ACADEMIA ONE
PEER REVIEWED JOURNALS

Open Herald: Periodical of Methodical Research
Volume 3, Issue 7, July, 2025
ISSN (E): 2810-6385
Website: https://academiaone.org/index.php/6

AN EFFECTIVE BIOLOGICALLY ACTIVE SUBSTITUTE FOR COLON ULCER TREATMENT IS "ASKOLIT", AGAINST FOR ULCERATIVE COLITIS

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Annotation
The article discusses about how effect ASKOLIT for ulcerative colitis and the distribution areas of the plants, as well as the unsurpassed role of leaves in promoting human health, the chemical composition and biological significance of vitamins ASKOLIT, as well as several recommendations for the treatment of ulcerative colitis modern medicine. traditional medicine based on the compound leaves plants Calendula officinalis and inula helenium, as well as data on the mechanism of action on the colon wall and their discussion.
Keywords:
ulcerative colitis, askolit, biologically active substitute.

Introduction

Immune inflammation of the colon's mucous membrane is a hallmark of ulcerative colitis (UC), a chronic illness of the colon.Only the colon is impacted by UC (except from retrograde ileitis), the rectum is inevitably implicated, and inflammation is typically diffuse and restricted to the mucous membrane (excluding from acute severe colitis). It should be particularly highlighted that complete colitis encompasses a subtotal lesion of the colon proximal to the left bend, and proctosigmoiditis is included in the notion of left-sided UC. Depending on the current's characteristics, there are:Acute course, which lasts less than six months after the disease first manifests; Chronic continuous course, which lasts less than six months while receiving appropriate treatment; and Chronic recurrent course, which lasts more than six months.

The severity of the current exacerbation (attack) should be evaluated using the UC activity index (Mayo Index), which is typically used in clinical trials, and the straightforward Truelove-Witts criteria, which are typically used in routine clinical practice, in order to properly formulate a diagnosis and choose treatment strategies.

The so-called "super-severe or extremely severe attack" of UC is frequently observed in clinical practice; it is typified by diarrhea occurring more than ten to fifteen times per day, a progressive hemoglobin decline, fever above 38 °C, severe hypoproteinemia and electrolyte imbalances, and elevated C-reactive protein (CRP) values. Such colitis is treated differently from other types of colitis.

Literature written in English refers to this illness as "acute severe UC."It is unknown what causes UC and other inflammatory bowel disorders (IBD). A genetic predisposition, deficiencies in innate and acquired immunity, gut microbiota problems, and environmental variables are some of the

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factors that contribute to the development of the illness. There are around 100 known genetic polymorphisms linked to UC. Perversion of adaptive immunity results from genetic determinism, which also alters the innate immune response, autophagy, the epithelial barrier, and the processes of microbe identification. A major flaw that predisposes people to developing IBD is a failure of dendritic cells to recognize bacterial molecular markers, or patterns, which causes signaling pro-inflammatory pathways to become hyperactivated.

Because of a decline in the percentage of anaerobic bacteria, mostly Firmicutes and Bacteroidetes, IBD also exhibits a reduction in the variety of intestinal microflora. In light of this, triggering factors such as smoking, stress, vitamin D deficiency, a diet high in animal protein and low in dietary fiber, intestinal infections—particularly those caused by cytomegalovirus and Clostridium difficile—and intestinal infections all contribute to the development of IBD.

Proinflammatory cytokines like tumor necrosis factor alpha (TNFa), interlekines 1, 12, 23, 17 (IL 1, IL 12, IL 23, IL 17), and other cell adhesion molecules are overexpressed as a result of the mutual influence of genetic and predisposing factors. These subpopulations of T lymphocytes include T helper cells of types 1, 2, and 17 and regulatory T lymphocytes at different stages of inflammation. These conditions lead to the loss of the colon mucosa, inflammatory lymphoplasmocytic infiltration, and macroscopic alterations that are typical of ulcerative colitis.

In Europe, the highest frequency of UC is presently 505/100000 of the population; in other areas, the incidence varies between 0.6 and 24.3 cases per 100,000. In North America, the greatest prevalence of UC is 19.2/100,000, while in Europe, it is 24.3/100,000.There is little information available on the prevalence of UC in Uzbekistan. Western areas and northern latitudes have greater rates of UC prevalence. Although UC is less common in Asia, its incidence and prevalence are rising at the moment. Compared to members of the Asian and Mongoloids, Caucasians are more likely to have the illness.The 20–30 age range is when the incidence peaks, and in many nations, the 60–70 age range is when the incidence peaks again. The frequency is almost equal for men and women.

