

UNRAVELING THE MULTIFACETED LANDSCAPE OF ULCERATIVE COLITIS: A CONTEMPORARY CLINICAL PERSPECTIVE AND TREATMENT WITH ASKOLIT

Ibragim R. Askarov

Andijan State University Professor of the Department of Chemistry, Doctor of Chemistry,
Honored Inventor of Uzbekistan, Chairman of the TABOBAT Academy of Uzbekistan

<https://orcid.org/0000-0003-1625-0330>

Khabibullo N. Kodirov

Andijan State Medical Institute Assistant of the Department of Propaedeutics of Internal
Medicine

<https://orcid.org/0000-0001-6574-7576>

E-mail: kodirovkhabibullo6@gmail.com

Abstract: Ulcerative colitis (UC), a chronic inflammatory bowel disease (IBD), presents a significant clinical challenge. This article provides a contemporary clinical perspective, synthesizing current understanding of its pathogenesis, diagnostic modalities, and therapeutic strategies. We explore the complex interplay of genetic predisposition, environmental triggers, and immunological dysregulation that characterizes UC. Furthermore, we discuss the evolving landscape of treatment options, including conventional therapies and novel biologic agents, emphasizing personalized management based on disease severity and patient-specific factors.

Keywords: Ulcerative colitis, AskolitA, biologically active substitute. Inflammatory bowel disease, Pathogenesis, Diagnosis, Treatment, Biologics, Endoscopy, Immunomodulators, Personalized medicine.

Relevance:

This article provides a clinically relevant overview of UC for practicing physicians and researchers, synthesizing current knowledge and highlighting the importance of personalized management strategies. The evolving understanding of UC pathogenesis and the development of novel therapies necessitate a contemporary clinical perspective to optimize patient care.

Research Objective:

To provide a comprehensive and clinically relevant overview of ulcerative colitis, encompassing its pathogenesis, diagnostic modalities, and therapeutic strategies, with an emphasis on personalized medicine and contemporary treatment options.

Materials and Methods:

Use reactive and equipment. ASKOLIT- Vitamin b₁₂ “Rhydburg Pharmaceuticals” (Germany), vitamin c - “Carl Roth GmbH” (Germany), B₉ “ds nutritional products GmbH” (from Germany), B₁, B₂, B₃, B₆, PP vitamins “BLDPharm” (China) is obtained. At the level of HPLC in clean water, acetonitrile, chemical clean in the brand acetic acid and sodium alkali manufacturing of chemical agents to use led.

The plant composition of the water-soluble vitamins produced in Japan, the amount of the Shimadzu LC-Nexera was carried out in liquid Lite 40 highly effective [1].

Prepare the standard solution. ASKOLIT - C (CAS 50-81-7), B₁ (CAS 59-43-8), B₆ (CAS 58-56-0), B₃ (CAS 59-67-6), B₁₂ (CAS 68-19-9), and PP (CAS 98-92-0) vitamin solution (100 mg/l) of 5 mg of vitamin A from the amount of 50 ml of 0.1 N HCl solution and dissolved is prepared. B₂ (CAS 83-88-5) and B₉ (CAS 59-30-3) of the standard solution to 50 ml of 5 mg of this vitamin of vitamin 0.025% sodium dissolved in the alkali solution was prepared. Then the initial B₁, B₆, B₃, B₁₂, PP vitamin 200 µg and mixed at the concentration of each vitamin were obtained from 14.286 mg/l solution was prepared. 7.143 hence, 3.571, 1.786 mg/l standard solution was prepared. Also vitamin C 286, 143, 71.5, 57.2 mg/l with the concentration of the standard solution are prepared. Graphics for drawing Kalibrlovchi 0 mg/l concentration in pure water was used.

The sample extract select prepare. Water-soluble vitamins for the extraction of 1 g from our sample for checking workshops and measure out 50 ml of volume konussimon was put into the tube and 25 ml of 0.1 N HCl was added to a solution of Li. The mixture GT sonic the hedgehog-D3 (China) branded ultrasonic bath at 60 °C at a temperature of 20 minutes for the extraction was. Then the mixture sovitilib, filtrlangan with the water in the tube and measuring 25 ml was delivered. Extract from the amount of 1.5 ml of 0.22 µm Li syringe filter was filtered and used for the analysis laid out and in vialaga.

