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ORIGINAL ARTICLE

The Non-Invasive Markers for the Diagnosis and Follow-Up of Inflammatory Bowel Diseases: Serum Adamts-7 And Adamts-12

İnflamatuvar Barsak Hastalıklarının Tanı ve Takibi İçin İnvazif Olmayan Belirteçler: Serum Adamts-7 ve Adamts-12

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ABSTRACT

Aim: In this study, we aimed to evaluate the usability of ADAMTS-7 and ADAMTS-12 which are associated with inflammation as non-invasive markers for the diagnosis and follow-up of IBD
Materials and Methods: A total of 200 patients with IBD were included in this study. 96 of patients had ulcerative colitis (UC) (48 active, 48 in remission), 104 had Crohn's disease (CD) (53 active, 51 in remission), and 40 healthy controls were included. Disease activity scores in UC and CD, laboratory parameters, and ADAMTS-7 and ADAMTS-12 values in all groups were studied and compared using statistical methods.
Results: ADAMTS-7 and ADAMTS-12 levels were significantly lower in the UC group than in the healthy control group (p=0.035 and p < 0.001; respectively). ADAMTS-7 and ADAMTS-12 levels were also significantly lower in the CD group than in the healthy control group (p=0.026 and p < 0.001; respectively). In patients with CD, a significant difference was identified between the levels of ADAMTS-7 and ADAMTS-12 in patients with perianal disease compared to those without perianal disease (p=0.026 and p < 0.001, respectively). There was no significant difference in ADAMTS-7 and ADAMTS-12 levels between UC and CD patients(p>0.05). UC groups age was between 18-64 years with mean 47.26 and CD group's age were between 18-65 years with mean 41.52 .
Conclusions: The study shows that serum ADAMTS-7 and ADAMTS-12 levels can be valuable as non-invasive tests for the diagnosis of UC and CD and for predicting the presence of perianal disease in CD.

Keywords: ADAMTS-7, ADAMTS-12, colitis ulcerative, crohn's disease.

ÖZ

Amaç: Bu çalışmada, inflamasyonla ilişkili non-invaziv belirteçler olan ADAMTS-7 ve ADAMTS-12'nin İBH'nin tanı ve takibinde kullanılabilirliğini değerlendirmeyi amaçladık.
Gereç ve Yöntemler: Bu çalışmaya toplam 200 IBH hastası dahil edildi. Hastaların 96'sında Ülseratif Kolit (ÜK) (48 aktif, 48 remisyonda), 104'ünde Crohn Hastalığı (CH) (53 aktif, 51 remisyonda) vardı ve 40 sağlıklı kontrol çalışmaya dahil edildi. Tüm gruplardaki ÜK ve CH'deki hastalık aktivite skorları, laboratuvar parametreleri ve ADAMTS-7 ve ADAMTS-12 değerleri incelendi ve istatistiksel yöntemler kullanılarak karşılaştırıldı.
Bulgular: ADAMTS-7 ve ADAMTS-12 düzeyleri ÜK grubunda sağlıklı kontrol grubuna göre anlamlı derecede düşüktü (sırasıyla p=0,035 ve p < 0,001). ADAMTS-7 ve ADAMTS-12 düzeyleri aynı zamanda CH grubunda sağlıklı kontrol grubuna göre anlamlı derecede düşüktü (sırasıyla p=0,026 ve p < 0,001). CH hastalarında perianal hastalığı olan hastalarda perianal hastalığı olmayan hastalara kıyasla ADAMTS-7 ve ADAMTS-12 düzeyleri arasında anlamlı bir fark saptandı (sırasıyla p=0,026 ve p < 0,001). ÜK ve CH hastaları arasında ADAMTS-7 ve ADAMTS-12 düzeylerinde anlamlı bir fark yoktu (p>0,05). ÜK grubunda yaş aralığı 18-64 arasında ve ortalama 47.26 iken CH grubunda yaş aralığı 18-65 arasında ve ortalama 41.52 idi.
Sonuçlar: Bu çalışma serum ADAMTS-7 ve ADAMTS-12 düzeylerinin Ülseratif kolit ve Crohn hastalığı tanısında ve Crohn hastalığında perianal hastalık varlığının tahmininde invaziv olmayan testler olarak değerli olabileceğini göstermektedir.

