

FECAL CALPROTECTIN AS A NON-INVASIVE MARKER OF ENDOSCOPIC ACTIVITY IN INFLAMMATORY BOWEL DISEASE: A RETROSPECTIVE STUDY

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Abstract

Introduction: Inflammatory bowel disease (IBD) is a chronic, relapsing–remitting inflammatory disorder of the gastrointestinal tract. Its monitoring relies mainly on endoscopy and on clinical scores, which are partly subjective. Fecal and blood biomarkers are increasingly used to reduce the need for endoscopy.

Objective: To assess the correlation of fecal calprotectin (FC), C-reactive protein (CRP), hemoglobin and platelet count with clinical and endoscopic activity, and to compare the diagnostic performance of FC and CRP for detecting endoscopic activity.

Methods: We conducted a retrospective study of IBD patients who underwent FC testing between July 2021 and July 2023 at Mohammed VI University Hospital, Oujda, Morocco. A total of 235 assessments from 155 patients were analyzed. Disease activity was defined using GETAID-recommended scores: Harvey–Bradshaw Index and CDEIS in Crohn's disease (CD), Mayo score and UCEIS in ulcerative colitis (UC). Correlations were assessed using Spearman's coefficient (ρ), and diagnostic performance using receiver operating characteristic (ROC) curves.

Results and discussion: The cohort included 101 patients with CD, 33 with UC and 21 with unclassified or undocumented IBD; 54.8% were women and the mean age was 37.8 years. Median FC was 78 $\mu\text{g/g}$ and median CRP 4.39 mg/L. FC correlated weakly with endoscopic activity ($\rho = 0.27$, $p = 0.002$; $\rho = 0.30$, $p = 0.003$ in CD) and with clinical activity ($\rho = 0.21$, $p = 0.002$). CRP also correlated weakly with endoscopic activity ($\rho = 0.24$, $p = 0.006$) but not with clinical activity ($\rho = 0.11$, $p = 0.10$). Clinical and endoscopic activity were not correlated ($\rho = 0.01$, $p = 0.89$). The AUC for endoscopic activity was 0.667 (95% CI: 0.567–0.767; $p = 0.001$) for FC and 0.603 (95% CI: 0.493–0.712; $p = 0.07$) for CRP; the difference was not significant ($p = 0.35$). In CD, the AUC of FC was 0.723 (95% CI: 0.619–0.827). At $\geq 354 \mu\text{g/g}$, FC had a specificity of 90.9% for endoscopic activity.

Conclusion: FC was the only marker with a significant AUC for endoscopic activity, with a better performance in CD, and high FC values were highly specific for endoscopic inflammation. However, its correlations with disease activity were weak, its advantage over CRP was not significant, and clinical scores did not reflect endoscopic activity. FC is a useful complementary tool to select patients for endoscopy, but it cannot replace endoscopy.

Keywords: Inflammatory bowel disease; Crohn's disease; Ulcerative colitis; Fecal calprotectin; C-reactive protein; Endoscopy; Biomarkers

INTRODUCTION

Inflammatory bowel disease (IBD), which includes

Crohn's disease (CD) and ulcerative colitis (UC), is a chronic and destructive inflammatory disease of the gastrointestinal tract. Its main complications, such as stenosis, perforation and failure of medical treatment, are frequent, may lead to surgery, and can make endoscopic examination difficult or even impossible [1,2].

Relapses are detected either clinically, using activity scores, or by endoscopy, which remains the reference examination for IBD [3]. However, clinical scores are subjective, and endoscopy is invasive, costly and not always well tolerated. Alternative approaches include cross-sectional imaging, which is expensive and does not always allow an accurate assessment of inflammatory activity, and biological or fecal markers such as CRP and fecal calprotectin [3,4].