Vitamin products to determine. Sworth tandy solution and sample extract LC-40 pump, TB-40 autosampler, SP-M40 photo-with diode matrix from detector (PDA) is LC-Nexera chromatographic liquid Lite 40 highly effective and LabSolutions ver. 6.92 software were analyzed using. The Geese C18 pack pants (150 × 4.6 mm; 5 µm, Shimadzu, Japan) by reverse-phase

columns of the asetonitril (a) and acetic acid in water at 0,2and 5% solution of (b) consisting of a gradient in the phase mobility (table 1) were used. In'ektsiya size 1mk at 0, 0 is the flow rate,6 ml/min and columns thermostat temperature of 40 °C has been identified as. Of each vitamin analytical signal (peak area) of the threeunits of the wave lengths, 265, 291, 550 nm at recordeddi (1-3 pictures). Gradiyenti for 15 minutes was used to determine vitamin c (table 2) and 265 nm wave length is the measure of the analytical signal.

1 table.

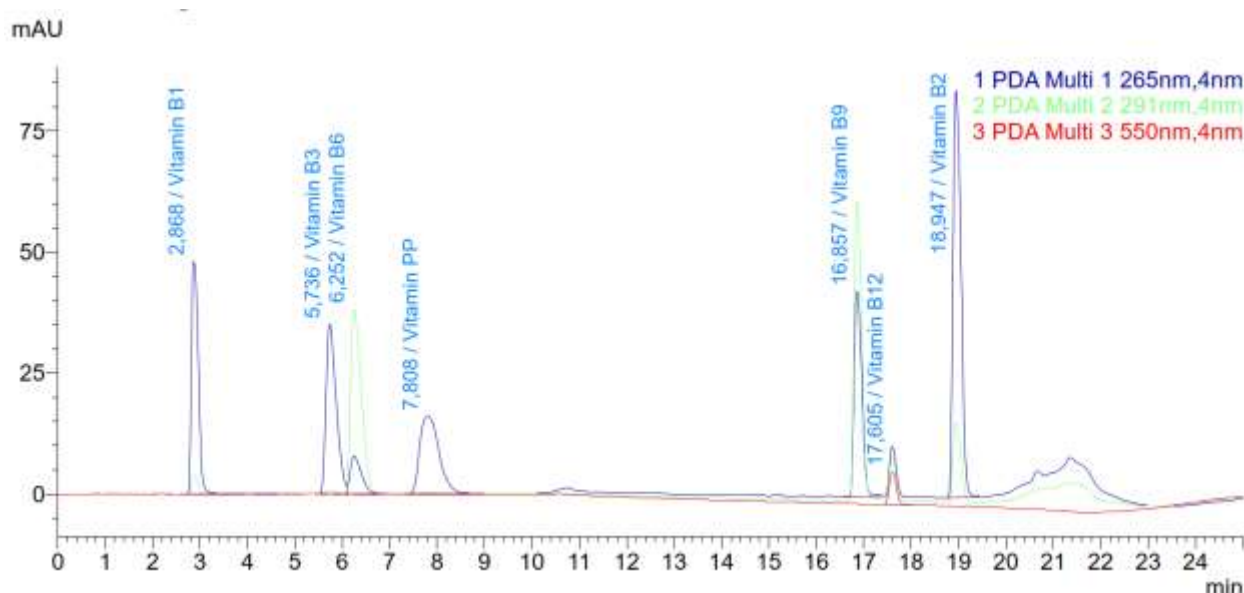
In the determination of vitamin harakat gradiyenti measure the phase of the program.

Time, minutes	Atsetonitril (A), %	0.5% acetic acid li (B), %
0	0	100
3	0	100
14	20	80
17	50	50
18	0	100
25	Finish	

