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ORIGINAL ARTICLE

The role of Serum Visfatin as Diagnostic Marker of Active Inflammatory Bowel Disease

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Abstract

Background: ***The mucosal immune system's degradation of the gastrointestinal tract mucosa is the underlying cause of inflammatory bowel diseases (IBDs).***

Aim of the Work: ***The goals of this research are to ascertain visfatin serum concentrations in newly diagnosed IBD patients and assess visfatin's diagnostic value alongside disease activity and colonoscopic findings.***

Patients and Methods: ***This case-control research was conducted at the outpatient clinic of Alexandria Fever Hospital. 90 subjects were included and categorized into three groups: 30 patients newly diagnosed with Crohn's disease,30 cases newly diagnosed with ulcerative colitis, and 30 case-control individuals matched for age and sex. All groups were evaluated by clinical findings, gastrointestinal endoscopy with histopathology, and laboratory investigations, including serum visfatin and inflammatory markers.***

Results: ***This research demonstrated that there was an incredibly significant variance among Crohn's disease, Ulcerative colitis, and control as regards serum visfatin, fecal calprotectin, ESR, and CRP. The sensitivity and specificity of serum visfatin at a cutoff point ≥2.25 were 76.7% and 86.7%, respectively, for the detection of Crohn's disease with 0.891 AUC, p<0.001. The specificity and sensitivity of serum visfatin at a cutoff point ≥2.25 were 83.3% and 86.7%, respectively, for the detection of UC with 0.919 AUC, p<0.001.***

Conclusion: ***This research discovered that elevated visfatin levels were associated with newly diagnosed active colonic inflammatory disorder. Serum visfatin is one potential new indicator for predicting activity and severity in inflammatory bowel disease patients.***

Keywords: Active Crohn's; Active Ulcerative colitis; SerumVisfatin

1. Introduction

The immune system-induced destruction of GIT mucosa[1] characterizes Inflammatory Bowel diseases. The origins and development of IBDs are complex, involving a combination of factors such as genetic predisposition, disturbed gut microbiota composition, and immune system activation .[2] A colonoscopy is used to diagnose IBD initially, distinguish CD from UC, and evaluate the severity and activity of the disease. However, it is an intrusive operation that necessitates intestinal preparation. Also, many patients find it painful and inconvenient. Erythrocyte sedimentation rate (ESR), fecal calprotectin (FC), and C-reactive protein (CRP) are examples of nonspecific biomarkers that are frequently employed as noninvasive indicators to diagnose IBD but lack specificity or sensitivity for disease progression. [3]

Visfatin, an adipokine originating from visceral adipose tissue, has been identified at elevated levels in the systemic circulation of cases with different diseases .[4] IBD is characterized by inflammation of the mesenteric adipose tissue proximal to the inflamed bowel, which alters the concentrations of adipokines locally or in the blood. Visfatin was found to increase the epithelial expression of tumor necrosis factor-alpha (TNF-α), interleukin (IL)-l, IL-6, and adhesion molecules; this could be a noninvasive, easily measurable possible marker to diagnose and monitor disease activity and severity. [5]

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So, we aimed to determine the serum concentration of visfatin in newly diagnosed IBD patients, evaluate its role in the diagnosis of inflammatory bowel disease, and correlate it with illness activity and colonoscopic findings.

2. Patients and methods

Characteristics of Participants

This case-control study enrolled 60 participants with Inflammatory Bowel Disease from the Gastroenterology Outpatient Clinic at Alexandria Fever Hospital. The study lasted one year, from May 2022 to April 2023. The participants were categorized into two groups: 30 with Crohn's disease and 30 with Ulcerative Colitis. Additionally, a control group consisting of 30 case-control individuals, matched for age and gender, was included.

Inclusion and exclusion Criteria:

This study targeted Patients (aged more than 18 years old) with IBD who were newly diagnosed by clinical findings and lower gastrointestinal endoscopy with histopathological validation. Case-control patients who have GIT symptoms and are indicated for colonoscopy and IBD are excluded, with no other systemic disease according to exclusion criteria. Those who had already initiated treatment, individuals with diabetes, overweight participants, those with metabolic syndrome, or pregnant women were excluded from the research.

