotype of IBD in Asians and Westerners. Since the UC phenotype is very uniform across the globe, the observed epidemiological trend is probably due to environmental factors.[6]

1.8. Pathophysiology

There are both genetic and environmental factors that contribute to IBD, and evidence from genome-wide association studies indicates that genetic variations that increase the risk of developing Crohn's disease may have experienced positive selection by warding off infectious diseases like tuberculosis. These genetically predisposed people are believed to develop IBD as a result of their aberrant inflammatory response to environmental stimuli, including gut microorganisms. Tumour necrosis factor alpha (TNF-a), interleukin (IL)-12, and IL-23 are among the inflammatory mediators released as a result, causing tissue damage and involving a wide range of innate and adaptive immune cell responses. A correlation between microbial dysbiosis and IBD seems to exist. For instance, there is a relative increase in Enterobaceteriaceae and a decrease in diversity, mostly in Firmicutes and Bactericides. One of the significant functional alterations in the bacteria is the decrease in anti-inflammatory metabolites such butyrate and other short-chain fatty acids. Although microbial dysbiosis has been identified as a contributing factor to IBD, a causal relationship has not yet been proven. There is growing evidence that the development of IBD may be influenced by the mycobiome (fungal species) and virome. Although acute and chronic inflammatory cells infiltrate the intestinal wall in both situations, the distribution of lesions and histological characteristics differ significantly. Pathogenesis of inflammatory bowel disease was mentioned in (Figure 5).

1. Bacterial antigens are taken up by specialised M cells, pass between leaky epithelial cells or enter the lamina propria through ulcerated mucosa.
2. After processing, they are presented to type 1 T-helper cells by antigen-presenting cells (APCs) in the lamina propria.
3. T-cell activation and differentiation results in a Th, T cell-mediated cytokine response
4. With secretion of cytokines, including interferon gamma (IFN-y). Further amplification of T cells perpetuates the inflammatory process with activation of nonimmune cells and release of other important cytokines, including interleukin (IL)-12, IL-23, IL-1, IL-6 and tumour necrosis factor alpha (TNF-a). These pathways occur In all normal individuals exposed to an inflammatory insult and this is self-

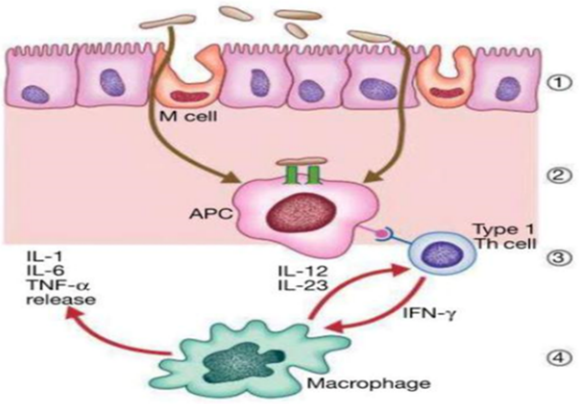

Figure 5: Pathogenesis of inflammatory bowel disease
Source: Ralston S, Penman I, Strachan M, HobsonR, Editors. Davidson's Principles of Medicine. 23^{rd} ed.International edition; 2018, P-815

limiting in healthy subjects. In genetically predisposed persons, dysregulation of innate immunity may trigger inflammatory bowel disease.

Inflammation in ulcerative colitis usually begins in the rectum (proctitis) and then, in certain cases, extends throughout the entire colon (pancolitis). Long-term pancolitis can cause the intestine to shrink and post-inflammatory "pseudopolyps" to form; these are patches of atrophy where the remaining mucosa is normal or hypertrophied. The deeper layers of the intestinal wall are unaffected by the inflammation, which is limited to the mucosa. Both acute and chronic inflammatory cells infiltrate the lamina propria and the crypts, resulting in 'cryptitis'. Abscesses from crypts are frequent. Mucus is lost by goblet cells, and in chronic situations, glands may deform. Colon cancer may develop after dysplasia, which is characterized by an increase in cell division rate, aberrant cell nuclei, and cell accumulation within crypts.[7]