Four clinical syndromes are part of the clinical picture of UC: intestinal illness. Common intestinal symptoms include blood in the stool (95–100%), diarrhea, which occurs more at night (65% of cases), tenesmus (more frequently associated with proctitis and proctosigmoiditis), and occasionally tenesmus along with constipation and a distal restricted lesion. Tenesmus is more common in the clinical picture of proctitis and proctosigmoiditis, and diarrhea may not be present. Abdominal discomfort is uncommon in UC, in contrast to BC. Moderate spastic abdominal pain syndrome is possible, usually in front of a chair.

Endotoxemia is a symptom of systemic inflammation brought on by the colon's inflammatory process being overactive. To differing degrees, endotoxemia coexists with moderate and severe types of UC. General intoxication, fever, tachycardia, anemia, elevated ESR, leukocytosis, thrombocytosis, and elevated levels of acute-phase proteins, such as fibrinogen and CRP, are the primary symptoms.

Diarrhea, toxemia, high protein loss in the feces via exudation, and poor water and electrolyte absorption are the causes of metabolic diseases. Typical clinical signs include dehydration, hypoproteinemia, hypoalbuminemia with the onset of edematous syndrome, hypokalemia and other electrolyte problems, hypovitaminosis, and weight loss (often to the point of fatigue). 20–25% of UC cases include extra-intestinal systemic symptoms (ECP), which typically coexist with more severe forms of the illness.

The primary intestinal symptoms of aggravation are accompanied by autoimmune signs linked to the activity of the inflammatory process, which go away with them when the condition is treated.

Regardless of whether the underlying disease is in an exacerbation or remission, autoimmune manifestations that are unrelated to the process' activity (often referred to as "concomitant autoimmune diseases" in English literature) tend to worsen and frequently indicate a poor prognosis for the illness.Intestinal bleeding, colon perforation, toxic dilatation, and colorectal malignancy are among the intestinal consequences of ulcerative colitis. as surgery is typically necessary to correct these issues.

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The following criteria are used to establish a diagnosis or condition based on pathognomonic evidence: instrumental examination, laboratory testing, physical examination, and anamnestic data.For UC, there are no clear-cut diagnostic standards. A combination of the patient's medical history, clinical presentation, and common endoscopic and histological alterations are used to make the diagnosis.

Establishing the diagnosis of UC, evaluating its activity, and resolving the colectomy problem all need a colonoscopy. Although there are no particular endoscopic symptoms, the primary way of diagnosing UC is endoscopic examination of the colon. These are characterized by mucous membrane-limited diffuse inflammation that begins in the rectum and spreads proximally, with a distinct boundary of inflammation. The absence of vascular pattern, the presence of erosions and ulceration, and contact vulnerability—the release of blood upon contact with the endoscope—are the best indicators of endoscopic activity in UC. Colorectal cancer must be ruled out in order to discover chronic intestinal constriction against the backdrop of UC.

Medication prescriptions, surgery, emotional assistance, and dietary advice are examples of therapeutic approaches for UC. Currently, the "Treat-to-target (T2T)" strategy—which translates to "Treatment to achieve the goal"—defines the objectives of UC therapy on a global scale. Achieving long-term therapy effects, avoiding complications, decreasing hospitalizations, lowering the risk of surgery and colon cancer, enhancing quality of life, and lowering the incidence of impairment in patients with chronic illnesses are the goals of this idea. The objectives of UC therapy, as seen from the perspective of routine clinical practice, are to attain and sustain endoscopic and clinical remission free of steroids for an extended period of time (discontinuation of GCS within 12 weeks following the initiation of therapy).

According to the "T2T" approach for UC, the main objective of treatment should be the full alleviation of clinical symptoms as described by the patient, which include normalization of stool and the absence of blood in the stool. Endoscopic remission must be obtained. Timely surgical therapy is a specific objective as the disease progresses and/or life-threatening complications arise.The severity of the attack, the size of the colon lesion, the presence of HCV, the length of the anamnesis, the efficacy and safety of prior therapy, the risk of UC complications, and the existence of risk factors for a poor prognosis of the course of UC all influence the choice of conservative or surgical treatment.

The fact that the therapeutic ASKOLIT is a biologically active substance whose entire composition is composed of medicinal plants is another significant aspect of modern medicine. A common treatment for colon ulcers is ASKOLIT. It is advised to consume a decoction of the physiologically active ingredient "ASKOLIT" 30 minutes after meals or 100–150 milliliters per hour before meals. The fact that the therapeutic ASKOLIT is a biologically active substance whose entire composition is composed of medicinal plants is another crucial aspect of modern medicine.

ASKOLIT is widely used in Colon ulcers. A decoction of the biologically active substance "ASKOLIT" is drunk 100-150 ml an hour before meals or recommended to be drunk half an hour after meals. this ASKOLIT biologically active substance helps to heal wounds as well as ensure that the intestinal flora works in one meior. this biologically active substance can then be consumed by patients whose side effects have disappeared. Recommended by the Sanitary and Epidemiological Center of the Ministry of Health of Uzbekistan.