The study protocol received approval from the National Liver Institute's ethical committee, and all subjects provided informed consent.

Laboratory Investigations

After a detailed medical history, complete clinical examination, and laboratory examination, stool samples were collected for measuring fecal calprotectin (FC), extracted by using the BIOHIT extraction tubes and extraction buffer before measurement by BIOHIT calprotectin enzyme-linked immunosorbent assay(ELISA)technique. Serum samples were stored at -20°C until visfatin measurement was achieved. Serum visfatin level was measured by ELISA using the Elabscience Human VF (Visfatin) ELISA Kit as per the manufacturer's instructions. All patients also went for other laboratory investigations: hemoglobin, CRP, and ESR.

Endoscopic Examination

Ileocolonoscopic examination with biopsies from suspected lesions in the colon and terminal ileum for histopathological confirmation was achieved. Severity was determined according to the Montreal classification for both Crohn's disease and Ulcerative colitis.

Histopathologic Examination

Table 1. The histopathological features that differentiate between UC and CD.

TYPICAL FEATURES	ULCERATIVE COLITIS	CROHN'S DISEASE
LOCALIZATION OF INFLAMMATION	Limited to the mucosa, sometimes submucosa	Transmural
NON-ACTIVE INFLAMMATION	Diffuse(continuous)	Focal(discontinuous), with skip lesions
LYMPHOID AGGREGATES	Frequent in mucosa, submusosa	Common, transmural
GRANULOMAS	Absent, except cryptitis	Common, transmural
ACTIVE INFLAMMATION (CRYPTITIS, CRYPT ABSCESSES)	Diffuse(continuous)	Focal(discontinuous)
ULCERS	Superficial	Deep, fissure-like, aphtous
FISTULAE	Absent except in fulminant UC	Common
INFLAMMATORY PSEUDOPOLYPS	Common	Rare
CRYPT ARCHITECTURAL IRREGULARITY	Diffuse(continuous), marked	Focal(discontinuous)
ATROPHY	Present, pronounced	Uncommon, mild
PANETH CELL METAPLASIA	Present	Uncommon
PYLORIC GLAND METAPLASIA	Rare	Present
NEURONAL/MUSCULAR HYPERTROPHY	Rare/absent	Common/present
SEROSITIS	Absent except in fulminant UC	Present
WALL THICKNESS	Normal	Increased
STRICTURES	Uncommon	Common
FAT WRAPPING	Absent	Common

Statistical Analysis

Data collection involved inputting information into the computer using the Statistical Package for Social Science (SPSS) program for subsequent statistical analysis (version 13; Inc., Chicago, IL). Mean, standard deviation (SD), and range were used to display quantitative data, while frequency and percentage were used to express qualitative data. Statistical significance was determined by a P-value less than 0.05, and a highly significant difference was acknowledged if $p \leq 0.001$.

3. Results

Among control group findings, there are 17 cases with nonspecific ileitis, 8 cases with non-specific colitis (including two cases of hyperplastic inflammatory polyps), and 5 cases of normal colonoscopic study.