In Ayurveda Raktatisara, Raktaja Pravahika,Grahani shows symptoms having resemblance with Ulcerative Colitis.[8] According to Ayurveda, Ulcerative Colitis can be understood as Pittaja grahani. Grahani roga (Chronic mahagadas) (Major disorders). Grahani is the seat of Jatharagni (digestive fire) and is supported and nourished by strength of Agni. "Rogaa:Sarve api mande agnou"- Grahani roga is caused by mandagni (reduced digestive fire or digestive capacity). Grahani, Arsha (haemorrhoids) and Atisara (diarrhea) manifest mainly due to improper Agni (digestive fire). Nidana sevana (etiological factors) causes drava guna vruddhi (increased liquidity quality) of pitta, leading to Agnimandhya (reduced appetite). This leads to Grahani dushti, which gradually causes Grahani roga. Grahani roga is stage where the normal functioning of Grahani is hampered, which is clinically expressed as the elimination of ama mala (poorly formed stools). In Pittaja Grahani, this is associated with symptoms like shula (pain), shoth (inflammation), Atisara(diarrhea), vrana (wound)and raktasrava(bloody discharge).[9]

Also according to Ayurveda, it can be correlated to the disease Pravahika which manifests in the form of Atipravahana of Purisha (repeated defecation with tenesmus), Atidrava Purisha Pravritti(watery stool),Udarashoola(abdominal pain) ,Picchila, Saphena (Sticky and Frothy) and Raktayukta Purisha (blood-mixed stool).[10] In Ayurveda, the ancient system of Indian medicine is mainly based on the concept of three major constitutional types (Vata, Pitta, Kapha)which is known as Prakriti. In Charak Samhita erroneous dietary pattern,faulty lifestyle and psychological factors along with suppression of Agni are mentioned as root cause of Pittatisara which in chronic stage manifest as Raktatisara.[11,12] In Charak Samhita Pittatisara patients passes stool of different colours like green, blue, yellow, black associated with Rakta and Pitta (Stool with blood and mucus) and smelling extremely unpleasant. If Pittaja Atisara patient not following Kriya Muktva(treatment and precautions) and continues to take Pitta aggravation Ahara which further aggregates Pitta this resulted into aggravating Rakta Dhatu (~blood)which manifested as Raktatisara (~bleeding diarrhea). The factors affecting on UC was shown in (Figure 6).

A major symptom of Ulcerative Colitis is bloody diarrhoea shown in (Figure 7). Emotional stress is also play a provoking role in relapse. In ancient system of Indian Medicine Raktatisara is also having symptoms blood in stool. Psychological stress with Krodh (anger) and Irsha (~jealousy) is the main cause of Pittatisara which later on turned in Raktatisara (~bleeding diarrhea).[13]

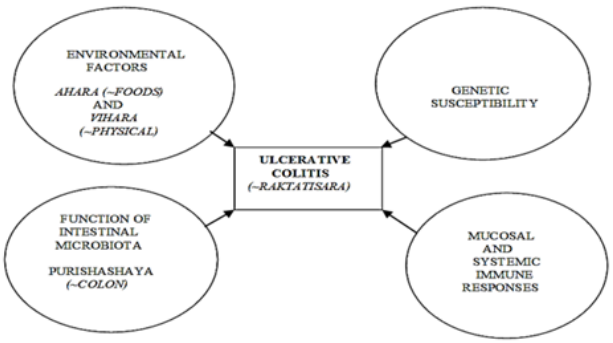

Figure 6: The most suitable theory for the aetiology of Ulcerative colitis involves a complex interaction between gastrointestinal microbiota, genetic susceptibility, environmental factors, and mucosal or moregeneralized abnormal immune responses.
Source: Dabas R, Dixit V, Kar A. Critical Review of Raktatisara Vis-a-visUlcerative Colitis