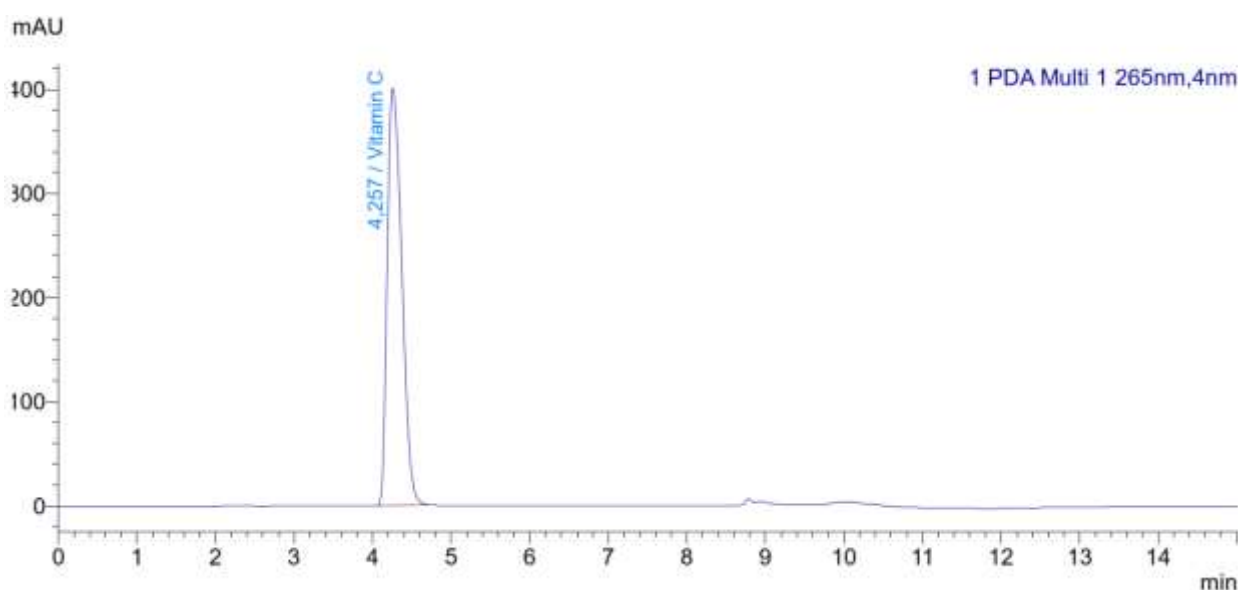
2table.

In determining the amount of vitamin c in harakat gradiyenti measure the phase of the program.

Time, minutes	Atsetonitril (A), %	0.5% acetic acid li (B), %
0	0	100
2	0	100
6	50	50
6,01	0	100
15	Finish	



1-picture. Vita see also:s standard solutionshis nest xromatrammasi.



2-picture. Vita see also: C xromatogrammasi of the standard solution.

The results obtained.

Namu to extract in the composition vitaminlarni determine. The sample extract xromatogrammac (3-4 picturess) were taken and the results on the basis of 100 g of the sample, the amount of vitamins in the composition of the following formula is 3-tablesbrought.

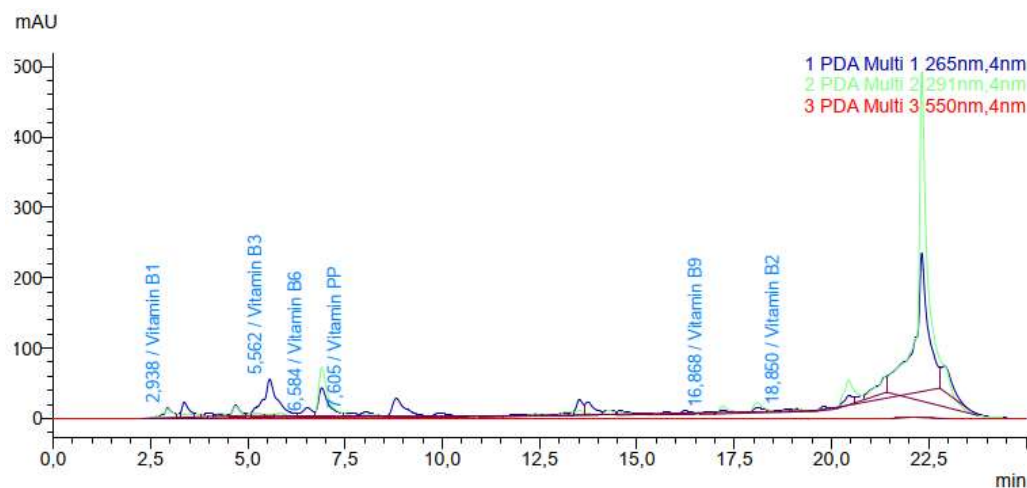
$$X = \frac{C_{\text{st.vitus}} \cdot V_{\text{extract}}}{m_{\text{samples}}} \cdot 100 \text{ g}$$