Anahtar Kelimeler: ADAMTS-7, ADAMTS-12, crohn hastalığı, ülseratif kolit.

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Introduction

Inflammatory bowel diseases (IBD) are a group of chronic inflammatory disorders of unknown etiology in genetically predisposed individuals, characterized by periods of chronic exacerbation and remission.[1] The prevalence of IBD is increasing worldwide, in both industrialized and developing countries, making IBD a global health problem that seriously affects quality of life. In 2019, there were approximately 4.9 million cases of IBD worldwide[2],

Two major types of inflammatory bowel diseases are ulcerative colitis (UC) and Crohn's disease (CD).[3] Although there are some similarities in both types, they also have different clinicopathological features. CD is a transmural, inflammatory, progressive disease of the gastrointestinal (GI) tract. CD most commonly affects the terminal ileum and proximal colon but can also affect any part of the GI tract, from the mouth to the anus.[4] Complications such as stenosis, abscess, and fistula may occur in patients with CD. [5] CD involvement is focal, asymmetrical, and segmental, and the mucosa and lumen are normal in other areas. UC extends proximally from the anal verge in an uninterrupted pattern and involves part or the entire colon. The rectum is involved in more than 95% of the cases. The mucosa is diffusely and symmetrically involved in UC.[6, 7] Symptoms, laboratory examinations, endoscopic and radiological findings are used in the diagnosis of IBD and in the evaluation of disease activity.(8) Biochemical and fecal markers have been shown to be useful for disease follow-up, activity evaluation, and treatment response. However, a specific biomarker for UC and CD has not yet been identified. The most commonly used marker is fecal calprotectin, which is used to differentiate between irritable bowel syndrome, IBD, and other inflammatory conditions, and to evaluate IBD activity. It correlates with the severity of symptoms, especially in UC, and may predict relapse.[6, 9] In addition, it is a reliable marker in the evaluation of response to treatment.[7] In a study, Kamil Kozol et al. investigated lactadherin levels in IBD. This study revealed the need for non-invasive markers for diagnosing active IBD.[10]

Disintegrin-like and Metalloproteinase with Thrombospondin type-1 motif (ADAMTS) was first described in 1997 by Kuno et al. in association with colon cancer.[11] Some of the important physiological functions of ADAMTS are tissue remodeling, coagulation, angiogenesis, and ovulation. They are also active in pathological processes such as degradation of the extracellular matrix and basement membrane, tumor cell invasion, and metastasis.[12] ADAMTS-7 is found in the adult human heart, pancreas, kidneys, skeletal muscle, and liver. ADAMTS-7 proteinase is involved in many inflammatory processes, such as arthritis, atherosclerosis, and biliary fibrosis.[13] ADAMTS-7 leads to upregulation of proinflammatory cytokines such as TNF-α and IL-1b, thus ADAMTS-7 levels are expected to increase during the inflammatory process.

ADAMTS-12 has been detected in the musculoskeletal system, cartilage, tendons, and fetal lungs. It has been shown to be associated with many conditions, such as arthritis, intervertebral disc degeneration, inflammation, tumor invasion, and tumor metastasis. ADAMTS-12 performs essential roles in modulation and recovery from inflammatory processes such as colitis, endotoxic sepsis and pancreatitis.[14]

ADAMTS-12 also contributes to the development of events such as schizophrenia, gonadal differentiation, trophoblast invasion, and pediatric stroke.[15] In absence of ADAMTS-12, an elevated eosinophilic inflammation, together with increased levels of mast cells and IL-33, were observed in the alveolar environment suggesting a protective role of this proteinase against this inflammatory process. A protective role of ADAMTS-12 in inflammatory processes is critical. ADAMTS-12 is an inflammation-related protein which is necessary for normal inflammatory response. In this study, we aimed to evaluate the relationship between IBD and ADAMTS-7 and ADAMTS-12 which are related with inflammatory processes.

