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Original Article

Serum Calprotectin: A promising Biomarker for Inflammatory Bowel Disease [IBD]

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ABSTRACT

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Background: These days, inflammatory bowel disease [IBD] is growing more common. Its diagnosis relies on invasive techniques like colonoscopy and biopsy, and its activity is monitored by fecal calprotectin levels that have a low compliance rate, so there is a pressing need for a serum biomarker that is non-invasive, accepted, and accurate for diagnosing and monitoring IBD activity.

The aim of the work: The goal of our research is to study serum calprotectin as a candidate biomarker in IBD.

Methods: The study included 50 patients with IBD who were recruited from Ain Shams University Hospitals' Gastroenterology clinic. Sixty percent were diagnosed with ulcerative colitis, with half in activity and the other half in remission, and 40% were diagnosed with Chron's disease, with half in activity and the other half in remission. A control group of 20 apparently healthy individuals comparable in age and sex were also included in the study. All subjects had their serum calprotectin tested by ELISA in addition to their ESR and CRP measurements.

Results: Serum calprotectin levels were significantly higher in patients with IBD than in controls and in clinically active patients than those in remission in both UC and CD groups. Although there was a positive association between serum calprotectin levels and CRP and ESR, serum calprotectin had a higher diagnostic value than CRP and ESR due to its higher sensitivity and specificity. Our findings demonstrated that serum calprotectin and platelet count had a direct relationship, while serum calprotectin and serum albumin and hemoglobin levels had an inverse relationship.

Conclusion: Serum calprotectin levels are raised and linked to clinical activity in IBD patients, implying that it could be utilised as a clinically useful indicator of disease activity.

Keywords: Serum calprotectin; Biomarker; Inflammatory bowel disease; Crohn's disease; Ulcerative colitis.



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INTRODUCTION

Inflammatory bowel disease [IBD], which encompasses Ulcerative colitis [UC] and Crohn's disease [CD], is a set of gastrointestinal illnesses characterized by chronic inflammation. Although the cause of IBD is unknown many variables including environmental, genetic, and immunologic are thought to play a crucial part in the disease's etiology and pathogenesis. IBD is hypothesized to be the result of a falsely regulated immunological response to the host

intestinal microbiota in genetically predisposed individuals ^[1].

The standard method for initial diagnosis, disease severity assessment, and therapy response evaluation of IBD is presently colonoscopy. For UC, the Mayo Endoscopic Score and for CD, the Simple Endoscopic Score [SES-CD] are two of the most prominent and widely utilized scores in clinical practice. In addition to colonoscopy findings such as erythema, ulcerations, and bleeding, the Mayo

score is based on the patient's clinical history of stool frequency, rectal bleeding, and general assessment by the clinician [2], while SES-CD is based mostly on ileocolonoscopy findings, such as the number and size of ulcers, as well as the presence and kind of narrowing throughout the colon [3].

The health and financial impact of IBD grows as the disease's incidence rises. The importance of IBD biomarkers is heightened as a result of these alterations. Because colonoscopy is an invasive, costly, and time-consuming patient procedure, it is vital to develop easy, cheap, and non-invasive IBD diagnostics [4]. The serum markers of acute phase response, CRP and ESR, have been explored as IBD biomarkers. They are, however, not limited to IBD, since they are raised in a range of settings such as infections, autoimmune disorders, and cancer [5]. Several studies have looked into serum calprotectin as a possible marker for diagnosis of IBD [6].

Calprotectin is made up of two tiny anionic proteins that bind calcium and zinc. Its expression has been found in immune cells such as macrophages, granulocytes, and monocytes during the early stages of differentiation. Calprotectin release weakens cell-cell contacts, causing endothelial permeability to be altered, resulting in leukocyte extravasation [7,8].

THE AIM OF THE WORK

Our study was prompted by the need for a non-invasive and accurate serum biomarker for IBD diagnosis, activity monitoring and following response to therapy in Egyptian population aiming at introducing serum calprotectin in routine laboratory workup for those patients in the near future.

