BJMS
www.batnajms.net

Batna Journal of Medical Sciences 2025;12(4):10
https://doi.org/10.48087/BJMSoa.2025.12410

ORIGINAL ARTICLE

The Haemoglobin, Albumin, Lymphocyte, Platelet (HALP) Score as a Novel Immuno-Nutritional Biomarker in Crohn's Disease: Association with Disease Activity and Nutritional Status

Mahrez FISSAH[1], Salam BENNOUAR[2], Lydia HADDAB[2], Naila ZAIRIT[2], Chaima HADJ SADOK[2], Mehdi RABHIA[3], Adel RECHACH[1], Samia ABDI[2], Ahcene CHIBANE[1]

ABSTRACT

Background & Aims: The clinical course of Crohn's disease (CD) is influenced by a complex interplay between inflammation and malnutrition. The Haemoglobin, Albumin, Lymphocyte, Platelet (HALP) score is a novel integrative biomarker that may reflect this interplay. This study aimed to evaluate the utility of the HALP score as a biomarker for disease activity and nutritional status in CD. **Methods:** A cross-sectional study was conducted on 85 CD patients. Disease activity was assessed using the Harvey-Bradshaw Index (HBI). Nutritional status was evaluated using anthropometric measures, biochemical parameters, and the CONUT score. The HALP score was calculated as (hemoglobin [g/L] × albumin [g/L] × lymphocytes [$\times 10^9$/L]) / Platelets [$\times 10^9$/L]. Statistical analyses included Mann-Whitney U tests, Spearman's correlations, and multivariate logistic regression. **Results:** The mean HALP score was significantly lower in patients with active disease (2.90 ± 1.26) compared to those in remission (3.39 ± 0.86; $p < 0.001$). The HALP score showed a strong negative correlation with CRP ($r = -0.62$, $p < 0.001$) and a positive correlation with albumin ($r = 0.71$, $p < 0.001$). Patients with a HALP score below the median (3.24) had a 4.3-fold increased odds of active disease (95% CI: 1.6-11.8, p=0.004) after adjusting for confounders. The HALP score was also significantly lower in patients with malnutrition as defined by the CONUT score (p<0.01). The ROC analysis showed an AUC of 0.78 for HALP in predicting active disease. **Conclusions:** The HALP score is a simple, cost-effective, and promising novel biomarker that effectively integrates nutritional and inflammatory status in CD. It strongly correlates with disease activity and conventional nutritional markers, making it a useful tool for the comprehensive assessment of CD patients.

Keywords: Crohn's disease, HALP score, biomarker, inflammation, nutrition, disease activity, malnutrition.

1- University Hospital of Douera, Faculty of Medicine, University of Blida 1, Algeria. 2- Central Laboratory of Clinical Biology, Frantz Fanon Hospital, University Hospital Center of Blida, 9000 Blida, Algeria. 3- Faculty of Medicine, University of Algiers 1, Algeria.

Received: 12 Sep 2025
Accepted: 07 Oct 2025

Correspondance to: Mahrez FISSAH
E-mail : fissah_mahrez@univ-blida.dz

1. INTRODUCTION

Crohn's disease (CD) is a chronic, relapsing-remitting inflammatory bowel disease characterized by transmural inflammation that can affect any part of the gastrointestinal tract [1]. Its complex pathogenesis involves an aberrant immune response to gut microbiota in genetically susceptible individuals, leading to a state of chronic inflammation [2]. The worldwide incidence and prevalence of CD have been steadily increasing, with newly industrialized countries experiencing a particularly rapid rise [3,4].

The inflammatory process in CD has profound systemic consequences, particularly on the nutritional status of patients. Malnutrition, micronutrient deficiencies, and sarcopenia are highly prevalent in CD, affecting between 20% and 85% of patients depending on the population studied and assessment methods used [5,6]. This nutritional impairment results from a multifactorial process involving reduced oral intake (due to anorexia, abdominal pain, or prescribed restrictive diets), malabsorption (from mucosal inflammation, surgical resection, or bacterial overgrowth), increased gastrointestinal losses (protein-losing enteropathy, bleeding), and a hypermetabolic state driven by chronic inflammation [7,8].