Here, X – amount of vitamin content in 100 grams of fruit, mg;

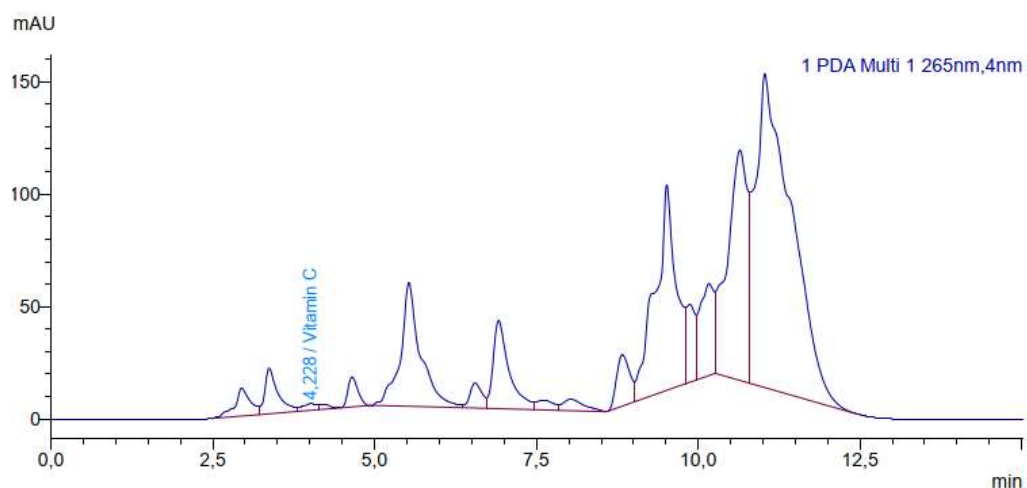
$C_{\text{st. vitus}}$ – extract vitamin content in YuSSX detected with the method of concentration, mg/l;

V_{extract} – the size of the sample extract, l;

m_{sample} – extract extracted for the preparation of the mass of the sample.



3-point. The sample extract and vitamin content xromatogramma identify insi.



4-picture. The sample extract and vitamin content xromatogramma identify insi.

3-the table.

Amount of vitamin a in the extract and hold the time.

Vitamin	hold time, sec	Concentration, mg/l,	the amount of 100 g of the sample, mg
vitamin b ₁	2,938	6,631	16,578
vitamin b ₃	5,562	35,897	89,743
vitamin pp	7,605	2,925	7,313
vitamin b ₉	16,868	0,642	1,605
vitamin b ₂	18,85	0,956	2,390

vitamin b ₆	6,584	0,849	2,123
vitamin b ₁₂	is detected	0	0,000
vitamin c	4,228	0,889	2,223

Results:

- **Pathogenesis:** The pathogenesis of UC involves a complex interplay of genetic, environmental, and immunological factors. Genetic susceptibility, evidenced by familial clustering and association with specific gene loci (e.g., IL23R, ATG16L1), predisposes individuals to the disease. Environmental triggers, such as alterations in the gut microbiome and dietary factors, may initiate or exacerbate inflammation. Immunologically, UC is characterized by a dysregulated mucosal immune response, involving an aberrant T-cell activation and cytokine production. The disruption of the epithelial barrier and subsequent exposure of the lamina propria to luminal antigens contribute to the chronic inflammatory cascade.

- **Diagnosis:** A comprehensive diagnostic approach is crucial for accurate disease characterization. Clinical evaluation, including detailed history and physical examination, is essential. Endoscopic evaluation with mucosal biopsy remains the gold standard for confirming the diagnosis and assessing disease severity. Histopathological examination reveals characteristic features, such as crypt abscesses, mucosal inflammation, and depletion of goblet cells. Serological markers, such as anti-neutrophil cytoplasmic antibodies (pANCA), may aid in the diagnostic process. Fecal calprotectin, a non-invasive marker of intestinal inflammation, is increasingly used to assess disease activity and monitor treatment response.

- **Treatment:** The therapeutic approach to UC is tailored to disease severity and individual patient factors. Conventional therapies include 5-aminosalicylates (5-ASAs), corticosteroids, and immunomodulators (e.g., azathioprine, 6-mercaptopurine). 5-ASAs are the mainstay of maintenance therapy for mild to moderate UC. Corticosteroids are used for induction of remission in moderate to severe flares. Immunomodulators are employed for maintenance therapy and to reduce corticosteroid dependence.