PATIENTS AND METHODS

Fifty patients with IBD from Ain Shams University Hospitals' Gastroenterology clinic with ages ranging from 15 to 62 years and 20 apparently healthy subjects comparable in age and sex as a control group were included in this case control study that was conducted in the period between January 2020 and October 2021.

Ethical considerations: All subjects gave a

written informed consent before participation and the study was carried out after approval of the Ethical Committee of Ain Shams University [REC FWA 00017585] MS 488/2019. The study protocol conforms to the ethical guidelines of the 16. 1975 Declaration of Helsinki.

Patients with diabetes and obesity were excluded from the study. IBD was diagnosed based on conventional clinical data, which was validated by colonoscopy and biopsy. The Crohn's disease activity index [CDAI] was used to determine the degree of disease activity in the CD group, with a CDAI of less than 150 suggesting remission and greater than or equal to 150 indicating active illness. The modified Truelove and Witts index [TLW] was employed in the UC group, with remission defined as less than or equal 3 and activity defined as ≥ 4 [9,10].

All of the individuals in the study had a complete medical history taken and a comprehensive clinical assessment. Complete blood count [CBC] was performed using a Coulter counter, ESR was measured using the Westergren method, CRP, serum albumin levels, and serum creatinine levels were all assessed using a Beckman Coulter AU480 autoanalyzer. A quantitative commercially available ELISA kit was used to quantify serum calprotectin [Human Calprotectin ELISA kit, Bioassay Technology laboratory co. cat# E4010Hu, Shanghai, China].

For the measurement of serum calprotectin; all subjects' venous blood samples were collected on serum separator tubes under strict aseptic conditions using sterile plastic syringes. After allowing the samples to clot for 30 minutes, they were centrifuged at 3000 rpm for 15 minutes, and the serum was removed and kept at -20°C . Serum calprotectin was measured using a sandwich ELISA as specified by the manufacturer instructions measuring optical density at 450 nm.

Statistical analysis and interpretation:

Data was gathered, edited, coded, and entered into IBM SPSS version 23 [Statistical Package for Social Science] [Armonk, NY]. The quantitative data were presented as mean, standard deviations and ranges when their distribution was found parametric and median

with inter-quartile range [IQR] when their distribution was found non-parametric. Also, qualitative variables were presented as number and percentages. The P value was considered significant as follows: P value > 0.05: Non-significant, P value < 0.05: Significant.

RESULTS

According to descriptive statistics of the patient group's demographic data, 33 [66%] of the 50 patients were females and 17 [34%] were males, resulting in a female to male ratio of 1.9:1. They ranged in age from 15 to 62 years old, with a mean of 32.68 ± 10.41 . Thirty patients [60%] were diagnosed with UC, whereas 20 patients [40%] were identified as CD [Table 1].

In terms of serum calprotectin levels, there was a statistically significant difference between patients with IBD and the control group, with the patients' group demonstrating a statistically significant rise above the control group [P = 0.000] [Table 2].

At a cutoff point of 48 ng/ml, serum calprotectin could differentiate between patients with IBD and healthy controls with sensitivity of 98% and specificity of 95% with area under the curve [AUC] of 0.991. At a cutoff point of 4.1 mg/L, CRP could differentiate between patients with IBD and healthy controls with sensitivity of 64% and specificity of 85% with AUC of 0.758. However, at a cutoff point of 13 mm/hr ESR level could differentiate between patients with IBD and healthy controls with sensitivity of 62% and specificity of 85% with AUC of 0.703 [Figure 1].

Comparison between UC group and CD group revealed no significant change in serum calprotectin, ESR, CRP, hemoglobin levels, platelet count, serum creatinine or serum albumin [P > 0.05] [Table 3].