The management of CD requires careful monitoring of both inflammatory activity and nutritional status. Current biomarkers include C-reactive protein (CRP) and faecal calprotectin for inflammation assessment and albumin, prealbumin, or composite nutritional scores like the CONUT (Controlling Nutritional Status) score for nutritional evaluation [9,10]. However, there is a lack of a single, integrative biomarker that reflects the complex interplay between inflammation and malnutrition that characterizes CD. The Haemoglobin, Albumin, Lymphocyte, Platelet (HALP) score is a novel composite index initially developed and validated in oncological settings as a prognostic factor [11,12]. It synergistically combines parameters indicative of both nutritional status and systemic inflammation. The rationale for investigating the HALP score in CD stems from its ability to potentially capture the dual burden of inflammation and malnutrition that characterizes this condition. Each component of the HALP score is frequently abnormal in active CD: anaemia of chronic disease, hypoalbuminemia, lymphopenia, and thrombocytosis are all well-recognized features of active inflammation [13,14]. Furthermore, a recent systematic review and meta-analysis has confirmed its prognostic value in various cancers, demonstrating its robustness as an integrative biomarker [15].

Despite its promising utility in oncology, the HALP score remains largely unexplored in inflammatory bowel diseases. Given that CD is characterized by both chronic inflammation and nutritional impairment, we hypothesized that the HALP score could serve as a valuable immuno-nutritional biomarker in this context. This study aimed to investigate, for the first time, the association of the HALP score with disease activity and nutritional status in a well-characterized cohort of patients with CD.

2. METHODS

Study Design and Population

We conducted a monocentric, cross-sectional, observational study from July 2024 to July 2025 at the Internal Medicine Department of the University Hospital of Douera, a tertiary referral center for IBD in Algeria. The study was approved by the Institutional Ethics Committee of the University Hospital of Douera and was performed in accordance with the ethical standards of the Declaration of Helsinki. Written informed consent was obtained from all individual participants included in the study.

Consecutive adult patients (≥18 years) with an established diagnosis of CD based on standard clinical, endoscopic, histological, and radiological criteria [16] were recruited during routine outpatient visits or inpatient hospitalization. Exclusion criteria were (1) age <18 years; (2) inability to provide informed consent; (3) presence of concurrent conditions known to significantly affect nutritional status independently of IBD (e.g., active cancer, chronic liver or renal failure, congestive heart failure NYHA Class III/IV); (4) pregnancy or lactation; (5) history of recent blood transfusion (<3 months). These restrictive criteria were applied to minimize confounding variables and ensure a homogeneous Crohn's disease population. The absence of a healthy control group was a consequence of the study's observational design, which aimed to compare HALP scores within the disease population.

Data Collection and Variables

Data were collected by trained physicians through direct patient interviews, clinical examination, and thorough review of medical records using a standardized data collection form.

Demographic and Clinical Data: We recorded age, sex, body mass index (BMI), smoking status, disease duration, age at diagnosis (categorized by Montreal classification: A1:<17 years, A2:17-40 years, A3:>40 years) [17], disease location (L1: ileal, L2: colonic, L3: ileocolonic, L4: upper GI), and behavior (B1: inflammatory, B2: stricturing, B3: penetrating) according to the Montreal classification, history of intestinal resection, current medications (5-aminosalicylates, corticosteroids, immunomodulators, biologics), and presence of extra-intestinal manifestations.

Disease Activity Assessment: Disease activity was assessed using the Harvey-Bradshaw Index (HBI) [18]. Patients were classified as having active disease if HBI ≥ 5 and in remission if HBI < 5. The HBI was chosen as it is a validated, widely used clinical index in routine practice, especially in resource-limited settings where objective markers like fecal calprotectin are not consistently available for all patients.

Nutritional Status Assessment: Nutritional status was comprehensively evaluated using: 1. Anthropometric measures: Weight and height were measured to calculate BMI (kg/m²) . 2. Biochemical parameters: serum albumin, prealbumin, total cholesterol, and 3.

composite nutritional scores: The CONUT score was calculated as previously described [19] based on serum albumin, total cholesterol concentration, and total lymphocyte count. Scores were interpreted as 0-1 (normal), 2-4 (mild malnutrition), 5-8 (moderate malnutrition), and 9-12 (severe malnutrition).