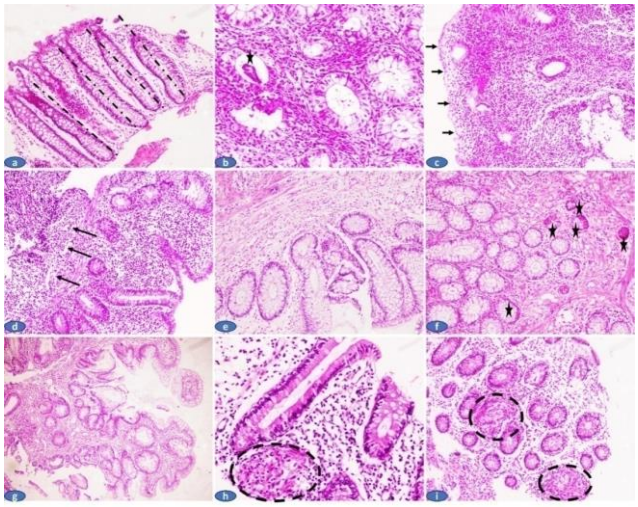

Figure 1. The histopathological findings of the studied cases: a) A case of non-specific colitis showed preserved crypt architecture (test tube-like) (interrupted lines) (H&E 100X), b) A case of UC showed cryptitis and crypt abscess (asterisk) (H&E 200x),c) A case of UC showed surface erosion/ulceration (arrows) and exhausted reactive colonic glands (H&E 100x), d) A case of IBD showed marked inflammation dissecting and infiltrating muscularis mucosa to the submucosa (arrows) (H&E 100x), e) A case of IBD showed shortening and irregularity of crypts (H&E 100x), f) A case of UC showed Paneth cell metaplasia (asterisks) (H&E 100x), g) A case of UC showed pseudo-polyp formation (H&E 40x), h) A case of Crohn's disease showed mucosal small non-caseating granuloma formation (circle) (H&E 100x), i) A case of Crohn's disease showed numerous mucosal small non-caseating granuloma formations (circles) (H&E 100x).

Table 1. Distribution of the studied cases according to demographic data

		N	%
STUDIED GROUPS	Crohn's disease	30	33.3
	Ulcerative colitis	30	33.3
	Control group	30	33.3
CROHN'S DISEASE (N=30)			
CROHN'S DISEASE AGE OF DIAGNOSIS	A2	10	33.3
	A3	20	66.7
CROHN'S DISEASE LOCATION	L1	20	66.7
	L3	10	33.3
CROHN'S DISEASE BEHAVIOR	B1	30	100
SES-CD*	min-max	6-36	
	mean ± SD	13.4 ± 9.4	
	median (IQR)	9 (7-18.75)	
ULCERATIVE COLITIS (N=30)			
ULCERATIVE COLITIS SEVERITY	S1	9	30.0
	S2	12	40.0
	S3	9	30.0
ULCERATIVE COLITIS EXTENT	E1	12	40.0
	E2	10	33.3
	E3	8	26.7

Among Crohn's patients, 66.7 % had a disease location of L1, 33.3% had L3 and 30% had S3. All cases are B1 for disease behavior. Among UC patients 30 % had the severity of S1, 40% had S2 and 30% had S3 and according to extension 40% had E1,33.3% had E2 and 26.7% had E3.

Table 3. Comparison between studied cases according to hemoglobin and inflammatory markers

		CD (N=30)	UC (N=30)	CONTROL GROUP (N=30)	TEST OF SIGNIFICANCE (P-VALUE)
HB	min-max	8.7-14.5	9.7-14.6	11.9-15.2	ANOVA = 21.541 (<0.001)**
	mean ± SD	11.5 ± 1.55	11.75 ± 1.32	13.55 ± 1.04	
		p1=0.756, p2<0.001**,		p3<0.001**	
ESR	min-max	12.8-89.7	9.9-86.8	4.2-18.6	ANOVA = 43.209 (<0.001)**
	mean ± SD	54.33 ± 22.03	47.32 ±	11.53 ± 4.55	
		p1=0.336, p2<0.001**,		p3<0.001**	
CRP	min-max	1.4-95.8	1.3-96.5	0.8-4.3	ANOVA = 15..3 (<0.001)**
	mean ± SD	32.6 ± 26.83	32.31 ± 32.2	1.78 ± 1.09	
		p1=0.999, p2<0.001**,		p3<0.001**	

SD: Standard deviation

**: p≤0.01 is a highly statistically significant

p1: p-value for comparing CD and UC groups.

p2: p-value for comparing between CD and control groups.

p3: p-value for comparing between UC and control groups.

As regards Hb, there was a statistically significant variance among CD, UC, & control groups (p<0.001). The Post Hoc test revealed that both the CD and Ulcerative colitis group had a significantly lower Hb level than the control group ;(p2<0.001) and (p3<0.001). For both ESR and CRP, there was a statistically significant variance among CD, UC, & control groups (p<0.001). Table 3.

Table 4. Comparison between UC severity and extent(Montreal score) according to fecal calprotectin and serum visfatin.