When clinical and laboratory data from patients with CD in clinical activity and those in remission were examined, it was shown that patients in clinical activity had higher ESR, CRP and serum calprotectin values than those in remission with a P value < 0.05 and had lower albumin and hemoglobin levels [P value < 0.01 and < 0.05 respectively]. In addition, there was a statistically significant difference in terms of

SES-CD between both groups. No significant difference in platelet count and serum creatinine levels between the two groups was observed [Table 4].

At a cutoff point of 120 ng/ml, serum calprotectin could differentiate between active cases of CD and in-remission cases with sensitivity of 90% and specificity of 90% with AUC of 0.970. At a cutoff point of 6.7 mg/L, CRP could differentiate between the two groups with sensitivity of 80% and specificity of 80% with AUC of 0.895. At a cutoff point of 20 mm/hr, ESR could differentiate between the two groups with sensitivity of 70% and specificity of 80% with AUC of 0.770 [Figure 2].

When clinical and laboratory data from patients with UC in clinical activity and those in remission were analyzed, it was shown that patients in clinical activity had higher ESR, CRP, platelet count and serum calprotectin values than those in remission, with a P value < 0.05 and had significantly lower albumin and hemoglobin levels [P value < 0.01 and P value < 0.05 respectively]. No significant difference was found in serum creatinine levels or Mayo score between the two groups with a P value of 0.79 and 0.07 respectively [Table 5].

At a cutoff point of 104 ng/ml, serum calprotectin could differentiate between active cases of UC and in remission cases of UC with sensitivity of 73.33% and specificity of 93.33% with AUC of 0.884. At a cutoff point of 5.8 mg/L, CRP could differentiate between the two groups with sensitivity of 73.33% and specificity of 93.33% with AUC of 0.820. However at a cutoff point of 18 mm/hr, ESR could differentiate between the two groups with sensitivity of 66.67% and specificity of 86.67% with AUC of 0.711 [Figure 3]

Our results revealed no significant statistical correlation between serum calprotectin levels and age, serum creatinine with P value > 0.05. However, there were statistically significant correlation between serum calprotectin and ESR, CRP, hemoglobin level, platelet count, serum albumin, SES-CD, TLW and CDAI with P value < 0.01. An almost significant statistical correlation was observed between serum calprotectin levels and Mayo score [P = 0.057] [Table 6].

Table [1]: Demographic data for the study patients

Patients group [No. = 50]		
Gender	Females	33 [66.0%]
	Males	17 [34.0%]
Age [years]	Mean \pm SD	32.68 \pm 10.41
	Range	15 – 62
Inflammatory Bowel Disease	Ulcerative colitis	30 [60.0%]
	Crohn's disease	20 [40.0%]

Table [2]: Comparison between control and patients' groups regarding serum calprotectin levels

S. Calprotectin [ng/ml]	Control group	Patients group	Test value	P value
	No. = 20	No. = 50		
Median [IQR]	20 [19 – 25]	100 [76 – 156]	-6.391 \neq	0.000
Range	16 – 68	44 – 296		

Table [3]: Comparison between UC group and CD group regarding laboratory data

		IBD		Test value	P value
		UC	CD		
		No. = 30	No. = 20		
ESR [mm/hr]	Median [IQR]	15 [10 – 30]	20 [10 – 30.0]	-1.207 \neq	0.227
	Range	5 – 90	9 – 90		
CRP [mg/L]	Median [IQR]	4.25 [2.4 – 12]	7.15 [4.5 – 23]	-1.575 \neq	0.115
	Range	0.8 – 57	0.7 – 76		
Haemoglobin [g/dl]	Mean \pm SD	11.89 \pm 1.18	11.42 \pm 1.54	1.212 \bullet	0.232
	Range	9 – 14.5	6.7 – 13.5		
Platelet count [x10³/μL]	Mean \pm SD	255.20 \pm 114.10	246.45 \pm 95.18	0.283 \bullet	0.778
	Range	138 – 594	157 – 517		
Serum Creatinine [mg/dl]	Mean \pm SD	0.93 \pm 0.13	0.96 \pm 0.24	-0.543 \bullet	0.590
	Range	0.7 – 1.2	0.6 – 1.2		
Serum Albumin [g/dl]	Mean \pm SD	3.41 \pm 0.80	3.42 \pm 0.85	-0.057 \bullet	0.955
	Range	2.2 – 5.2	2.4 – 5.1		
Serum Calprotectin [ng/ml]	Median [IQR]	86 [76 – 156]	122 [78 – 156]	-0.556 \neq	0.579
	Range	52 – 276	44 – 296		