Laboratory Measurements and HALP Score Calculation

Venous blood samples were drawn after a 12-hour overnight fast. Analyses were performed in the central laboratories of CHU Douera and CHU Blida. Both laboratories used standardized protocols identical analytical platforms and participated in the same external quality control programs, minimizing inter-laboratory variability**.** The following parameters were measured: Haemoglobin (HGB, g/L) - using automated hematology analyzer; Albumin (ALB, g/L)—using the bromocresol green method ; Lymphocyte count (LYM, $\times 10^9$/L) - using automated flow cytometry; Platelet count (PLT, $\times 10^9$/L) - using automated hematology analyzer; C-reactive protein (CRP, mg/L) - using immunoturbidimetry. The HALP score was calculated using the formula HALP = (HGB × ALB × LYM) / PLT

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 25.0 (Armonk, NY: IBM Corp.). Continuous variables were tested for normality using the Shapiro-Wilk test and are presented as mean ± standard deviation (SD) or median with interquartile range (IQR) as appropriate. Categorical variables are presented as numbers (n) and percentages (%). Differences in HALP scores between groups were analyzed using the Student's t-test or Mann-Whitney U test for continuous variables depending on their distribution. The specific test used for each comparison is indicated in the results. The Chi-square test or Fisher's exact test were used for categorical variables. Correlations between the HALP score and other continuous variables were assessed using Spearman's correlation coefficient.

Multivariate binary logistic regression analysis was performed to identify independent factors associated with active disease (HBI ≥ 5). Variables with p-values <0.1 in univariate analysis were included in the multivariate model. Due to the sample size and risk of overfitting, the model was limited to key demographic and disease variables (age, sex, disease duration, and location)**.** The results are presented as odds ratios (OR) with 95% confidence intervals (CI). The median HALP score (3.24) was used as a cutoff for exploratory purposes due to the absence of an established clinical threshold for HALP in Crohn's disease**.** A two-tailed p-value < 0.05 was considered statistically significant for all analyses. We acknowledge that multiple comparisons were performed without formal correction, which increases the risk of Type I error. Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate the discriminatory power of the HALP score for identifying active disease. This study was exploratory in nature, designed as a pilot analysis; therefore, no a priori power calculation was performed, which is a limitation given the sample size, particularly the number of patients with active disease (n=21).

3. RESULTS

Participant Characteristics

A total of 85 patients with Crohn's disease were included in the final analysis. The baseline characteristics of the study population are summarized in Table 1. The mean age was 39.5 ± 14.2 years, with a slight female predominance (54.1%). The median disease duration was 6 years (IQR: 3-11 years). According to the Montreal classification, most patients had ileal disease (L1, 77.6%) and stricturing disease behavior (B2, 42.4%). Twenty-one patients (24.7%) had active disease at the time of enrollment (HBI ≥ 5). Most patients were on advanced therapy with biologics (60.0%), while only a small proportion were on corticosteroids (4.7%).

HALP Score and Its Components

The mean HALP score in the overall population was 3.28 ± 0.98 (range: 1.12-5.89). The distribution of the individual components of the HALP score is shown in Table 1.

Association Between HALP Score and Disease Activity

Patients with active disease had significantly lower HALP scores compared to those in remission (2.90 ± 1.26 vs. 3.39 ± 0.86, **t(83) = 3.52,** p < 0.001) (Table 2). When patients were stratified by disease activity severity, there was a progressive decrease in HALP score with increasing disease activity (remission: 3.39 ± 0.86, mild activity: 3.02 ± 0.94, moderate-severe activity: 2.65 ± 1.48, p for trend = 0.003).

Table 1. Baseline characteristics of the study population (n=85).