UC SEVERITY		S1 (N=9)	S2 (N=12)	S3 (N=9)	TEST OF SIGNIFICANCE (P-VALUE)
Fecal calprotectin (mg/kg)	min-max	50-1436	106-2896	165-1762	K = 6.379 (0.041)*
	mean ± SD	383.67 ± 495.33	1026.83 ± 819.83	902.89 ± 665.67	
	median (IQR)	209 (51.5-670)	876.5 (342-1605.5)	725 (331-1733)	
		p1=0.017*,	p2=0.048*,	p3=0.791	

UC EXTENT	Serum visfatin (ng/ml)	min-max	0.7-4	1.5-6.7	2.6-10.4	K = 10.916 (0.004)**
		mean ± SD	2.26 ± 0.97	4.27 ± 1.64	5.51 ± 2.57	
		median (IQR)	2.4 (1.6-2.9)	3.75 (3.1-5.7)	5.4 (3.3-7.25)	
			p1 =0.014*, p2 =0.001**,		p3 =0.354	
			E1 (n=12)	E2 (n=10)	E3 (n=8)	
	Fecal calprotectin (mg/kg)	min-max	50-2896	165-1896	246-1741	K = 3.216 (0.2)
		mean ± SD	674.0 ± 889.55	872.4 ± 642.52	886.13 ± 567.59	
		median (IQR)	235.5 (52.25-1324.5)	745.5 (286.25-1582.75)	799.5 (434.25-1514)	
	Serum visfatin (ng/ml)	min-max	0.7-5.7	1.5-6.7	2.4-10.4	K = 7.645 (0.022)*
		mean ± SD	2.73 ± 1.34	4.31 ± 1.75	5.65 ± 2.66	
		median (IQR)	2.65 (1.63-3.6)	3.7 (3.2-5.93)	5.75 (3.18-7.5)	
			p1`=0.056, p2`=0.009**,		p3`=0.427	

SD: Standard deviation IQR: Interquartile range
K: Kruskal-Wallis H *: p≤0.05 is statistically significant
**: p≤0.01 is highly statistically significant
p1: p-value for comparing between S1 and S2
p2: p-value for comparing between S1 and S3
p3: p-value for comparing between S2 and S3
p1: p-value for comparing between E1 and E2
p2: p-value for comparing between E1 and E3
p3: p-value for comparing between E2 and E3
Montreal score
S: UC Severity
S1: Mild UC
S2: Moderate UC
S3: Sever UC
E: UC extent
E1: Ulcerative proctitis
E2: Left-sided(distal UC)
E3: Extensive UC (pancolitis)

Regarding both serum visfatin and fecal calprotectin in UC severity, there was a statistically significant variance among S1, S2, & S3 (p=0.004 and p=0.041 respectively). Concerning both markers, S1 had a significantly lower level than S2 and S3. As regards serum visfatin in UC extent, there was a statistically significant variance

among E1, E2, & E3 with (p=0.022). The post-hoc test revealed that the E1 serum visfatin mean(2.73 ± 1.34) was significantly lower than the E3 serum visfatin mean (5.65 ± 2.66) with (p2=0.009).Table 4 .

Table 2. Spearman correlation coefficient between Serum visfatin and different parameters of the studied participant groups.

	SERUM VISFATIN					
	CD (n=30)		UC (n=30)		Control (n=30)	
	r	P	r	P	r	P
HEMOGLOBIN	-0.112	0.554	-0.02	0.917	-0.247	0.118
ESR	0.248	0.187	0.183	0.334	-0.231	0.22
CRP	0.325	0.08	0.163	0.389	-0.1	0.601
FECAL CALPROTECTIN	0.638	<0.001**	0.789	<0.001**	0.14	0.461

r =Spearman correlation coefficient
**: p≤0.01 is highly statistically significant

Among the 30 CD cases, serum visfatin demonstrated a statistically significant positive association with fecal calprotectin (p<0.001). Among the 30 ulcerative colitis cases, serum visfatin demonstrated a statistically significant positive association with fecal calprotectin (p<0.001). Among the 30 control participants, serum visfatin showed no statistically significant correlation. Table 5.