P value > 0.05: Non significant; P value < 0.05: Significant; \bullet : Chi-square test; \bullet : Independent t-test; \neq : Mann-Whitney test

Table [4]: Comparison between CD patients in remission and those in activity regarding clinical and laboratory data

		CD cases		Test value	P value
		In remission	In activity		
		No. = 10	No. = 10		
ESR [mm/hr]	Median [IQR]	17.5 [10 – 20]	30.0 [15 – 40]	-2.064 \neq	0.039
	Range	9 – 30	10 – 90		
CRP [mg/L]	Median [IQR]	4.5 [2.3 – 6.7]	23 [7.6 – 30]	-2.989 \neq	0.003
	Range	0.7 – 20	5 – 76		
Haemoglobin [g/dl]	Mean \pm SD	12.09 \pm 1.17	10.75 \pm 1.63	2.111 \bullet	0.049
	Range	10 – 13.5	6.7 – 12.2		
Platelet count [x10³/μL]	Mean \pm SD	214.30 \pm 50.76	278.60 \pm 119.39	-1.567 \bullet	0.134
	Range	163 – 300	157 – 517		
Serum Creatinine [mg/dl]	Mean \pm SD	1.02 \pm 0.20	0.89 \pm 0.26	1.255 \bullet	0.225
	Range	0.6 – 1.2	0.6 – 1.2		
Serum Albumin [g/dl]	Mean \pm SD	4.15 \pm 0.54	2.69 \pm 0.20	7.952 \bullet	0.000
	Range	3 – 5.1	2.4 – 3		
SES-CD score	Median [IQR]	2.5 [1 – 3]	8 [7 – 12]	-3.051 \neq	0.002
	Range	0 – 7	2 – 23		
Serum Calprotectin [ng/ml]	Median [IQR]	78 [60 – 100]	156 [130 – 180]	-3.558 \neq	0.000
	Range	44 – 124	100 – 296		

P value > 0.05: Non significant; P value < 0.05: Significant; \bullet : Independent t-test; \neq : Mann-Whitney test SES-CD score: Simple Endoscopic Score for CD

Table [5]: Comparison between UC patients in remission and those in clinical activity regarding clinical and laboratory data