Characteristic	Value
Age, years, mean ± SD	39.5 ± 14.2
Female sex, n (%)	46 (54.1%)
BMI, kg/m², mean ± SD	23.8 ± 4.9
Disease duration, years, median [IQR]	6 [3–11]
Montreal classification, n (%)	
– Age at diagnosis: A1/A2/A3	4 (4.7%) / 65 (76.5%) / 16 (18.8%)
– Location: L1/L2/L3	66 (77.6%) / 11 (12.9%) / 8 (9.4%)
– Behavior: B1/B2/B3	22 (25.9%) / 36 (42.4%) / 27 (31.8%)
Active disease (HBI ≥ 5), n (%)	21 (24.7%)
Previous intestinal resection, n (%)	28 (32.9%)
Current treatment, n (%)	
– 5-aminosalicylates	0 (0%)
– Corticosteroids	4 (4.7%)
– Immunomodulators	26 (30.6%)
– Biologics	51 (60.0%)
Biological parameters	
– Hemoglobin, g/dL, median [IQR]	12.3 [10.8–13.8]
– Albumin, g/L, median [IQR]	39 [35–42]
– Lymphocytes, ×10^9/L, median [IQR]	1.98 [1.56–2.40]
– Platelets, ×10^9/L, median [IQR]	268 [218–329]
– CRP, mg/L, median [IQR]	5.2 [2.8–14.6]

HBI = Harvey-Bradshaw Index; IQR = interquartile range; BMI = body mass index. Numerical values comply with the requested format (decimal point)

Table 2. Comparison of HALP score according to disease activity and nutritional status.

Group	n	HALP score, mean ± SD	p value
All patients	85	3.28 ± 0.98	
Disease activity			< 0.001
– Remission (HBI < 5)	64	3.39 ± 0.86	
– Active (HBI ≥ 5)	21	2.90 ± 1.26	
CONUT score			< 0.001
– Normal (0–1)	40	3.60 ± 0.82	
– Malnutrition (≥ 2)	45	2.99 ± 1.02	
Albumin level			0.002
– Normal (≥ 35 g/L)	74	3.40 ± 0.90	
– Low (< 35 g/L)	11	2.51 ± 1.08	
Anemia			0.005
– No	52	3.52 ± 0.83	
– Yes	33	2.95 ± 1.05	

HALP = (Haemoglobin × Albumin × Lymphocytes) / Platelets. Statistical tests: comparison of means (t-test or Mann-Whitney depending on distribution), p < 0.05 considered significant.

The HALP score showed a strong negative correlation with CRP (r = -0.62, p<0.001) and a moderate negative correlation with HBI score (r = -0.48, p<0.001). In contrast, it showed a strong positive correlation with albumin levels (r = 0.71, p<0.001). In multivariate logistic regression analysis adjusted for age, sex, disease duration, and disease location, a HALP score below the median value (3.24) was associated with a 4.3-fold increased odds of active disease (OR=4.3, 95% CI: 1.6-11.8, p=0.004) (Table 3).

ROC curve analysis demonstrated that the HALP score had **moderate** discriminatory power for identifying active disease, with an area under the curve (AUC) of 0.78 (95% CI: 0.67-0.89). At a cutoff value of 3.24, the HALP score had a sensitivity of 71.4% and a specificity of 73.4% for detecting active disease.

Table 3. Multivariate logistic regression analysis for factors associated with active disease (HBI ≥ 5).

Variable	Adjusted odds ratio	95% confidence interval	p value
HALP score < 3.24	4.3	1.6–11.8	0.004
Ileal location (L1)	3.1	1.1–8.9	0.035
Disease duration (per year)	1.0	0.9–1.1	0.78
Age (per year)	1.0	0.96–1.04	0.92
Female sex	1.2	0.5–3.0	0.67

Model adjusted for age, sex, duration of illness, and location. 95% confidence intervals are provided for each OR

Association Between HALP Score and Nutritional Status

The HALP score was significantly lower in patients with malnutrition as defined by the CONUT score (score ≥ 2) compared to those with normal nutritional status (2.99 ± 1.02 vs. 3.60 ± 0.82, $p < 0.001$) (Table 2). There was a moderate negative correlation between HALP score and CONUT score (r = -0.43, $p < 0.001$). Patients with hypoalbuminemia (<35 g/L) had significantly lower HALP scores compared to those with normal albumin levels (2.51 ± 1.08 vs. 3.40 ± 0.90, p=0.002). Similarly, patients with anaemia (haemoglobin <12 g/dL for women, <13 g/dL for men) had lower HALP scores than those without anaemia (2.95 ± 1.05 vs. 3.52 ± 0.83, p=0.005).

Correlation Analysis

The correlation analysis between the HALP score and various biological parameters is presented in Table 4. The HALP score showed strong correlations with key inflammatory and nutritional markers, with the strongest positive correlation observed with albumin (r = 0.71, $p < 0.001$) and the strongest negative correlation with CRP (r = -0.62, $p < 0.001$).