Table 3. Performance of fecal calprotectin & serum visfatin in the diagnosis of inflammatory bowel diseases.

		AUC	SENSITIVITY	SPECIFICITY	PPV	NPV	ACCURACY	P
INFLAMMATORY BOWEL DISEASE	Fecal calprotectin cutoff ≥50.5 (mg/kg)	0.973 (0.946-0.999)	93.3%	80%	90.3%	85.7%	88.9%	< 0.001**
	Serum visfatin cutoff ≥2.25 (ng/ml)	0.905 (0.846-0.964)	80%	86.7%	92.3%	68.4%	82.2%	< 0.001**
CROHN`S DISEASE	Fecal calprotectin cutoff ≥55.5 (mg/kg)	0.973 (0.941-1.0)	90%	86.7%	87.1%	89.7%	88.3%	< 0.001**
	Serum visfatin cutoff ≥2.25	0.891 (0.813-	76.7%	86.7%	85.2%	78.8%	81.7%	< 0.001**

	(ng/ml)	0.969)						
ULCERATIVE COLITIS	Fecal calprotectin cutoff ≥50.5	0.973 (0.94-1.0)	96.7%	80%	93.5%	96.6%	95%	< 0.001**
	Serum visfatin cutoff ≥2.25	0.919 (0.851-0.986)	83.3%	86.7%	84%	74.3%	78.3%	< 0.001**

AUC: Area under the curve, PPV: Positive predictive value, NPV: Negative predictive value

**: p≤0.001 is highly statistically significant

Table 6 shows that the specificity & sensitivity of fecal calprotectin at a cutoff point ≥50.5 were 93.3% and 80% respectively for the detection of IBD with 0.973, AUC, p<0.001. The sensitivity and specificity of serum visfatin at a cutoff point ≥2.25 were 80% and 86.7% respectively for the detection of IBD with 0.905, AUC, p<0.001.Figure 2&Figure 3

Table 6 demonstrate that the specificity & sensitivity of fecal calprotectin at a cutoff point ≥55.5 were 90% and 86.7% respectively for the detection of CD with 0.973, AUC, p<0.001. The specificity & sensitivity of serum visfatin at a cutoff point ≥2.25 were 76.7% and 86.7% respectively for the detection of CD with 0.891, AUC, p<0.001.Figure 3

Table 6 illustrates that the sensitivity & specificity of fecal calprotectin at a cutoff point ≥50.5 were 96.7% and 80% respectively for the detection of UC with 0.973, AUC, p<0.001. The specificity & sensitivity of serum visfatin at a cutoff point ≥2.25 were 83.3% and 86.7% respectively for the detection of UC with 0.919, AUC, p<0.001.Figure 4

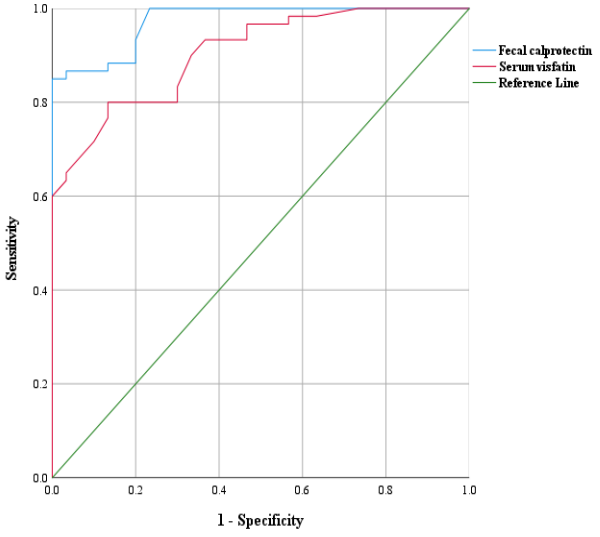

Figure 2. ROC curve of fecal calprotectin and serum visfatin in the diagnosis of inflammatory bowel diseases