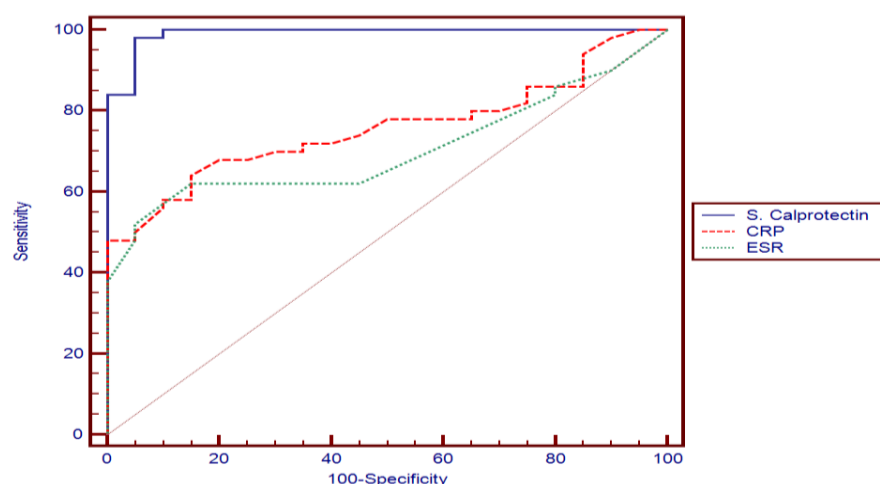
		UC cases		Test value	P value
		In remission No. = 15	In activity No. = 15		
ESR [mm/hr]	Median [IQR] Range	17.5 [10 – 20] 9 – 30	30.0 [15 – 40] 10 – 90	-2.064 \neq	0.039
CRP [mg/L]	Median [IQR] Range	4.5 [2.3 – 6.7] 0.7 – 20	23 [7.6 – 30] 5 – 76	-2.989 \neq	0.003
Haemoglobin [g/dl]	Mean \pm SD Range	12.09 \pm 1.17 10 – 13.5	10.75 \pm 1.63 6.7 – 12.2	2.111 \bullet	0.049
Platelet count [$\times 10^3/\mu\text{L}$]	Mean \pm SD Range	214.30 \pm 50.76 163 – 300	278.60 \pm 119.39 157 – 517	-1.567 \bullet	0.134
Serum Creatinine [mg/dl]	Mean \pm SD Range	1.02 \pm 0.20 0.6 – 1.2	0.89 \pm 0.26 0.6 – 1.2	1.255 \bullet	0.225
Serum Albumin [g/dl]	Mean \pm SD Range	4.15 \pm 0.54 3 – 5.1	2.69 \pm 0.20 2.4 – 3	7.952 \bullet	0.000
SES-CD score	Median [IQR] Range	2.5 [1 – 3] 0 – 7	8 [7 – 12] 2 – 23	-3.051 \neq	0.002
Serum Calprotectin [ng/ml]	Median [IQR] Range	78 [60 – 100] 44 – 124	156 [130 – 180] 100 – 296	-3.558 \neq	0.000

P value > 0.05: Non significant; P value < 0.05: Significant; \bullet : Independent t-test; \neq : Mann-Whitney test

Table [6]: Correlation between serum calprotectin and other parameters under study

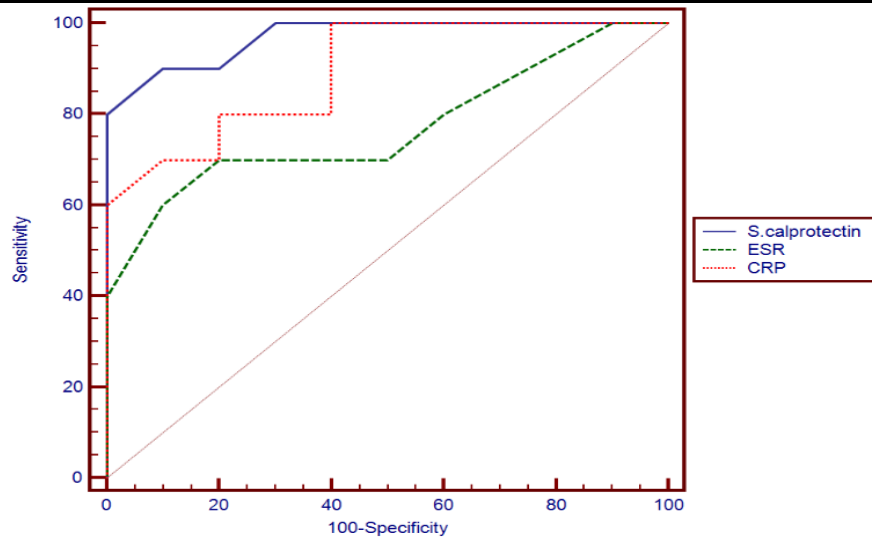
	Serum Calprotectin [ng/ml]	
	r	P value
Age [years]	-0.033	0.818
ESR mm/hr	0.761**	0.000
CRP mg/L	0.804**	0.000
Hemoglobin [g/dl]	-0.472**	0.001
Platelet count [$\times 10^3/\mu\text{L}$]	0.606**	0.000
Serum Creatinine [mg/dl]	-0.082	0.573
Serum Albumin [g/dl]	-0.667**	0.000
Mayo Score	0.351	0.057
SESCD score	0.622**	0.003
TLW for UC	0.723**	0.000
CDAI for CD	0.880**	0.000