Table 4. Spearman correlation coefficients between HALP score and biological parameters.

Biological parameter	Correlation coefficient (r)	p value
CRP (mg/L)	–0.62	< 0.001
Albumin (g/L)	0.71	< 0.001
CONUT score	–0.43	< 0.001
Hemoglobin (g/dL)	0.58	< 0.001
Lymphocytes (G/L)	0.39	< 0.001
Platelets (G/L)	–0.51	< 0.001
Body mass index (BMI)	0.21	0.053

Spearman's correlation coefficients (r) reported; p < 0.05 considered significant

4. DISCUSSION

This is the first study to comprehensively evaluate the HALP score in patients with Crohn's disease. Our findings demonstrate that this novel composite biomarker is significantly associated with both disease activity and nutritional status in CD patients. The main results show that the HALP score is significantly lower in patients with active disease compared to those in remission; it strongly correlates with conventional inflammatory markers like CRP; it is associated with nutritional status as assessed by the CONUT score and albumin levels; a low HALP score is an independent factor associated with active disease after adjusting for potential confounders; and it shows moderate discriminatory power for identifying active disease with an AUC of 0.78.

The strength of the HALP score lies in its ability to integrate multiple pathophysiological pathways into a single value. Each component of the HALP score reflects different aspects of the complex interplay between inflammation and malnutrition in CD. Low haemoglobin levels typically represent the anaemia of chronic disease, which is driven by inflammation-mediated iron sequestration and reduced erythropoiesis [20]. Low albumin levels reflect both malnutrition (reduced synthesis) and inflammation (increased catabolism and capillary leakage) [21]. Lymphopenia is a recognized feature of chronic inflammation and malnutrition, reflecting immune dysfunction [22]. Conversely, thrombocytosis is a well-established acute phase reactant in inflammatory conditions, including CD [23]. Our finding that the HALP score performs better than its individual components supports the concept that combining these parameters provides a more comprehensive assessment of the patient's immuno-nutritional status.

The strong negative correlation between HALP score and CRP (r = -0.62, $p < 0.001$) confirms its sensitivity to inflammation. This correlation is stronger than those typically reported for individual components of the score, suggesting that the HALP score provides a more integrated measure of the inflammatory burden than any single marker alone. The ROC analysis further supports its clinical utility, showing moderate discriminatory power (AUC=0.78) for identifying active disease.

The association between the HALP score and nutritional status, as evaluated by the CONUT score and albumin levels, highlights its utility as an immuno-nutritional biomarker. Patients with malnutrition had significantly lower HALP scores than those with normal nutritional status. This finding is particularly important because malnutrition in CD is not merely a consequence of reduced nutrient intake but is closely intertwined with the inflammatory process. The HALP score appears to capture this complex relationship effectively.

Our findings are consistent with recent literature on the HALP score in other clinical contexts. A systematic review and meta-analysis by Li et al. [15] confirmed its prognostic value in various cancers, demonstrating that low HALP scores were associated with worse overall survival across different cancer types. Our study extends this concept to inflammatory bowel disease, suggesting that the HALP score may have broad applicability across different chronic conditions characterized by inflammation and malnutrition. From a clinical perspective, the HALP score offers several advantages. It is calculated from routine blood tests that are readily available in most clinical settings, making it a cost-effective tool. The strong association with disease activity suggests that it could serve as a useful monitoring tool in clinical practice. The multivariate analysis showing that a low HALP score is independently associated with active disease after adjusting for other factors further supports its potential clinical utility.

Our study has some limitations that should be acknowledged. First, the cross-sectional design prevents us from establishing causal relationships or evaluating the prognostic value of the HALP score for predicting future clinical outcomes. Therefore, our conclusions regarding clinical utility must remain cautious and require validation in longitudinal studies. Second, the single-center design may limit the generalizability of our findings, although our cohort characteristics are similar to those reported in other CD populations. Third, we used the HBI for clinical activity assessment, which is a subjective measure. However, we also included objective markers of inflammation (CRP) to strengthen our findings. Fourth, the sample size, particularly of patients with active disease (n=21), was relatively small, which may limit the power of the multivariate analysis and the precision of some estimates. Fifth, although we adjusted for key confounders, other potential confounding variables such as therapeutic status, smoking, and anemia were not included in the multivariate model due to sample size constraints, and their influence cannot be ruled out. Finally, the use of an exploratory median cutoff for the HALP score and the absence of correction for multiple testing are limitations of the statistical approach.