Calprotectin is a calcium- and zinc-binding heterodimer of the S100A8 and S100A9 proteins. It accounts for up to 60% of the cytosolic proteins of neutrophils and is also expressed by monocytes and macrophages; it has antibacterial and antifungal properties [5]. The migration of neutrophils into the inflamed intestinal mucosa and lumen allows calprotectin

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to be measured in stool (fecal calprotectin, FC), making it a biomarker of intestinal inflammation and a predictor of clinical relapse in IBD [6]. FC is not specific to IBD: it also increases in other inflammatory and infectious conditions (bacterial enteritis, diverticulitis, autoimmune enteropathy, intestinal polyps, colorectal cancer) and with the use of non-steroidal anti-inflammatory drugs (NSAIDs) [5]. FC is resistant to degradation by intestinal enzymes and remains stable in stool for several days at room temperature. Blood markers such as CRP an acute-phase protein synthesized by hepatocytes [7] hemoglobin and platelet count can also reflect inflammatory flares, but they are not specific to intestinal inflammation [7,8].

The aim of this study was to evaluate the correlation of FC with endoscopic and clinical activity, to compare it with other inflammatory markers (CRP, hemoglobin, platelet count), and to assess its diagnostic performance using ROC curve analysis.

MATERIALS AND METHODS

Study design and population

We conducted a retrospective study of IBD patients who underwent FC testing, together with CRP and complete blood count, between July 2021 and July 2023 at Mohammed VI University Hospital, Oujda, Morocco. Each FC measurement with its concomitant blood tests and clinical and/or endoscopic evaluation was considered an assessment; patients could contribute several assessments during follow-up.

Inclusion criteria were an established diagnosis of IBD based on standard clinical, endoscopic, radiological and histological criteria and at least one FC measurement with concomitant blood tests. Exclusion criteria were: suspected concomitant gastrointestinal infection, gastrointestinal malignancy, pregnancy, and current use of antibiotics, cytotoxic drugs, NSAIDs or alcohol.

The study was conducted in accordance with the Declaration of Helsinki. Data were collected retrospectively from medical and laboratory records and anonymized before analysis. Owing to the retrospective design, the requirement for informed consent was waived.

Assessment of disease activity

Disease activity was assessed using the scores and thresholds recommended by the GETAID (Groupe d'Étude Thérapeutique des Affections Inflammatoires du Tube Digestif) [9]. In CD, clinical activity was assessed using the Harvey–Bradshaw Index (HBI), which includes general well-being, abdominal pain, number of liquid stools, abdominal mass and extra-intestinal complications [10]; clinical remission was defined as $HBI \leq 4$, mild activity as 5–7, moderate as 8–16 and severe as > 16 [11]. In UC, clinical activity was assessed using the Mayo score (UC-DAI) [12]: ≤ 2 , inactive disease; 3–5, mild; 6–10, moderate; ≥ 11 , severe [9].

CD patients underwent ileocolonoscopy and UC patients complete colonoscopy. Endoscopic lesions were scored using

the Crohn's Disease Endoscopic Index of Severity (CDEIS) [13] and the Ulcerative Colitis Endoscopic Index of Severity (UCEIS) [14], using the GETAID scoring forms [9]. The CDEIS is based on elementary lesions (non-ulcerated lesions, superficial and deep ulcerations) and their extent in five segments (ileum, right colon, transverse colon, left colon and rectum), with additional points for ulcerated and non-ulcerated stenosis; it ranges from 0 to 44 [13]. The UCEIS is based on three descriptors vascular pattern (0–2), bleeding (0–3) and erosions/ulcers (0–3) and ranges from 0 to 8 [14]. Endoscopic remission was defined as $CDEIS < 3$ in CD and $UCEIS \leq 1$ in UC; higher values were considered endoscopically active [15,16]. Scores outside the possible range of each index (one Mayo score of 15 and one UCEIS of 10) were considered data-entry errors and excluded.