** : Significant at P value < 0.05; TLW: The modified Truelove and Witts index; CDAI: Crohn's disease activity index



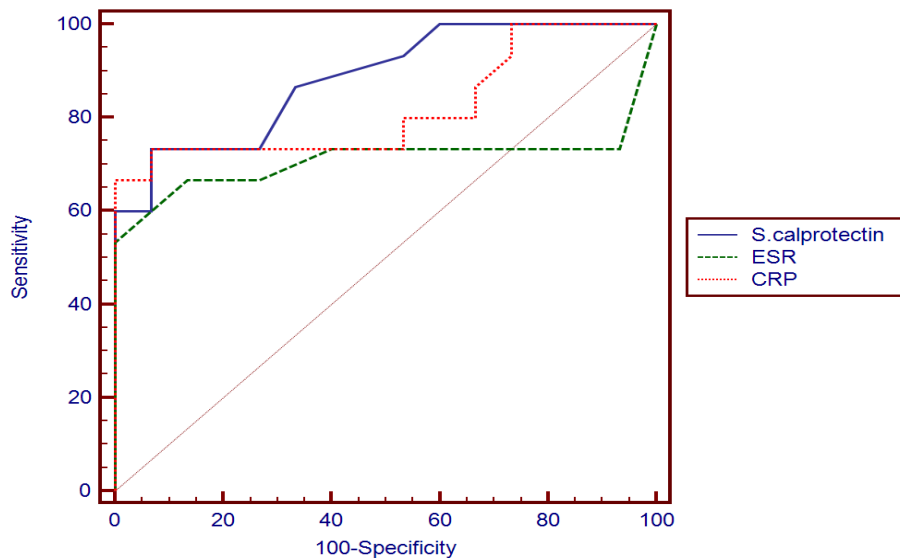
Parameters	Cut off point	AUC	Sensitivity	Specificity	+PV	-PV
Serum Calprotectin [ng/ml]	>48	0.991	98.0%	95.0%	98.0%	95.0%
CRP [mg/L]	> 4.1	0.758	64.0%	85.0%	91.4%	48.6%
ESR [mm/hr]	> 13	0.703	62.0%	85.0%	91.2%	47.2%

Figure [1]: Receiver operating characteristic curve [ROC] for serum calprotectin levels, CRP levels and ESR levels to differentiate between patients and controls



Parameters	Cut off point	AUC	Sensitivity	Specificity	+PV	-PV
Serum Calprotectin [ng/ml]	> 120	0.970	90.0%	90.0%	90.0%	90.0%
CRP [mg/L]	> 6.7	0.895	80.0%	80.0%	80.0%	80.0%
ESR [mm/hr]	> 20	0.770	70.0%	80.0%	77.8%	72.7%

Figure [2]: Receiver operating characteristic curve [ROC] for serum calprotectin, CRP and ESR to differentiate between active cases and in remission cases in CD group



Parameters	Cut off point	AUC	Sensitivity	Specificity	+PV	-PV
Serum Calprotectin [ng/ml]	> 104	0.884	73.33%	93.33%	91.7%	77.8%
CRP [mg/L]	> 5.8	0.820	73.33%	93.33%	91.7%	77.8%
ESR [mm/hr]	> 18	0.711	66.67%	86.67%	83.3%	72.2%

Figure (3): Receiver operating characteristic curve (ROC) for serum calprotectin, CRP and ESR to differentiate between active cases and in remission cases in UC group

DISCUSSION

Inflammatory bowel disease [IBD], namely UC and CD, are chronic inflammatory illnesses that go through phases of aggravation and remission [11]. Patients' quality of life and mental health are much impacted by recurrent IBD, and measuring disease activity can help guide treatment and estimate prognosis [12].