- **Biologic agents,** such as anti-tumor necrosis factor (TNF) antibodies (e.g., infliximab, adalimumab), anti-integrin antibodies (e.g., vedolizumab), and anti-interleukin-12/23 antibodies (e.g., ustekinumab), have revolutionized the treatment of UC. These agents target specific components of the immune system, providing effective control of inflammation in patients with moderate to severe disease. Tofacitinib, a Janus kinase (JAK) inhibitor, offers an alternative oral therapy for patients who are intolerant or unresponsive to biologics.

- **the most important thing is the addition of natural biological food clot ASKOLIT**
- Surgical intervention, including colectomy, may be necessary in patients with severe refractory disease or complications such as toxic megacolon or dysplasia.
- Personalized medicine, Tailoring treatment based on disease severity, patient-specific factors, and biomarker data is increasingly emphasized.

Discussion:

The management of UC requires a multidisciplinary approach, involving gastroenterologists, surgeons, and other healthcare professionals. Long-term management focuses on achieving and maintaining clinical remission, preventing complications, and improving patients' quality of life. Regular monitoring of disease activity, including endoscopic evaluation and biomarker assessment, is essential. Patient education and support are critical components of comprehensive care.

The evolving landscape of UC treatment offers promising avenues for improving patient outcomes. Further research is needed to elucidate the precise mechanisms underlying disease pathogenesis and to develop more targeted and effective therapies. The integration of precision medicine approaches, including genomics and proteomics, holds the potential to personalize treatment and optimize patient care.

Conclusions:

Ulcerative colitis is a complex and challenging disease. A comprehensive understanding of its pathogenesis, diagnostic modalities, and therapeutic strategies is essential for effective management. A personalized approach, incorporating conventional and novel therapies, is crucial for achieving optimal patient outcomes. Continued research and collaboration are vital for advancing our understanding of UC and improving the lives of affected individuals.

References:

1. Ungaro R, Mehandru S, et al. Ulcerative colitis. *Lancet*. 2017;389(10080):1756-1770
2. Neurath MF, Travis SPL. Mucosal healing in inflammatory bowel diseases: a systemic review. *Gut*. 2012;61(11):1619-1635
3. Feagan BG, Sandborn WJ, D'Haens G, et.al. Tofacitinib as induction and maintenance therapy for ulcerative colitis. *N Engl J Med*. 2017;376(18):1723-1736
4. Travis SPL, Lémann M, Lichtenstein GR, et al. ECCO consensus on the diagnosis and management of ulcerative colitis in adults. *J Crohns Colitis*. 2013;7(1):1-33. (European Crohn's and Colitis Organisation guidelines on UC management.)

5. Rutgeerts P, Sandborn WJ, Feagan BG, et al. Infliximab for induction and maintenance therapy for ulcerative colitis. *N Engl J Med*. 2005;353(23):2462-2476. (A pivotal study demonstrating the efficacy of infliximab in UC.)
6. Sands BE, Feagan BG, van Assche G, et al. Vedolizumab as induction and maintenance therapy for ulcerative colitis. *N Engl J Med*. 2013;369(8):699-712. (Key trial for the integrin inhibitor vedolizumab in UC.)
7. Sandborn WJ, Ghosh S, Panaccione R, et al. Tofacitinib as induction and maintenance therapy for ulcerative colitis. *N Engl J Med*. 2017;377(11):1041-1050. (Landmark trial for the JAK inhibitor tofacitinib in UC.)
8. Dignass A, Lindsay JO, Sturm A, et al. Second European evidence-based consensus on the diagnosis and management of ulcerative colitis: Part 1: Definitions and diagnosis. *J Crohns Colitis*. 2012;6(1):5-19. (Focuses on the diagnostic aspects of UC.)
9. Dignass A, Lindsay JO, Sturm A, et al. Second European evidence-based consensus on the diagnosis and management of ulcerative colitis: Part 2: Current management. *J Crohns Colitis*. 2012;6(10):991-1030. (Focuses on the treatment strategies for UC.)
10. Roda G, Jovani M, Torres J, et al. Fecal calprotectin in ulcerative colitis: a systematic review and meta-analysis. *Inflamm Bowel Dis*. 2017;23(12):2083-2092. (A comprehensive review on the role of fecal calprotectin in UC.)