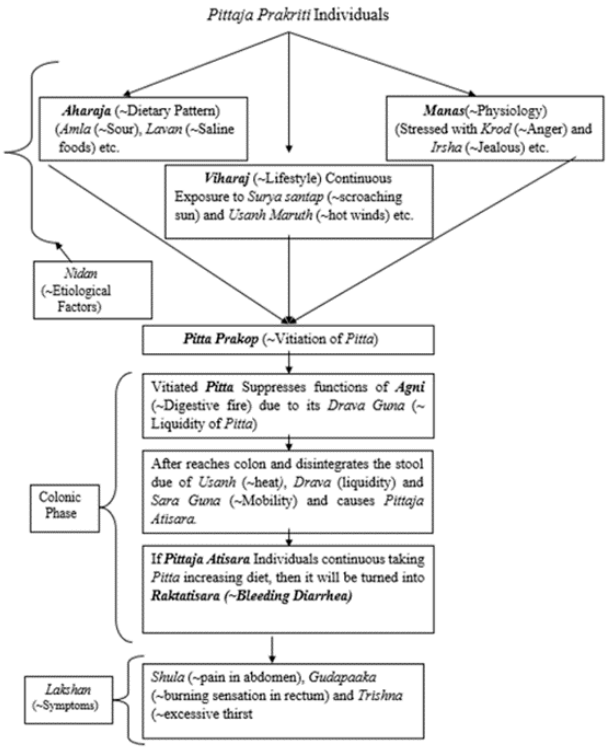

Figure 7: Etiopathogenesis of raktatisara (~hemorrhagicDiarrhoea)
Source: Dabas R, Dixit V, Kar A. Critical Review of Raktatisara Vis-a-vis Ulcerative Colitis]

1.9. Investigations/diagnosis

Confirming the diagnosis, defining the disease's spread and activity, and identifying its complications all require investigations. Anaemia brought on by bleeding or a lack of iron, folic acid, or vitamin B12 absorption may be revealed by a complete blood count. Chronic inflammation can also be indicated by a high platelet count. Protein loss enteropathy, inflammatory diseases, or inadequate nutrition can all result in a decrease in serum albumin content. ESR and CRP are raised during flare-ups and in reaction to the development of abscesses. Even in cases where the CRP is normal, fecal calprotectin may be increased due to its great sensitivity to gastrointestinal inflammation. When diagnosing inflammatory bowel disease and later tracking its progression, it is especially helpful in differentiating it from irritable bowel syndrome.

Other diagnostic methods consist of:

2. Endoscopic Procedures

2.1. Colonoscopy

During this examination, your doctor will use a thin, flexible, lighted tube with a camera on the end to observe your whole colon. Tissue samples are collected during the operation for analysis in a lab as shown in (Figure 8). We call this a tissue biopsy. The diagnosis cannot be made without a tissue sample.

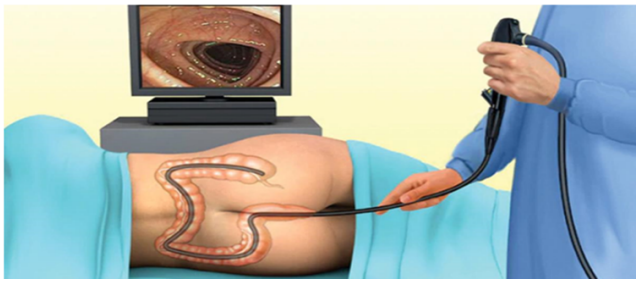

Figure 8: Colonoscopy
Source: https://gastroenterology.smileshospitals.com/colonoscopy

2.2. Flexible sigmoidoscopy

The rectum and sigmoid colon, the lowest portion of your colon, are examined by your healthcare professional using a thin, flexible, illuminated tube. It might be better to do this test rather than a full colonoscopy if your colon is extremely irritated.

2.3. Imaging procedures

2.3.1. X-ray

If you have severe symptoms, your provider may use a standard X-ray of your abdominal area to rule out serious complications, such as a megacolon or a perforated colon.

2.3.2. CT scan

A CT scan of your abdomen or pelvis may be performed if a complication from ulcerative colitis is suspected. A CT scan may also reveal how much of the colon is inflamed.

2.4. Computerized tomography (CT) enterography and magnetic resonance (MR) enterography

To rule out any mild intestinal irritation, several noninvasive tests might be suggested. Compared to traditional imaging tests, these assays are more sensitive for detecting intestinal inflammation. An alternative that doesn't involve radiation is MR enterography.[5]

3. Treatment in Allopathy

3.1. Active proctitis

A 1 g mesalazine suppository helps the majority of patients with ulcerative proctitis, although some also need oral 5-aminosalicylate (5-ASA) therapy.Topical glucocorticoids are only used for people who cannot tolerate topical mesalazine since they are less effective. Immunosuppressive

drugs and systemic glucocorticoids may be necessary for patients with resistant illness. Proximal constipation may need to be treated with a stool softener.