Despite the fact that colonoscopy is the best approach for diagnosing IBD, it is intrusive and

costly, hence an urgent demand for non-invasive, accepted, and accurate blood-based biomarkers has emerged [4]. CRP and ESR, two serum indicators of acute phase response, are too little sensitive and specific for intestinal inflammation since their levels are frequently elevated in infections, autoimmune disorders, and other conditions [13]. Serum calprotectin has been investigated as an acute phase reactant increasing with IBD exacerbations [11].

Calprotectin is mainly found in neutrophil

granules and has antimicrobial activity ^[14]. Calprotectin expression is up-regulated in neutrophils after induction by lipopolysaccharides or chemokines, and it is translocated to the extracellular fluid. It then initiates a many biological processes such as signal transduction, cell homeostasis, and inflammation. It is also involved in the activation of phagocyte NADPH oxidase. Serum calprotectin has been poorly studied, but recent literature suggests a potential role as an inflammatory biomarker in IBD ^[16].

In our study, serum calprotectin levels in patients with IBD [UC and CD groups] were considerably higher than in the control group [$P < 0.01$], with a sensitivity and specificity of 98% and 95%, respectively. This is consistent with the findings of Leach *et al.* ^[15] and Okada *et al.* ^[16], who also found elevated serum calprotectin levels in patients with IBD.

CRP and ESR levels were also shown to differ statistically between IBD patients and control groups. CRP had sensitivity and specificity of 64% and 85%, respectively, whilst ESR had sensitivity and specificity of 62% and 85%, demonstrating that serum calprotectin has a stronger diagnostic value for IBD than CRP and ESR. This is in line with the findings of Kalla *et al.* ^[17] who studied serum calprotectin levels in 156 individuals and reported that in comparison to other biomarkers such as CRP and serum albumin, serum calprotectin was the best determinant for IBD diagnosis and may signal need for surgery in IBD patients, particularly those with CD. Furthermore, Okada *et al.* ^[16] claimed that CRP was found to be a less sensitive and specific biomarker for IBD than serum calprotectin and found no correlation between serum calprotectin and serum albumin, WBC count, or platelet count.

In our investigation, there was no significant change in serum calprotectin, ESR, CRP and other laboratory markers assessed between the CD and UC groups, which indicates that serum calprotectin, as a biomarker, was not able to distinguish patients with UC from those with CD. Failure of localization of the disease in patients with IBD may be considered a limitation of the present study. These findings were in contrast to the results reported by Okada *et al.* ^[16] and Elshayeb *et al.* ^[18] who demonstrated higher serum calprotectin levels in patients with

CD than in patients with UC. The discrepancies in outcomes could be explained by ethnic disparities and/or sample sizes.

In the CD group, the difference in serum calprotectin levels, ESR, CRP, hemoglobin, albumin levels, and SES-CD score between the active and in remission subjects was statistically significant. While in the UC group, the difference in serum calprotectin levels, ESR, CRP, hemoglobin, albumin levels, and platelet count between active and in remission cases was statistically significant.

The sensitivity and specificity of serum calprotectin to differentiate between the active cases and in remission cases in the CD group were 90% and 90% while for CRP they were 80% and 80% and for ESR were 70% and 80% respectively denoting that serum calprotectin had higher diagnostic characteristics in judging the activity of CD.

The sensitivity and specificity of serum calprotectin to predict disease activity among the UC group were 73.33% and 93.33% while for CRP they were 73.33% and 93.33%; and for ESR were 66.67% and 86.67% respectively. The diagnostic characteristics of serum calprotectin were similar to that of CRP but higher than ESR in judging the activity of UC.