When investigating these impacts, we have come to a number of experimental results. We chose and then looked at the combination with the greatest antioxidant in these tests since it is known that patients with ulcerative colitis have a lack of microelements called flavonoids and an increased demand for amino acids.

Materials and research methods

Use reactive and equipment. South acid “Macklin” from (China), Sali acid “Rhydburg Pharmaceuticals” (Germany) from, kversetin, apigenin, kempferol “Re” (China), zinc, and natural sources, the application of extraction and column chromatographic the method separated obtained.

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HPLC - level clean in water, asetonitril, chemical clean in the brand acetic acid and sodium in manufacturing of chemical agents was used.

The plant in the structure of polifenollar the amount of Japan Shimadzu company work produced LC-40 Nexera Lite high effective liquid in chromatographic out was carried.

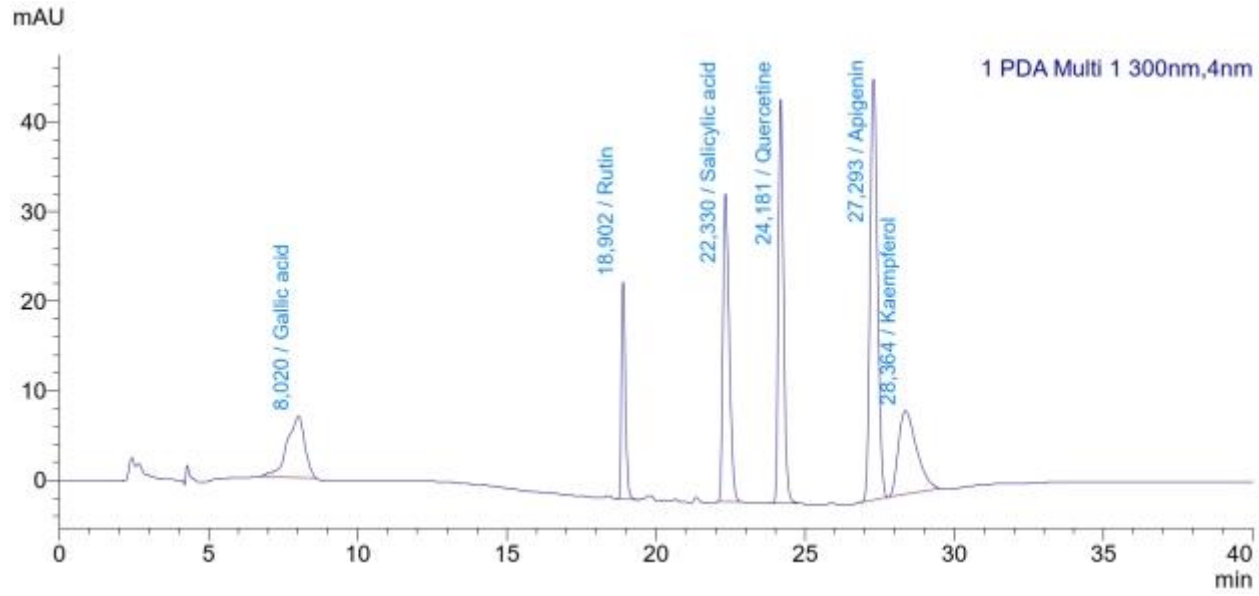
Standard solution preparation. South acid (5,2 mg), sali acid (5,2 mg), ruth, and (5 mg), kversetin (5 mg), apigenin (5 mg), kempferol (5 mg) 96 % in ethanol for 20 minutes in ultrasonic bath was dissolved and 50 ml tube were transferred, ethanol with the line is delivered. Every a solutionfrom 200 mk at from taken and were mixed and them way with a total of 4 different solution was prepared. Every a solution vialaga was poured and analysis for was used.

Plant ekstract prepare. Fenol combinationof products extraction make it to 1 g for checking our workshops and sample OHAUS company (Usa) by worked out have been NV222 branded scales 0,01 g in the accuracy of pulled out a50-ml volume of shape into the tube was put and 25 ml of 96 % ethanol was added. The mixture GT sonic the hedgehog-D3 (China) branded ultrasonic bath 60 oC at a temperature of 20 minutes for the extraction was. Then the mixture, measuring the tube in ethanol with 25 ml to was delivered. Extract from 1.5 ml , the amount of Mini-7 branded (BIOBASE, China) in centrifuga 7000 ayl./min at a speed of sentrifugalandi and 0,45 mkm syringe filter filtered and analysis for was used.chromatographic conditions. combination of products you select determine. S worth tandy solution, the sample extract C18 pack pants GEESE (150 × 4.6 mm; 5 mkm, Shimadzu, Japan) reverse phase for and atsetonitril (A) and acetic acid of water at 0,5 % solution (B) up I find gradiyenti the mobility of the phase of (1table) were used. size 1to 0 at mk, flow rate of 0,5 ml/min , and the columns of termostat 40 oC as has been defined. Fen combination of the world's analytical signal (peak area) 300 nmat recorded (1-picture).