3.2. Active left-sided or extensive Ulcerative Colitis

The once-daily oral and topical 5-ASA formulation (also known as the "top and tail approach") is typically successful in mild to moderately active patients. After a month, the tropical preparation (1g foam or liquid enema) is usually discontinued. Long-term use of oral 5-ASA reduces the likelihood of dysplasia and prevents relapse. Patients should take oral prednisolone (40 mg daily, reduced by 5 mg/week over an 8-week course) if they do not respond to this treatment within 2-4 weeks. It is never appropriate to take glucocorticoids for maintenance treatment.

Immunosuppressive treatment with a thiopurine should be started as soon as glucocorticoid resistance (lack of effectiveness) is evident or in patients who need repeated doses of glucocorticoids to stay under control. For bone protection, glucocorticoids should be used along with calcium and vitamin D supplements.

4. Severe Ulcerative Colitis

The best care is provided in a hospital, where a doctor and surgeon should work together to monitor patients who do not respond to maximal oral medication or who present with acute severe colitis. Clinically, for the frequency, temperature, pulse rate, stool blood, and presence of abdominal pain. Through laboratory testing: stool culture, haemoglobin, white blood cell count, albumin, electrolyte, ESR, and CRP.

Radiologically: on simple abdomen X-rays for colonic dilatation. In order to treat dehydration, intravenous fluids should be administered to all patients, and patients who are malnourished should get enteral fed assistance. It is recommended to administer intravenous glucocorticoids either bolus injection or intravenous infusion. In order to prevent the necessity for an immediate colectomy, patients who do not respond to glucocorticoids should be evaluated for rescue therapy with ciclosporin (intravenous infusion) or infliximab (5 mg/kg).

5. Treatment in Homeopathy

Chronic ulcerative colitis can be effectively treated with homeopathy. The treatment of ulcerative colitis using homeopathy is beneficial. It focuses on treating the root problem, which is to try to improve the body's immune system's operation. It is possible to treat digestive tract ulcers by fixing this. Patients who take homeopathic medications do not experience any negative side effects. It aids in lessening ulcerative colitis's severity and recurrence. If the patient begins treatment right away, surgery can be avoided. It enhances the patient's general health and lessens the need for immunosuppressive medication.

In addition, homeopathy places a strong emphasis on constitutional remedies. Following a thorough assessment of the patient's personality, symptoms, likes and dislikes, sleeping patterns, etc., these are recommended. By lowering stress, this enhances the immune system's ability to function normally. The following treatments will lessen intestinal bleeding, ulceration, and inflammation.

1. Arsenic album
2. Merc Cor
3. Merc Sol
4. Argentum nitricum
5. Phosphorus
6. Carcinosin
7. Kali bichromatum

5.1. Management of ulcerative colitis

Oral, tropical and IV administration of Antibiotics, Amino salicylates, Corticosteroids, anti-Tumor necrosis factors have been used to subside the active phase and to maintain the remission of disease. But, drug resistance, drug dependency and side effects of those drugs are high. Up to 60% of patients with extensive ulcerative colitis eventually required surgery (colectomy).

6. Treatment in Ayurveda

According to ayurveda principles Agnimandhya is the root cause of this disease. In all kinds of Atisara, sign of Ama and pakva should be determined first. This is the first line of Atisara treatment. The basic principle to consider during treatment mentioned as Ama (indigested) or Pakva (digested) features of the patient. Agni Dipana (enhance digestive fire), Ama Pachana (digestion of indigested particles), Grahi (checks diarrhea), Stambhana (checks bleeding), Dhatu Poshaka (nutrition supplement in tissue level), Sattvavajaya Chikitsa (psychotherapy) should be given according to the condition of the patient. Keeping in mind the strength of the patient, In case of Amavasta Langhana (Fasting) should be done first, then drinking of Yevagu (Thick gruel) made with Deepan, Pchana drugs like sunthi, chitrak etc is beneficial. Grahi drugs should be avoided in Amavasta condition as it may cause Pliha Vridhi (splenomegaly), Pandu (anemia), Anaha, Prameha, Kustha (skin disease), Jwara (fever), Śopha (edema), Gulma (abdominal lump), Grahani (IBS), Arsha (piles), Shula (pain), Alasaka, Hrid Graha (Cardiac discomfort) etc.In Pakva condition Grahi medicine can be given. According to the patient condition along with Shaman Chikitsa (Deepan, Pachana, Grahi medicine) Sodhana Chikitsa (Purification therapy) is beneficial. In Raktatisar basically Basti Karma is indicated, among different type of Basti described in Ayurveda classics Pichha Basti is considered best for the Raktatisara.