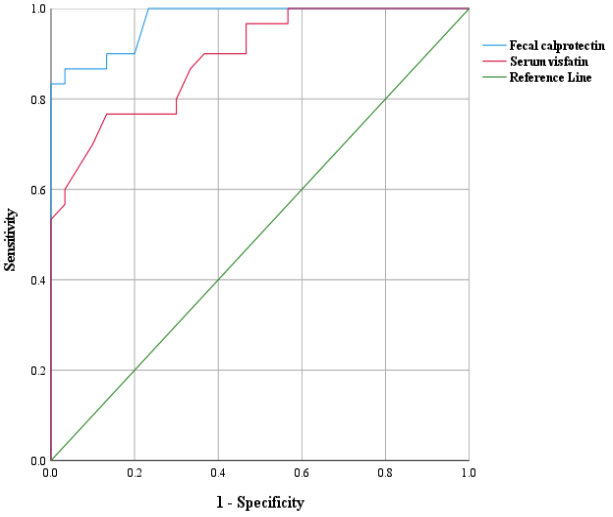

Figure 3. ROC curve of fecal calprotectin and serum visfatin in the diagnosis of CD

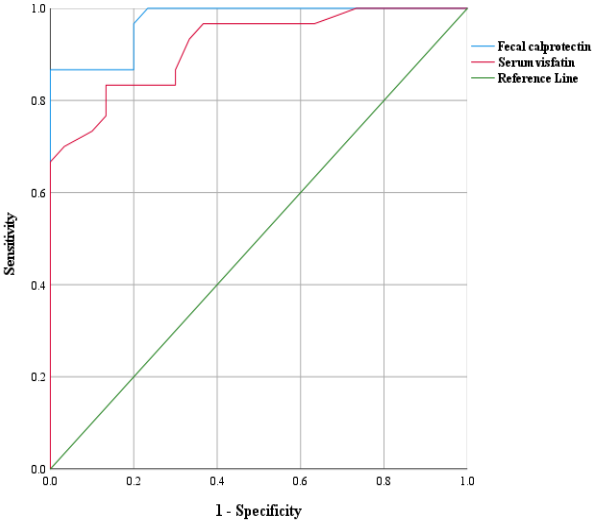

Figure 4. ROC curve of fecal calprotectin and serum visfatin in the diagnosis of UC

4. Discussion

In some circumstances, adipokines worsen inflammatory bowel disease by enhancing inflammation through the release of proinflammatory interleukins. Interleukin (IL)-l, tumor necrosis factor-alpha (TNF-alpha), IL-6, and adhesion molecules are examples of adipokines that promote epithelium expression. One such adipokine is visfatin. [6]

Many studies have shown that FC levels have been increased in many disease conditions involving colorectal adenocarcinoma, infection, uncontrolled coeliac disease, diverticulitis, and microscopic colitis. [7]

In the study of Saadoun et al.[8] a total of 85 newly diagnosed Inflammatory Bowel Disease cases were included, with 56 diagnosed with UC and 29 with CD. Additionally, 30 healthy controls were incorporated in the research. The phenotypes of IBD were categorized according to the Montreal classification. Various parameters, including hemoglobin, ESR, CRP, fecal calprotectin, and serum visfatin, were assessed. Consistent with the findings of our study, Saadoun et al.[8] stated that both groups of IBD patients exhibited significantly lower hemoglobin levels compared to the control group. Also, our study illustrated that IBD patients showed significantly higher ESR and CRP compared to the control group.

It has been demonstrated that cases with CD had higher serum visfatin levels compared to those with UC, and additional research has demonstrated an enhanced expression of visfatin genes in IBD patients in comparison to healthy people .[8] This research aims to determine the serum concentrations of visfatin in newly diagnosed IBD patients, evaluate its role in the diagnosis of IBD, and correlate it with disease activity and colonoscopic findings. In agreement with our results, Saadoun et al.[8] showed that Crohn's group had significantly greater visfatin levels than the control group. At the same time, there was not a significant variance in visfatin among Crohn's & UC groups. Saadoun et al.8 highlighted that according to the Montreal classification, serum visfatin levels were significantly greater in cases with Crohn's disease with stricture phenotype (B2). Our study failed to distinguish Crhon's disease location (according to Montreal: L1 and L3) using both visfatin and fecal calprotectin or one of them, as it demonstrated that there was no significant variance among CD Age (A2 & A3) concerning Serum visfatin and Fecal calprotectin.