Materials and Methods

200 IBD patients older than 18 years of age who were followed up and treated at Gastroenterology-IBD Clinic between September 2012 and June 2015 were included in the study. 96 of the patients had UC (48 active - 48 in remission), 104 had CD (53 active, 51 in remission),and 40 healthy controls were included in the study. CD and UC are diagnosed based on a comprehensive evaluation of clinical, endoscopic, histological, surgical, imaging and laboratory findings. We used American College of Gastroenterology guideline for description of IBD. Patients with chronic renal failure, malignancy, chronic liver disease, cardiac disease, or pancreatic disease were excluded. Permission and approval of Medical Ethics Committee were obtained (Date: 30.05.2016, Decision no: 30/15). The study was conducted under the principles of the Declaration of Helsinki. In Ankara Dışkapı Training and Research Hospital, The Scientific Research Project Board awarded 5000 Turkish Liras in 2016. Data on patients and the control group were obtained from patient files and the hospital data-processing system.

The UC, CD, and control groups were evaluated by age, age at diagnosis, sex, disease involvement in the gastrointestinal tract, and disease behavior patterns. The control group consisted of people who had no history of disease, no medication use, and no symptoms. Routinely hemogram parameters, sedimentation, C-reactive protein (CRP) were examined, in addition to these tests serum ADAMTS-7 and serum ADAMTS-12 levels were studied in all groups.

The Truelove-Witts activity index [16] was used for the activation/remission status in patients with UC, and the Crohn's Disease Activity Index (CDAI)[17] was used for CD.

The samples taken, in addition to the tests used in the follow-up of the disease were kept at -80 °C after centrifugation. ADAMTS-7 (5–350 ng/mL) and ADAMTS-12 (2–100 ng/mL) levels were measured using the Eastbiopharm brand ELISA method at

Biochemistry Laboratory (China).

For group comparisons, the chi-square test was used for qualitative variables, the Mann-Whitney U test for quantitative variables comparing two groups, and the Kruskall-Wallis test for more than two groups. The statistical significance level (alpha) was set at 0.05. Data were analyzed using SPSS Version 29 (IBM SPSS, Armonk, New York, USA).

Results

Demographic Data of Patients and Laboratory Results

The demographic information, IBD disease types and behavioral patterns of the 200 IBD patients (96 UC, 104 CD) and 40 healthy controls are shown in Table 1. The laboratory data of the patients and the control groups are compared in Table 2.

Table 1. The demographic features, disease subtypes and behavioral patterns of patients and control group

	UC	CD	Healthy control
Age (years) ± SD	47.26 ± 13.67	41.52 ± 13.39	36.65 ± 7.22
Age at diagnosis (years) ± SD	40. 75 ± 12.96	37.5 ± 13.16	
Female/Male	37/59	40/64	20/20
Location/Extension			
Ileal	-	40	
Colonic	-	8	
Ileocolonic	-	56	
Extensive colitis	32	-	
Left colitis	32	-	
Proctitis	32	-	
Behavior pattern			
inflammatory	-	84	
structuring	-	7	
penetrating	-	13	