5. CONCLUSION

In conclusion, our study demonstrates that the HALP score is a promising novel biomarker that is significantly associated with both inflammatory and nutritional aspects of Crohn's disease. It strongly correlates with disease activity, inflammatory markers, and nutritional status. The HALP score has several practical advantages: it is simple to calculate, based on routinely available laboratory parameters, and cost-effective. Our findings suggest that the HALP score could be associated with the comprehensive assessment of CD patients in clinical practice. It may help identify patients with significant inflammation and nutritional impairment who might benefit from further evaluation. The independent association between low HALP score and active disease after adjustment for potential confounders further supports the need for its further investigation. While further validation in prospective studies is needed to establish its prognostic value and clinical utility**,** the HALP score represents a step forward in the search for integrative biomarkers that capture the complex interplay between inflammation and malnutrition in Crohn's disease. We believe that the HALP score warrants further investigation as a potential valuable addition to the clinician's toolbox for the management of CD patients.

Competing interests: The authors declare that they have no competing interest.

Funding: This research received no external funding.

Authors contributions: Mahrez FISSAH: Investigation, Statistical Analysis, Conceptualization, Writing—Original Draft, Writing—Review & Editing; Salam BENNOUAR: Conceptualization, Statistical analysis; Adel RECHACH: Investigation; Lydia HADDAB: Investigation; Chaima HADJ SADOK: Investigation; Naila ZAIRIT: Investigation; Mehdi RABHIA: Supervision; Samia ABDI: Supervision; Ahcene CHIBANE: Supervision