Waluga et al.[9] investigated the concentrations of serum adipokines in cases with inflammatory bowel disease prior to and following therapy and clinical remission. Subjects with active CD had significantly greater baseline concentrations of visfatin (23.2 ± 3.2 ng/mL; P < 0.05) compared to the control group (14.1 ± 5.3 ng/mL). Similarly, the initial serum concentrations of visfatin in participants with CD were significantly greater than those in our control group.

Saadoun et al.[8] collectively aimed to assess how blood visfatin levels in UC patients relate to the severity of their condition. The control group had significantly lower levels of ESR and CRP compared to the active patient group. As an example, ESR was 10.60 ± 2.75 vs. 7.30 ± 2.63 in the active patient group, and CRP was 4.73 ± 1.45 vs. 2.90 ± 1.45 in the control group, with a p-value of 0.001). Also, our research stated that the UC group had visfatin levels with a positive association with Erythrocyte sedimentation rate and C-reactive protein, which were significantly more significant in the control group.

Ahmed et al.[10] conducted a study to assess FC and C-reactive protein (CRP) as biochemical markers for predicting inflammatory bowel disease activity, specifically in cases with Ulcerative colitis. The results revealed a significant statistical difference in fecal calprotectin (p=0.001). In mild cases, the mean fecal calprotectin was 207.46, while in the moderate-severe group, it was substantially higher at 729.85. Similarly, CRP levels exhibited a significant difference (p=0.000), with the mean C-reactive protein in the mild group being 11.37 and in the moderate-severe group being elevated at 29.38. These findings indicate that both fecal calprotectin and CRP levels are markedly elevated in the moderate-severe group compared to those with mild activity, underscoring their potential as indicators of disease severity in UC patients. Moreover, our study illustrated that FC and serum visfatin could predict mild ulcerative colitis S1 compared to moderate S2 and severe S3 ulcerative colitis.

Furthermore, the study demonstrated that visfatin levels surpassed fecal calprotectin in distinguishing between E1 (ulcerative proctitis) and E3 (extensive ulcerative colitis). These findings align with our results, as supported by Saadoun et al.[8] who reported significantly higher serum visfatin levels in cases with Inflammatory Bowel Disease compared to the control group. This underscores the potential of serum visfatin as a valuable marker in discerning disease severity and specific phenotypes in IBD patients.

ROC curve analysis of visfatin to detect active UC revealed a high diagnostic efficacy with an AUC of 0.919. At the level of ≥2,25 ng/ml, it had 83.3% sensitivity and 86.7% specificity. In CD, ROC-AUC was 0.891. At the same cutoff, sensitivity was 76.7%, while specificity was 86.7%. Our results regarding UC are more robust than those published by Dogan et al.[11] who reported 72% sensitivity and 52% specificity in cases with active UC reached remission with treatment. The difference between our results and those reported by Dogan et al.[11] maybe due to the difference in the medical status of subjects and the different genes between races. The diagnostic accuracy was comparatively low in the research conducted by Wenxia et al.[12] where the area

under the curve (AUC) of serum visfatin for the diagnosis of CD & UC was 0.654 and 0.622, respectively.

Limitations of the study include the fact that the sample size of the studied groups was not small, which may have affected our statistical target. The insulin resistance had not yet been evaluated. In addition, the study was performed on the Egyptian population and cannot be a standard for all races other than Caucasians. Further research is needed to overcome these limitations and provide more comprehensive and reliable insights into the relationship between visfatin and IBD.

4. Conclusion

In this study, we establish a correlation between visfatin levels and inflammatory bowel disease activity. Visfatin levels may indicate the activity and diagnosis of a disease. In comparison to the control group, newly diagnosed patients with active CD and UC exhibited a significantly elevated serum visfatin level. In cases with UC, serum visfatin may serve as a novel indicator of the illness's progression and severity.

Disclosure

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Authorship

All authors have a substantial contribution to the article

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Conflicts of interest

There are no conflicts of interest.

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