6.1. Shaman chikitsa

Some Ayurvedic preparation mentioned in classical texts of Ayurveda which are useful in the treatment of Pittatisar and Raktatisar patients:

6.1.1. Churna (Powder)

1. Powder of Yestimadhu, Shankha Bhasma, Black mud and Nagkeshar with honey or Tandulodak (rice water) is an excellent haemostatic so it is useful in the treatment of Raktatisar.
2. 10gm paste of Black Sesame mixed with 2gm of sugar and taken with goat's milk. Or 3gm Paste of Priyangu taken with honey followed by Tandulodak (Rice Water) checks haemorrhage quickly. Or White Chandan mixed with sugar and honey followed by Rice water one relieved the burning sensation, thirst and haemorrhage.
3. Intake of Paste of Black sesame 5 parts and Paste of Sharkara (Sugar) 1 part with Goat milk stop rectal bleeding immediately.
4. Rasanjana, Ativisha bark, Indrayava, Haritaki, Sunthi, with honey followed by Tandulodak (Rice water) is useful in Raktastisar.
5. Daruharidra bark, Pipali, Shunthi, Lakha, Indrayeva, Kutki, siddha cow ghee mixed with peya is beneficial in Raktatisar.
6. Bark powder of Priyal, Shalmali, Plakshya, Shallaki and Candan with milk or Yesthimadhu, Sharkara, Lodhra, Vidari and Sariva mixed with goat milk is useful in the treatment of Pittatisar and Raktatisar.
7. Powder or Paste of Manjistha, Sariva, Lodhra, Padmakhya, Kumud, Nilotpal and Bhagri with Goat milk is useful in the treatment of Pittatisar and Raktatisar.
8. Sharkara, Kamal, Lodhra, Manjistha, Madhuyesthi and Till. Or Black till, Madhuyesthi, Mandjistha, and Nil kamal. Or Till, Mochrasa, Lodhra, Yesthimadhu, and Nilotpal. Or Kachhura and Till. Paste (Kalka) of these four preparation with Goat milk and honey are useful in the treatment of Raktatisara.
9. Madhukadi powder
10. Nagkeshar powder
11. Nilotpaladi yoga

6.1.2. Ghrita preperation

1. Shatavari Ghrita
2. Dravyadi Ghrit
3. Nyogradhi Ghrita

6.1.3. Kwatha (Decoction)

1. (a) Cold decoction (Shit Kasaya) of Shalmali vrinta with Yastimadu and honey cured Pittatisar and Raktatisar.
   (b) Administration of Bilvo Majja with Fadita followed by honey is useful in Raktatisara patient.
   (c) Kutajadi kasaya: Bark of Kutaj, Bark of Dadim Fruit, Root of Nagarmotha, Flower of Dhataki, Bilvo Fruit majja, Sughandhabala, Red Chandan and Patha decoction with honey is useful in all types of diarrhoea especially in bloody diarrhoea.
   (d) Dadimadi kwath: Decoction of Dadim Fruit Bark and Kutaj Bark is useful in chronic bloody diarrhoea.

6.1.4. Sodhana chikitsa

In Sodhana (Purification) therapy mainly Basti (enema) is indicated in Raktatisar patient. Among different type of Basti, Piccha Basti (slimy enema) and Anuvasana Basti (oil enema, mainly medicated Ghee) are useful in mild to moderate stage of Raktatisar to check bleeding, inflammation in anorectam, diarrhoea and abdominal pain.

1. Anuvasana Basti
2. Piccha basti

6.2. Method of preparation of Piccha Basti

The fresh flower or leaves stalks of Shalmali should be wrapped around with fresh Kusa grass and plastered with black mud and heated on cow dung fire. When the outside mud plaster is dried well, it should be brought down and the stalks of Shalmali are taken out. Then stalk of Shalmali are pounded in a mortar and make a bolus of 1 Pala (48 gm). Then bolus is pressed in 1 Prastha (540ml) of boiled milk and filtered. After that, filtered cow milk is mixed with sesame oil, Ghee and Yestimadhu paste in adequate quantity. Then this prepared enema should be administered through anal route to the patient in left lateral position. When the enema comes out, advice the patient to take food with milk or meat soup of wild animal.