1table.

Mobility phase gradiyenti program.

Time	**Atsetonitril** (A), %	0.5 % acetic acid (B), %
0	5	95
5	5	95
17	40	60
22	40	60
22,1	5	95
40	Finish	



1-picture. Standards, that's a 300 nm in chromatographic.

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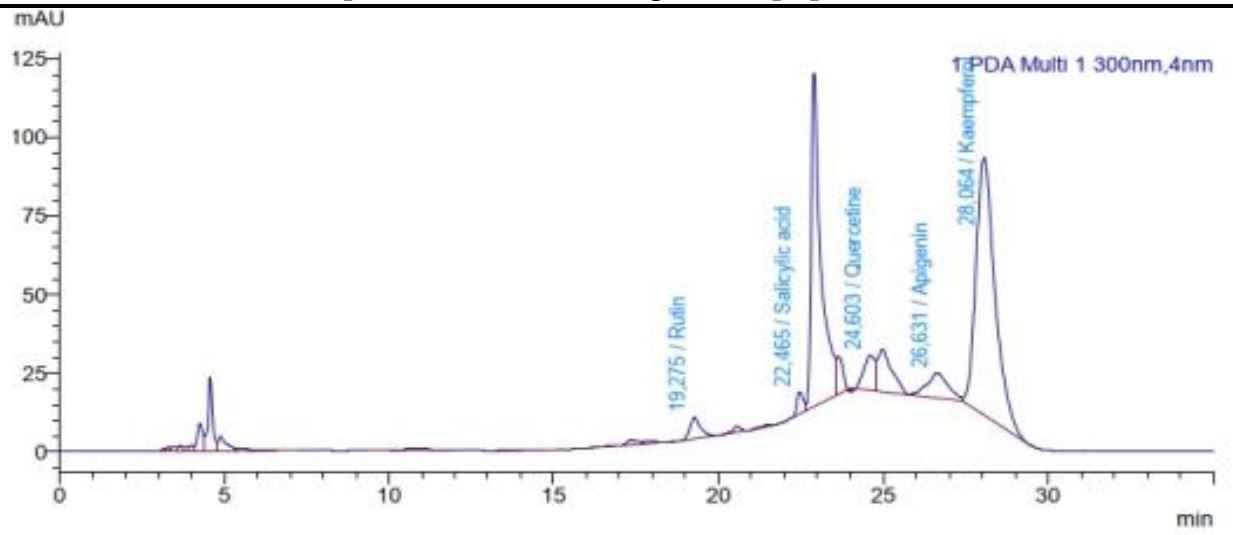

2-picture. The sample extract in the composition poliphenol chromatographic.

2table.

In the extract poliphenol the amount of and the hold time.

Phenol a combination of the name of	the hold time, sec	Concentration, mg/l	100 g of the sample , the amountof mg
Gallic aci the	Detected	0	0,000
ruth may	19,275	11,305	28,263
Salicylic aci	22,465	3,354	8,385
Quercetine	24,603	7,608	19,020
Apigenin	26,631	7,401	18,503
Kaempferol	28,064	139,605	349,013

Obtained results.

The sample extract in the composition phenol compounds amount to determine. Massos of 1 g of the sample extract chromatographic is (2-pic) and the results on the basis of the amount of compounds in the composition of 100 g of sample phenol with the following formula 3-tablesbrought.

$$X = \frac{C_{in\,ph\,in\,faen} \cdot V_{extract}}{m_{samples}} \cdot 100\ g$$

Here, X – phenol in the structure of the combination of the amount of 100 grams of fruit, mg;
Cph agent. xn – phenol detected with the combination of the method of extract in the composition yussx concentration, mg/l;
Vextract – the size of the sample extract, l;
msample – extract extracted for the preparation of the mass of the sample.

Conclusion

Ulcerative colitis is an inflammatory illness of unclear cause affecting the rectum. Pathology is harmful for the development of significant consequences with a high risk of mortality. It is possible to normalize the condition and enhance the quality of life with prompt medical treatment and full attention to all instructions. Because of its high phenol content and antioxidant qualities, which are crucial for wound healing, ASKOLIT is thought to be the most effective therapy and preventative for colon ulcers.

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