UC: Ulcerative colitis, CD: Crohn's disease, SD: Standard deviation

Table 2. Laboratory data and comparison of patients and control group

	UC	CD	Healthy control	p[a]	p[b]	p[c]
WBC (10^3/µL)	7.96 ± 2.26	8.26 ± 3.23	7,41 ± 1.47	0.955	0.428	0.434
HGB (gr/dl)	13.99 ± 1.86	13.04 ± 2.08	14.30 ± 1.29	**0.002**	0.783	**<0.001**
PLT (10^3/µL)	288.53 ± 108.07	315.09 ± 111.50	260.56 ± 38.68	0.095	0.493	**0.022**
NEU (10^3/µL)	5.16 ± 1.89	5.63 ± 2.78	4.30 ± 1.03	0.596	**0.028**	**0.02**
LYM (10^3/µL)	1.97 ± 0.67	1.84 ± 0.68	2.36 ± 0.62	0.175	**0.002**	**<0.001**
PCT (%)	0.24 ± 0.07	0.26 ± 0.08	0.22 ± 0.04	**0.033**	0.995	**0.045**
ESH (mm/h)	19.52 ± 17.91	31.37 ± 22.5	9.67 ± 9.53	**<0.001**	**<0.001**	**<0.001**
CRP (mg/L)	11.39 ± 22.98	27.10 ± 46.27	4.86 ± 6.05	**0.002**	**0.003**	**<0.001**

UC: Ulcerative colitis, CD: Crohn's disease, WBC: White Blood Cell, HGB: Hemoglobin, PLT: Platelet, NEU: neutrophil, LYM: Lymphocyte, PCT: Procalcitonin, ESH: Erythrocyte Sedimentation Rate, CRP: C-reactive protein

[a]:Statistical analysis of UC and CD Blood counts, ESR and CRP values

[b]:Statistical analysis of UC and healthy control group Blood counts, ESR and CRP values

[c]:Statistical analysis of CD and healthy control group Blood counts, ESR and CRP values

ADAMTS-7 and ADAMTS-12 Levels in Ulcerative Colitis

ADAMTS-7 and ADAMTS-12 levels were found to be lower in ulcerative colitis compared to the healthy control group (ADAMTS-7, ADAMTS-12 p values p=0.035, p<0.001, respectively). There was no significant difference in ADAMTS-7 and ADAMTS-12 levels between patients with active UC and UC in remission (p=0.364 and p=0.128, respectively). However, significant differences were found between the active UC and control groups (ADAMTS-7 and ADAMTS-12 p values were 0.017 and <0.001, respectively). In the comparison of UC in remission and the control group, ADAMTS-7 levels were found to be similar, whereas ADAMTS-12 levels were found to be significantly low in the UC remission group (ADAMTS -7, -12 p values were 0.193 and <0.001, respectively). There was no difference in ADAMTS-7 and ADAMTS-12 levels in UC according to the disease involvement sites (0.635 and 0.607, respectively). There was no statistically significant difference between ADAMTS-7 and ADAMTS-12 levels in UC patients in terms of the Truelove-Witts score (remission, mild, moderate, and severe) (0.606, p=0.489, respectively). ADAMTS-7 and ADAMTS-12 results for UC are shown in Table 3.

Table 3. ADAMTS-7 and ADAMTS-12 values in UC and their comparison between groups

	ADAMTS-7 (ng/ml)	p value	ADAMTS-12 (ng/ml)	p value
UC	115.66 ± 143.16	0.035	44.56 ± 25.43	<0.001
Healthy control	130.85 ± 119.59		66.43 ± 27.16	
UC activation	105.94 ± 139.01	0.364	42.08 ± 24.96	0.128
UC remission	125.39 ± 148.00		47.04 ± 25.93	

Table 3 Cont. ADAMTS-7 and ADAMTS-12 values in UC and their comparison between groups

		ADAMTS-7 (ng/ml)	p value	ADAMTS-12 (ng/ml)	p value
UC localization	**proctitis (n:32)**	86.07 ± 110.87	0.635	40.27 ± 20.73	0.607
	left colitis (n:32)	125.68 ± 164.06		45.47 ± 27.28	
	extensive (n:32)	135.24 ± 148.97		47.93 ± 27.88	
UC Truelove-Witts Score	**remission (n:48)**	125.39 ± 148.00	0.606	47.04 ± 25.93	0.489
	mild (n:14)	143.32 ± 189.69		46.73 ± 28.76	
	moderate (n:21)	69.99 ± 78.43		35.92 ± 13.08	
	severe (n:13)	135.09 ± 153.93		52.36 ± 36.87	

UC: Ulcerative colitis, ADAMTS: A Disintegrin-like and Metalloproteinase with Thrombospondin type-1 motif