Laboratory measurements

Stool samples were collected avoiding contamination with water or urine; the first morning stool was preferred. Approximately 1 g of stool was sent to the laboratory. FC was measured using a quantitative lateral-flow immunochromatographic assay (Quantum Blue® Calprotectin High Range, Bühlmann Laboratories, Schönenbuch, Switzerland). In this sandwich immunoassay, a monoclonal capture antibody specific for calprotectin is coated on the test line, and a second monoclonal detection antibody conjugated to colloidal gold is present on the conjugate release pad. After addition of the extracted and diluted stool sample, calprotectin–conjugate complexes bind to the test line, while unbound conjugate binds to a goat anti-mouse antibody on the control line. The signal is read with a dedicated reader. Results were reported as < 30 , < 100 or $> 1,800 \mu\text{g/g}$ when outside the quantification range. The normal value in our laboratory was $< 80 \mu\text{g/g}$; because some results were only reported as $< 100 \mu\text{g/g}$, FC was considered positive at $\geq 100 \mu\text{g/g}$.

CRP, hemoglobin and platelet count were measured on blood samples collected at the time of the assessment. CRP was considered positive above 5 mg/L.

Statistical analysis

Continuous variables are presented as mean (range) or median (interquartile range, IQR), and categorical variables as numbers and percentages. FC values outside the quantification range were assigned the limit value (e.g. $< 100 \mu\text{g/g} = 100 \mu\text{g/g}$). Because clinical and endoscopic scores are ordinal and FC values were censored and non-normally distributed, correlations were assessed using Spearman's rank correlation coefficient (ρ), in the whole cohort and separately in CD (HBI, CDEIS) and UC (UCEIS). The diagnostic performance of FC and CRP for endoscopic activity was evaluated by ROC curve analysis; AUCs with 95% confidence intervals were calculated, optimal cut-offs were determined by the Youden index, and the AUCs of FC and CRP were compared on paired assessments by bootstrap resampling (2,000 replicates). All tests were two-sided, and p

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< 0.05 was considered significant. Analyses were performed using SPSS software (IBM Corp., Armonk, NY, USA) and Python.

RESULTS

Study population

A total of 235 assessments from 155 patients were analyzed (Table 1). The cohort included 101 patients with CD (163 assessments), 33 with UC (48 assessments) and 21 with unclassified or undocumented IBD (24 assessments). Women accounted for 54.8% of patients (n = 85). The mean age was 37.8 years (range 4–74): 36.4 years (range 15–74) in CD and 42.2 years (range 15–64) in UC. For patients with unclassified IBD, disease activity was taken as recorded in the medical file.

Clinical activity was available for 206 assessments (170 inactive, 36 active) and endoscopic activity for 132 assessments (33 in remission, 99 active). The median FC level was 78 µg/g (IQR 30–151), and FC was below 100 µg/g in 125 assessments (53.2%). The median CRP level was 4.39 mg/L (IQR 1.35–10.48).

Correlation analyses

Correlations are summarized in Table 2. FC showed a weak but significant correlation with endoscopic activity ($\rho = 0.27$, $p = 0.002$, $n = 137$) and with clinical activity ($\rho = 0.21$, $p = 0.002$, $n = 214$). In CD, FC correlated with the CDEIS ($\rho = 0.30$, $p = 0.003$) and with the HBI ($\rho = 0.18$, $p = 0.022$), whereas no significant correlation was found in UC (UCEIS: $\rho = 0.12$, $p = 0.51$; Mayo score: $\rho = 0.25$, $p = 0.10$).

CRP correlated weakly with endoscopic activity ($\rho = 0.24$, $p = 0.006$) but not with clinical activity ($\rho = 0.11$, $p = 0.10$). Platelet count correlated weakly with clinical activity ($\rho = 0.16$, $p = 0.025$) but not with endoscopic activity ($\rho = 0.13$, $p = 0.13$). Hemoglobin was not correlated with clinical ($\rho = -0.09$, $p = 0.17$) or endoscopic activity ($\rho = -0.12$, $p = 0.17$). Clinical activity was not correlated with endoscopic activity ($\rho = 0.01$, $p = 0.89$).

Positivity of biomarkers according to disease activity

FC (≥ 100 µg/g) was positive in 44.4% (44/99) of endoscopically active assessments and negative in 75.8% (25/33) of assessments in endoscopic remission. In clinically active assessments, FC was positive in 41.7% (15/36) and negative in 65.3% (111/170) of clinically inactive assessments.