Serum calprotectin levels, ESR and CRP were higher in individuals with active IBD both in UC and CD groups than those in remission. This was consistent with studies by Alper *et al.* ^[19] and Megeed *et al.* ^[20], who associated ESR and CRP with IBD diagnosis and clinical, endoscopic and radiographic disease activity across time. This was also agreed upon on by Solem *et al.* ^[21]. This is also in line with the findings of Leach *et al.* ^[15], who investigated serum calprotectin in a juvenile IBD population and found it to be substantially linked with disease activity.

In another study, Meuwis *et al.* ^[22] examined the role of serum calprotectin as a marker for CD in 115 patients, finding that CD patients had higher serum calprotectin levels than healthy controls. Serum calprotectin levels in active disease were substantially higher than in inactive disease. They came to the conclusion that serum calprotectin, coupled with fecal calprotectin and CRP, might be utilized to predict recurrence after infliximab discontinuation.

Our findings were similar to those of Malham *et al.* [23], who demonstrated that higher serum calprotectin levels were linked to a more severe inflammatory state in the colon as well as significant clinical symptoms [i.e., active disease] in UC patients. Furthermore, Chen *et al.* [12] compared serum calprotectin, CRP, and ESR levels to IBD activity and discovered that serum calprotectin levels had greater sensitivity and specificity than CRP and ESR in predicting IBD activity.

On the other hand, McCann *et al.* [24] studied the performance of serum calprotectin versus fecal calprotectin and CRP in a group of 109 people with gastrointestinal disorders. They discovered no substantial correlation between serum calprotectin, fecal calprotectin, and CRP. As a result, they came to the conclusion that serum calprotectin is unlikely to be effective as a biomarker for intestinal inflammation.

Our work revealed a positive correlation between serum calprotectin and ESR, CRP and platelet count in patients with IBD; while it had an inverse relation with serum albumin and hemoglobin levels. Similar results were also reported by Leach *et al.* [15] and Meuwis *et al.* [22]. It is believed that because of the malabsorption of nutrients, serum albumin level decreases in proportion to the IBD activity. Platelet count is increased as an acute phase reactant while hemoglobin levels are decreased due to iron deficiency anemia caused by repeated bleeding from the digestive tract especially during flares of the disease or due to anemia of chronic disease. Anemia is a significant complication of IBD [25].

In the present study there was a significant correlation between serum calprotectin and SES-CD and CDAI in patients with CD, together with statistically significant correlation between serum calprotectin and TLW in patients with UC. The correlation of serum calprotectin with Mayo score in patients with UC was nearly significant denoting that serum calprotectin was correlated with both clinical and endoscopic features of disease activity of IBD.

Study's limitations: Sample size of this study was relatively small which may have an impact on the generalization of findings so authors recommend the following: a) An

extended research on larger number of IBD patients for better evaluation of the relationship between serum calprotectin and IBD clinical activity, studying the best cutoff values in clinical practice for monitoring intestinal inflammation and assessing the utility of serum calprotectin in anticipating IBD relapse and evaluating efficacy of medical therapy, b) Study of serum calprotectin levels together with fecal calprotectin for better evaluation of sensitivity and specificity of serum calprotectin, c) Study of serum calprotectin levels in other inflammatory diseases for better evaluation of its specificity. d) Further studies on how to decrease serum calprotectin for better control of IBD exacerbations.

Conclusion: Serum calprotectin levels are higher in patients with IBD and are related to clinical activity. The association between serum calprotectin, CRP, and ESR was found to be statistically significant. Because of its better sensitivity and specificity, serum calprotectin has a higher diagnostic value for IBD than CRP and ESR. According to our findings, high serum calprotectin levels can distinguish between patients who are clinically active and those who are in remission in both UC and CD groups. Our findings show that serum calprotectin may be a promising simple diagnostic and prognostic biomarker for IBD however more research is needed to reach a judgment in this regard.

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