6.2.1. Modified piccha basti

Now a days modified Piccha Basti has been prescribed by different Ayurveda physician for Raktatisar patients. Ingredients of Modified Piccha Basti according to different research article published in national and international journal:

6.3. Kwatha (Decoction)

Shalmali Vritta Kwatha-100-150ml Kalka (paste): Yesthimadhu Powder- 3gm, Lodhra Powder-3gm, Rasanjana-3gm, Mochrasa-3gm, Nagkeshar Powder-3gm, Shatapushpa Powder-3gm = Make a paste by adding 1 glass of water. Sneha: Panchatikta Ghrita or Changeri Ghrita or Jatyadi Ghrita- 20ml Milk: Goat milk- 100-200ml Honey- 1-2 tsf Pathya (to be taken) The following specific foods are generally recommended for patients of ulcerative colitis. However, not all patients will tolerate all of these

food items. Physician can provide a more individualized nutritional plan.

6.4. Cow ghee or medicated ghee

Such as Satavari ghrita can be used for these patients due to its Vatanulomana and Agni dipana properties.

6.4.1. Oat's milk

After boiling with three parts of water has been recommended in Susruta samhita for chronic disease to eliminate residue.

6.4.2. Takra (Butter milk)

It is helpful to maintain microflora in gut in Ulcerative colitis.

6.5. Others

Cow milk, Raw banana, Jamun, Vilwo (Fruit of Aegle marmelos), Anar (Pomegranate), Dadhi (Curd), Shali rice, Sathi rice (Old basmati rice >6 months), Lajja prepared from rice, barley, Mung dal, Masur dal, Arahar dal, Kidney beans, coriander, cumin, Sunthi etc are considered as a suitable diet for ulcerative colitis patient.

Patients should be assured with proper counselling in all the stages of the disease as ulcerative colitis involves Manasika Bhava (psychological factors) in its disease process.

6.6. Apathya (to be avoided)

You should stay away from any leafy greens, black gram beans, spicy foods, white sugar, wheat, pasta, vinegar, and foods that are acidic or salty. Steer clear of raw salads, tea, cold beverages, ice cream, Supari (Areca-nut) mango, alcohol, and smoking. Manashika bhava (psychological variables) like Shoka, Chinta (stress), Bhaya (fear), and worry are often discouraged. For one month, UC patients received Udumbara Kwatha Basti along with oral Ayurvedic medications such as Kutaj Ghan Vati, Udumbara Kvatha, and a mixture of Musta, Nagakesara, Lodhra, and Mukta Panchamrut Rasa. According to this study, the daily dosage of steroids and other anti-inflammatory medications was lowered by over 75%, as well as the symptoms and indicators.

In a different study, five patients with ulcerative colitis received eight days of Pichha basti as part of a yoga basti regimen. This study found that the indications and symptoms were reduced by almost 73%, which was a highly significant outcome. Fifty patients with UC clinical symptoms and a confirmed endoscopic diagnosis were enrolled in one research. For four weeks, each patient got intricate Ayurvedic treatment. Ayurvedic dietary recommendations (avoidance of spicy, sour, fried, hot, and heavy food items), recto-colonic administration of Ficus glomerata, and oral administration of herbal medications (Holarrhena antidysenterica, Ficus agglomerate, Cyperus rotundus, Mesua ferrea, and Symplocos racemosa) were all part of the treatment.

Laboratory tests and clinical feature changes were evaluated in the patients. The frequency of bowl movements and the presence of blood in feces have significantly decreased, according to the results. There was also a significant decrease in the need for traditional standard medications. Weight loss, weakness, and stomach pain were among the symptoms that were greatly alleviated. A statistical improvement was also observed in laboratory values for hemoglobin, ESR, erythrocytes, and pus cells in stool.

7. Discussion

7.1. Case studies

To Evaluate the Efficacy of Panchamrutha Parpati In Grahani Roga

The present study was aimed to evaluate the efficacy of Panchamrutha Parpati in Grahani roga.