CRP (> 5 mg/L) was positive in 57.1% (56/98) of endoscopically active assessments and negative in 58.1% (18/31) of assessments in endoscopic remission. It was positive in 45.7% (16/35) of clinically active assessments and negative in 50.0% (82/164) of inactive ones.

ROC curve analysis

ROC analyses were performed on assessments with both a biomarker value and an endoscopic evaluation (132 for FC,

129 for CRP) (Table 3). The AUC for endoscopic activity was 0.667 (95% CI: 0.567–0.767; $p = 0.001$) for FC and 0.603 (95% CI: 0.493–0.712; $p = 0.07$) for CRP. The difference between the two markers was not statistically significant ($\Delta\text{AUC} = 0.062$; 95% CI: -0.075 to 0.184; $p = 0.35$). In CD, the AUC was 0.723 (95% CI: 0.619–0.827; $p < 0.001$) for FC and 0.659 (95% CI: 0.542–0.776; $p = 0.008$) for CRP. In UC, only 4 assessments were in endoscopic remission, which did not allow a reliable ROC analysis.

The optimal FC cut-off identified by the Youden index was 31 µg/g (sensitivity 76.8%, specificity 57.6%), i.e. close to the lower limit of quantification of the assay. At the laboratory positivity threshold of 100 µg/g, sensitivity was 44.4% and specificity 75.8%; at 354 µg/g, sensitivity was 24.2% and specificity 90.9%. For CRP, the optimal cut-off was 2.13 mg/L (sensitivity 75.5%, specificity 45.2%).

DISCUSSION

IBD causes significant morbidity and requires endoscopy for diagnosis and follow-up [3]. In recent years, FC has been increasingly used as a non-invasive marker for monitoring IBD [3,4].

In this study, FC was weakly correlated with endoscopic activity, more clearly in CD ($\rho = 0.30$) than in UC, and it was the only marker with an AUC significantly greater than 0.5 for detecting endoscopic inflammation (0.667 overall and 0.723 in CD). However, the strength of these associations was limited: CRP was also weakly correlated with endoscopic activity, and the difference between the AUCs of FC and CRP was not significant.

Previous studies have reported FC thresholds of 50–100 µg/g for detecting intestinal inflammation and have shown that FC increases with the severity of inflammation [17,18]. In our study, clinical activity was not correlated with endoscopic findings, confirming that symptoms alone do not reliably reflect mucosal inflammation and that objective markers are needed.

In a meta-analysis, FC was more sensitive than CRP for detecting endoscopic activity in IBD [4]. In UC, FC correlated with clinical and endoscopic activity more closely than CRP [19], and several studies have shown that FC correlates well with endoscopic and histological activity [17,18]. Patients in sustained remission have very low FC levels (median < 40 µg/g), whereas those who relapse have significantly higher levels [20].

The correlations observed in our study were weaker than in most published series. Several factors may explain this: the retrospective design; the mixing of two endoscopic scales (CDEIS and UCEIS) in the overall analysis; the small number of UC assessments, with very few in endoscopic remission; the high proportion of FC results reported only as below the quantification limit (53.2%), which reduces the information available for rank-based analyses; and the possible delay between stool sampling and endoscopy.

Treatment targets in IBD now include endoscopic remission, and FC is used as a monitoring target. Published cut-offs

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vary: a threshold of 56 µg/g has been reported to predict sustained remission [21], and 250 µg/g was proposed in a study of 123 UC patients [22]. In our study, the Youden-optimal cut-off was low (31 µg/g), favoring sensitivity, whereas a value ≥ 354 µg/g had a specificity of 90.9%. In practice, a high FC value strongly suggests endoscopic activity, whereas a low value does not exclude it.

Distinguishing symptoms of irritable bowel syndrome from active IBD remains challenging, and such symptoms may lead to unnecessary escalation of IBD therapy, with potential side effects [23,24]. A simple, inexpensive non-invasive marker such as FC can help in this setting.