ADAMTS-7 and ADAMTS-12 Levels in Crohn's Disease

ADAMTS-7 and ADAMTS-12 levels were found to be lower in Crohn's disease compared with the healthy control group (ADAMTS-7, ADAMTS-12 p values p=0.026 and p<0.001, respectively). There was no difference in ADAMTS-7 and ADAMTS-12 levels according to the disease involvement regions in CD (p=0.497 and 0.746, respectively). There was no statistically significant difference in ADAMTS-7 and ADAMTS-12 levels between the remission, mild, moderate and severe groups according to the CDAI score in CD patients (ADAMTS-7 p=0.904, ADAMTS-12 p=0.979). There was no statistically significant difference in ADAMTS-7 and ADAMTS-12 levels between the inflammatory, structuring and penetrating groups in terms of behavioral patterns in patients with CD (ADAMTS-7 p= 0.119, ADAMTS-12 p= 0.249). A statistically significant difference was found between ADAMTS-7 and ADAMTS-12 levels in groups with and without perianal disease involvement in patients with CD (0.046 and 0.024, respectively). The ADAMTS-7 and ADAMTS-12 results in CD are shown in Table 4.

Table 4. ADAMTS-7 and ADAMTS-12 values in CD and their comparison between groups

		ADAMTS-7 (ng/ml)	p value	ADAMTS-12 (ng/ml)	p value
CD		112.06 ± 159.38	**0.026**	40.04 ± 23.22	<0.001
Healthy control		130.85 ± 119.59		66.43 ± 27.16	
CD localization	**ileal (n:40)**	102.14 ± 145.15	0.497	39.32 ± 23.08	0.746
	colonic (n:8)	109.32 ± 109.35		43.19 ± 23.83	
	ileocolonic (n:56)	119.53 ± 176.00		23.61	
CD CDAI score	**remission (n:46)**	126.47 ± 184.46	0.904	42.50 ± 26.61	0.979
	mild (n:18)	95.86 ± 127.82		36.53 ± 17.83	
	moderate (n:27)	102.97 ± 138.75		38.87 ± 20.81	
	severe (n:13)	82.11 ± 90.45		36.33 ± 15.81	
CD behavioral pattern	**inflammatory (n:84)**	125.28 ± 173.57	0.119	41.99 ± 24.97	0.249
	structuring (n:7)	77.57 ± 72.34		36.84 ± 16.35	
	penetrating (n:13)	45.21 ± 16.97		29.12 ± 4.56	
CD perianal disease	**+ (n:22)**	154.98 ± 197.75	**0.046**	51.44 ± 29.56	**0.024**
	- (n:82)	100.04 ± 148.10		36.73 ± 20.31	

CD: Crohn's disease, ADAMTS: ADAMTS: A Disintegrin-like and Metalloproteinase with Thrombospondin type-1 motif, CDAI: Crohn Disease Activity Index

When comparing ADAMTS-7 and ADAMTS-12 levels between UC and CD, no statistically significant difference was found (ADAMTS-7 and ADAMTS-12 p values: p=0.349 and p=0.113, respectively).

Discussion

Research on noninvasive, inexpensive, and easily accessible markers to be used in the diagnosis and treatment processes and to predict the clinical course of IBD is still ongoing. Fecal calprotectin is effective in showing nonspecific intestinal inflammation in both CD and UC.[18-20] But sometimes the collection of fresh stool samples from children can present difficulties. Consequently, the utilization of blood tests can be considered beneficial in the context of screening and subsequent monitoring. Therefore, new markers need to be investigated. The relationship between ADAMTS-7 and ADAMTS-12 and inflammation, as well as the increased inflammatory response and impaired balance between pro- and anti-inflammatory cytokines in the pathogenesis of IBD, suggests that these biomarkers could be used in IBD.

In our study, a statistically significant difference was found between the UC and CD groups and between the UC and healthy control groups in terms of mean age. This heterogeneity is considered as a disadvantage of our study.