The treatment of Grahani roga was reviewed analysed and importance of Panchamrutha Parpati was highlighted 20 patients were selected for study. All the patients were given Panchamrutha Parpati in dose of 125 mg mixed with 1 gms of Jeerakachurna,thrice a day after food with adequate quantity of madhu.

The treatment was given for period of 60 days. At the end of 60 days of treatment 80%of cases were improved.i.e. Severity of features reduced up to 70-80% and 20% did not respond to the treatment in any manner.

The drug Panchamrutha Parpati helped in raising the level of Haemoglobin percentage as well as body weight. This proves it's Balya and Rasayana actions in patients of Grahani.

8. Ayurvedic Management of Raktatisara

On June 23, 2012, a 24-year-old Hindu unmarried male patient from Jaipur was seen in the Outdoor Wing of Arogyashala at the National Institute of Ayurveda in Jaipur. His main complaints were minor burning during the defecation process and bleeding per rectum following a bowel movement that had been occurring for ten days.Constipation and H/O mass prolapse per rectum are absent. The patient's vitals were found to be within normal limits. Normal sleep patterns, mildly reduced appetite, and a changed bowel habit—two to three times per day—with mucus at the end of the defecation and soft stool consistency were all present. Proctoscopy examination of the per rectum revealed normal sphincter tone, a congested and irritated rectal mucosa, and very minor patches of mucus membrane ulcerations. Therefore, it was recommended that the patient have their stool examined for macroscopic, microscopic,

and occult blood. According to the test results, there were no ova, cysts, or germs in the stool, and the occult blood was positive. Hb% was 15 g/dl.The patient was prescribed certain Ayurvedic oral medications at that time, but his prior symptoms did not improve.

He recommended a colonoscopy once more on July 13th, and the results showed that the rectum's distal 20 cm area had a lot of vascular pattern, that there were several areas of superficial ulcerations, that there was no friability, that there was no contact bleeding, and that the results were suggestive of either ulcerative or infectious colitis. At that point, a colonoscopy was taken for additional confirmation. On July 16, the histopathologic study indicated that the patient had chronic active colitis.The treatment strategy was changed at that point to include oral medications, and Picchabasti3 was started for 15 days.Picchabasti's substance did not match the texts.

Shalmali4 (stem bark 10–15), Mocharasa, Yastimadhu, Lodhra, Nagkeshar, and Kutaja Churna with milk were the ingredients we used. This basti was administered in 70–80 ml Anuvasana form after the meal.The fourth day after beginning Picchabasti, the symptoms were relieved. The prior symptoms were completely alleviated. A second stool examination was conducted after the 15-day Picchabasti Course, and the results showed that the stool was negative for germs, cysts, and eggs as well as for occult blood.The patient is still taking oral medications and has no symptoms as of yet. [14]

9. Pravahika and its Management

Hypothesis of the dissertation is based on the fact that Pravahika is a disease due to Mandagni and selected drug Pippali mainly stimulates the Agni.

For the study 20 patients were selected between age group of 1 to 60 years based on symptomatology and microscopic and macroscopic examination of stools.Pippali Churna was given in the dose of 1 Karsha in two annakalas before food along with the anupana of 12 tabs of Patients began to show improvement after taking Pippali Churna for one day itself. Pravahika subsided after about an average of 8 days of the treatment. Among 20 patients selected.14 were reported. Among 20 patients selected,14 were reported to be cured, 4 were improved and 2 were not improved .also the Haemoglobin percentage of Patients increase. The study was concluded as the administration of Pippali Churna in Pravahika proved to be beneficial.

10. Conclusion

Ulcerative colitis remains a complex and multifaceted disease, with significant advances made in understanding its pathogenesis, treatment, and global epidemiology. While modern medicine has provided effective pharmacological and surgical interventions, the integration of alternative approaches, such as Ayurveda and homeopathy, offers additional pathways for managing the disease holistically. The future of Ulcerative Colitis research lies in personalized medicine, improved diagnostics, and long-term strategies to mitigate the disease's impact on patients' lives. This multidisciplinary approach holds promise for better therapeutic outcomes, increased remission rates, and enhanced patient care. Ongoing studies on the basis of Ayurveda are essential to improve patient outcomes and quality of life for those affected by ulcerative colitis. The main disadvantage of UC management by Ayurveda Methods is a time required but Ayurveda Methods are the safest way to treat the Ulcerative colitis.

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None.

12. Conflict of Interest

None.

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