Limitations. This study has several limitations: its retrospective, single-center design; the limited number of assessments with paired endoscopy ($n = 133$); the inclusion of repeated assessments from the same patients, which were analyzed as independent observations; the presence of patients with unclassified IBD; the censoring of FC values below the quantification limit; and the lack of a standardized interval between stool sampling and endoscopy. Prospective studies, analyzing CD and UC separately, are needed.

CONCLUSION

In our population, FC was the only marker with a significant diagnostic performance for endoscopic activity, particularly in CD, and high FC values were highly specific for endoscopic inflammation. However, its correlations with disease activity were weak and its advantage over CRP was not statistically significant. Clinical scores did not reflect endoscopic activity. FC is therefore a useful complementary tool to select patients for endoscopy, but it cannot replace endoscopy.

DECLARATIONS

Ethics approval: Waived owing to the retrospective, anonymized design. **Conflicts of interest:** The authors declare no conflicts of interest. **Funding:** This research received no external funding.

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Table 1. Characteristics of the study population

Characteristic	Value
Patients / assessments, n	155 / 235
Crohn's disease, n (%)	101 (65.2)
Ulcerative colitis, n (%)	33 (21.3)
Unclassified or undocumented IBD, n (%)	21 (13.5)
Female / Male, n (%)	85 (54.8) / 70 (45.2)
Mean age, years (range)	37.8 (4–74)
Crohn's disease	36.4 (15–74)
Ulcerative colitis	42.2 (15–64)
Median fecal calprotectin, µg/g (IQR)	78 (30–151)
FC < 100 µg/g, n (%) of assessments	125 (53.2)
Median CRP, mg/L (IQR)	4.39 (1.35–10.48)
Clinically active / inactive assessments, n	36 / 170
Endoscopically active / remission assessments, n	99 / 33

Table 2. Spearman correlations between biomarkers, clinical activity and endoscopic activity

Comparison	All IBD ρ (p) [n]	Crohn's disease ρ (p) [n]	Ulcerative colitis ρ (p) [n]
FC vs endoscopic activity	0.27 (0.002) [137]	0.30 (0.003) [97]	0.12 (0.51) [33]
FC vs clinical activity	0.21 (0.002) [214]	0.18 (0.022) [155]	0.25 (0.10) [47]
CRP vs endoscopic activity	0.24 (0.006) [133]	0.21 (0.039) [94]	0.14 (0.44) [33]
CRP vs clinical activity	0.11 (0.10) [206]	0.17 (0.039) [148]	−0.09 (0.54) [47]
Hemoglobin vs endoscopic activity	−0.12 (0.17) [135]	−0.19 (0.067) [96]	−0.11 (0.56) [32]
Hemoglobin vs clinical activity	−0.09 (0.17) [211]	−0.12 (0.15) [153]	−0.15 (0.32) [46]
Platelets vs endoscopic activity	0.13 (0.13) [128]	0.13 (0.22) [91]	0.11 (0.57) [31]
Platelets vs clinical activity	0.16 (0.025) [204]	0.15 (0.067) [148]	0.13 (0.39) [45]
Clinical vs endoscopic activity	0.01 (0.89) [137]	0.05 (0.62) [97]	0.04 (0.83) [33]

Table 3. Diagnostic performance of fecal calprotectin and CRP for detecting endoscopic activity (ROC analysis)

Biomarker	n	AUC (95% CI)	p-value	Cut-off	Sensitivity (%)	Specificity (%)
FC – all IBD	132	0.667 (0.567–0.767)	0.001	31 µg/g (Youden)	76.8	57.6
				100 µg/g	44.4	75.8
				354 µg/g	24.2	90.9
FC – Crohn's disease	97	0.723 (0.619–0.827)	<0.001			
CRP – all IBD	129	0.603 (0.493–0.712)	0.07	2.13 mg/L (Youden)	75.5	45.2
CRP – Crohn's disease	94	0.659 (0.542–0.776)	0.008			
FC vs CRP (paired, n = 129)		ΔAUC 0.062 (−0.075 to 0.184)	0.35			