ADAMTS-7 and ADAMTS-12 levels between the UC and the healthy control groups and between the CD and the healthy control group were different statistically. These results show that ADAMTS-7 and ADAMTS-12 levels may be valuable as a non-invasive tests for the diagnosis of IBD.

There was no statistical difference in ADAMTS-7 and ADAMTS-12 levels between patients with CD and UC. These results suggest that ADAMTS-7 and ADAMTS-12 levels in the serum are not useful as non-invasive tests to differentiate between CD and UC.

There was no statistically significant difference between the ADAMTS-7 and ADAMTS-12 levels of patients in the UC activation period and those in remission. Likewise, there was no statistically significant difference between the ADAMTS-7 and ADAMTS-12 levels of patients in the CD activation period and those in remission. These results suggest that ADAMTS-7 and ADAMTS-12 levels can not be used to predict and differentiate IBD activation and remission periods.

When CD and UC were examined according to their localizations, no statistically significant difference was found between ADAMTS-7 and ADAMTS-12 levels in the differentiation between ileal, colonic and ileocolonic CD. Similarly, ADAMTS-7 and ADAMTS-12 levels were not statistically different for proctitis, left-type, and extensive colitis in UC. According to these results, ADAMTS-7 and ADAMTS-12 levels were not considered as guiding markers in differentiating the localizations of CD and UC involvement.

No statistically significant difference was found between ADAMTS-7 and ADAMTS-12 levels when IBD was grouped according to disease activity scores (Trulove-Witts and CDAI). ADAMTS-7 and ADAMTS-12 levels were not considered usable markers in the discrimination of activation and remission, as well as in the differentiation of activation severity.

A statistically significant difference was found in both serum ADAMTS-7 and ADAMTS-12 levels in terms of CD

perianal disease. This may be due to the more severe inflammation in the presence of perianal disease. Complications such as abscesses and fistulas seen in perianal disease may also have contributed to this situation.

This study was subject to three limitations. Firstly, the mean age of the control group was found to be lower than that of the IBD group.

Secondly, the sample size could have been increased. On the other hand, the use of these non-invasive markers in IBD patients was precious.

Thirdly the drug information about the patients could not be obtained so this situation limits our study.

Conclusion

This is the first study to evaluate whether serum ADAMTS-7 and ADAMTS-12 levels can be used in the diagnosis, typing, evaluation of activation and disease involvement sites. ADAMTS-7 and ADAMTS-12 levels did not differ in determining activation and remission in both UC and CD. However, in both groups, ADAMTS-7 and ADAMTS-12 levels were significantly lower the control group. Based on past data, ADAMTS-7 levels should increase in inflammatory processes, but our study found them to be low in UC and CD group than control group. ADAMTS-12 deficiency has been shown to cause inflammatory processes based on past data. In our study, it was found to be low in UC and CD group than control group accordance with previous information. The most important finding in our study is that the evaluation of serum ADAMTS-7 and ADAMTS-12 levels can be valuable as a non-invasive test in demonstrating the presence of perianal disease in CD.

Conflict of Interest

All authors declare that they have no conflict of interest in this study.

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References

1. Koçhan K, Erdem E, Babacan G, Paker N, Gökden Y, Saltürk AD ve ark. Inflamatuvar barsak hastalıklarının aktivite tayininde endoskopik aktivite indeksleri ile laboratuvar parametreleri arasındaki ilişki. Akademik Gastroenteroloji Dergisi. AGD. 2014;13(3):101-6.

2. Aebisher D, Bartusik-Aebisher D, Przygórzewska A, Oleś P, Woźnicki P, Kawczyk-Krupka A. Key Interleukins in Inflammatory Bowel Disease-A Review of Recent Studies. Int J Mol Sci. 2024;26(1):121.

3. Wehkamp J, Götz M, Herrlinger K, Steurer W, Stange EF. Chronisch entzündliche darmerkrankungen. Dtsch Arztebl Int. 2016;113(5):72-82.

4. Baumgart DC. The diagnosis and treatment of Crohn's disease and ulcerative colitis. Dtsch Arztebl Int. 2009;106(8):123-33

5. Stange E, Travis S, Vermeire S, Beglinger C, Kupcinkas L, Geboes K, et al. {paVon} Herbay A, Warren BF, Gasche C, Tilg H, Schreiber SW, Scholmerich J, Rinisch W, European Crohn's and Colitis Organisation European evidence based consensus on the diagnosis and management of Crohn's disease: definitions and diagnosis. Gut.2016;55(1):1-15.

6. Vos MD, Louis EJ, Jahnsen J, Vandervoort JG, Noman M, Dewit O, et al. Consecutive fecal calprotectin measurements to predict relapse in patients with ulcerative colitis receiving infliximab maintenance therapy. Inflamm Bowel Dis. 2013;19(10):2111-7.

7. Judd TA, Day AS, Lemberg DA, Turner D, Leach ST. Update of fecal markers of inflammation in inflammatory bowel disease. J Gastroenterol Hepatol. 2011;26(10):1493-9.

8. Clark C, Turner J. Diagnostic modalities for inflammatory bowel disease: serologic markers and endoscopy. Surg Clin North Am. 2015;95(6):1123-41.

9. Schoepfer AM, Beglinger C, Straumann A, Trummler M, Vavricka SR, Bruegger LE, et al. Fecal calprotectin correlates more closely with the Simple Endoscopic Score for Crohn's disease (SES-CD) than CRP, blood leukocytes, and the CDAI. Am J Gastroenterol. 2010;105(1):162-9.

10. Kozioł K, Owczarek D, Cibor D, Zwolińska-Wcisło M. Serum level of lactadherin (MFGE8) in inflamatory bowel disease. Folia Medica Cracov. 2023;63(3):15-29.

11. Kuno K, Kanada N, Nakashima E, Fujiki F, Ichimura F, Matsushima K. Molecular cloning of a gene encoding a new type of metalloproteinase-disintegrin family protein with thrombospondin motifs as an inflammation associated gene. J Biol Chem. 1997;272(1):556-62.

12. Tortorella MD, Malfait F, Barve RA, Shieh H-S, Malfait A-M. A review of the ADAMTS family, pharmaceutical targets of the future. Curr Pharm Des. 2009;15(20):2359-74.

13. Zhang Y, Lin J, Wei F. The function and roles of ADAMTS-7 in inflammatory diseases. Mediators of Inflamm. 2015;2015:801546

14. Mohamedi Y, Fontanil T, Cal S, Cobo T, Obaya Á J. ADAMTS-12: Functions and Challenges for a Complex Metalloprotease. Front Mol Biosci. 2021;8:686763.

15. Demircan K, Akyol S, Armutcu F. A multi-functional gene family from arthritis to cancer: a disintegrin-like metalloproteinase with thrombospondin type-1 motif (ADAMTS). Annals of Clinical and Analytical Medicine. ACAM. 2013;4(5):429-34.

16. Sc T, Lj W. Cortisone in ulcerative colitis; final report on a therapeutic trial. Br Med J. 1955;2(4947):1041-8.

17. Best WR, Becktel JM, Singleton JW, Kern Jr F. Development of a Crohn's disease activity index: National Cooperative Crohn's Disease Study. Gastroenterology. 1976;70(3):439-44.

18. Røseth A, Schmidt P, Fagerhol M. Correlation between faecal excretion of indium-111-labelled granulocytes and calprotectin, a granulocyte marker protein, in patients with inflammatory bowel disease. Scand J of Gastroenterol. 1999;34(1):50-4.

19. Tibble J, Teahon K, Thjodleifsson B, Roseth A, Sigthorsson G, Bridger S, et al. A simple method for assessing intestinal inflammation in Crohn's disease. Gut. 2000;47(4):506-13.

20. Costa F, Mumolo M, Bellini Ma, Romano M, Ceccarelli L, Arpe P, et al. Role of faecal calprotectin as non-invasive marker of intestinal inflammation. Dig Liver Dis. 2003;35(9):642-7.