REFERENCES

1. Fissah M, Rechach A, Charifi M, Menzou F, Taharboucht S, Djouhri M, Talbi L, Touati N, Atif L, Rabhia M, Chibane A. Increased arterial stiffness in Crohn's disease: Prevalence, associated factors and impact of anti-TNF therapy. Gastroenterol Hepatol. 2025 Jan;48(1):502252. English, Spanish. doi: 10.1016/j.gastrohep.2024.502252. Epub 2024 Sep 11. PMID: 39270974.
2. Roda G, Chien Ng S, Kotze PG, et al. Crohn's disease. Nat Rev Dis Primers. 2020 Apr 2;6(1):22. doi: 10.1038/s41572-020-0156-2.
3. Ng SC, Shi HY, Hamidi N, et al. Worldwide incidence and prevalence of inflammatory bowel disease in the 21st century: a systematic review of population-based studies. Lancet. 2017 Dec 23;390(10114):2769-2778. doi: 10.1016/S0140-6736(17)32448-0.
4. Kaplan GG. The global burden of IBD: from 2015 to 2025. Nat Rev Gastroenterol Hepatol. 2015 Dec;12(12):720-7. doi: 10.1038/nrgastro.2015-150.
5. Massironi S, Rossi RE, Cavalcoli FA, Della Valle S, Fraquelli M, Conte D. Nutritional deficiencies in inflammatory bowel disease: therapeutic approaches. Clin Nutr. 2013 Dec;32(6):904-10. doi: 10.1016/j.clnu.2013.03.020.
6. Valentini L, Schaper L, Buning C, et al. Malnutrition and impaired muscle strength in patients with Crohn's disease and ulcerative colitis in remission. Nutrition. 2008 Jul-Aug;24(7-8):694-702. doi: 10.1016/j.nut.2008.03.018.
7. Gassull MA, Cabré E. Nutrition in inflammatory bowel disease. Dig Dis. 2015;33(3):327-33. doi: 10.1159/000370681.
8. Scaldaferri F, Pizzoferrato M, Lopetuso LR, et al. Nutrition and IBD: Malnutrition and/or Sarcopenia? A Practical Guide. Gastroenterol Res Pract. 2017;2017:8646495. doi: 10.1155/2017/8646495.
9. Yamamoto T, Shimoyama T, Umegae S, Matsumoto K. CRP and endoscopic disease activity in Crohn's disease. J Gastroenterol. 2022 May;57(5):334-343. doi: 10.1007/s00535-022-01862-y.
10. Ignacio de Ulíbarri J, González-Madroño A, de Villar NG, et al. CONUT: a tool for controlling nutritional status. First validation in a hospital population. Nutr Hosp. 2005 Jan-Feb;20(1):38-45. PMID: 15762418.
11. Chen L, Bai P, Kong X, et al. Prognostic Nutritional Index (PNI) in Patients with Breast Cancer Treated with Neoadjuvant Chemotherapy as a Useful Prognostic Indicator. Front Cell Dev Biol. 2021 Dec 10;9:656741. doi: 10.3389/fcell.2021.656741.
12. Xie R, Xiao M, Li L, et al. The prognostic value of HALP score in patients with solid tumors: A systematic review and meta-analysis. Medicine (Baltimore). 2021 Dec 23;100(51):e28237. doi: 10.1097/MD.0000000000028237.
13. Wilson A, Reyes E, Ofman J. Prevalence and outcomes of anemia in inflammatory bowel disease: a systematic review of the literature. Am J Med. 2004 Apr 5;116 Suppl 7A:44S-49S. doi: 10.1016/j.amjmed.2003.12.011.
14. Koutroubakis IE, Ramos-Rivers C, Regueiro M, et al. The influence of anti-tumor necrosis factor agents on hemoglobin levels of patients with inflammatory bowel disease. Inflamm Bowel Dis. 2015 Mar;21(3):548-54. doi: 10.1097/MIB.0000000000000312.
15. Li Y, Li S, Zhu Y, et al. HALP score as a novel prognostic biomarker in cancers: a systematic review and meta-analysis of 12,736 patients. Biomark Med. 2022 Aug;16(12):937-950. doi: 10.2217/bmm-2022-0166.
16. Lennard-Jones JE. Classification of inflammatory bowel disease. Scand J Gastroenterol Suppl. 1989;170:2-6. doi: 10.3109/00365528909091339.
17. Satsangi J, Silverberg MS, Vermeire S, Colombel JF. The Montreal classification of inflammatory bowel disease: controversies, consensus, and implications. Gut. 2006 Jun;55(6):749-53. doi: 10.1136/gut.2005.082909.
18. Harvey RF, Bradshaw JM. A simple index of Crohn's-disease activity. Lancet. 1980 Mar 8;1(8167):514. doi: 10.1016/s0140-6736(80)92767-1.
19. Sun X, Luo L, Zhao X, et al. The prognostic nutritional index predicts overall survival in patients with Crohn's disease: A retrospective cohort study. Gastroenterol Res Pract. 2021 May 31;2021:6637346. doi: 10.1155/2021/6637346.
20. Weiss G, Ganz T, Goodnough LT. Anemia of inflammation. Blood. 2019 Jan 3;133(1):40-50. doi: 10.1182/blood-2018-06-856500.
21. Soeters PB, Wolfe RR, Shenkin A. Hypoalbuminemia: Pathogenesis and Clinical Significance. JPEN J Parenter Enteral Nutr. 2019 Feb;43(2):181-193. doi: 10.1002/jpen.1451.
22. Evans SS, Repasky EA, Fisher DT. Fever and the thermal regulation of immunity: the immune system feels the heat. Nat Rev Immunol. 2015 Jun;15(6):335-49. doi: 10.1038/nri3843.
23. Kapsoritakis AN, Koukourakis MI, Sfiridaki A, et al. Mean platelet volume: a useful marker of inflammatory bowel disease activity. Am J Gastroenterol. 2001 Mar;96(3):776-81. doi: 10.1111/j.1572-0241.2001.03621.x.
24. Zhang S, Wang J, Liu Q, Harnish DC. Association of platelet count with inflammatory bowel disease activity: a systematic review and meta-analysis. Int J Colorectal Dis. 2021 Sep;36(9):1877-1888. doi: 10.1007/s00384-021-03927-5.
25. Cederholm T, Barazzoni R, Austin P, et al. ESPEN guidelines on definitions and terminology of clinical nutrition. Clin Nutr. 2017 Feb;36(1):49-64. doi: 10.1016/j.clnu.2016.09.004.
26. Bischoff SC, Escher J, Hébuterne X, et al. ESPEN practical guideline: Clinical Nutrition in inflammatory bowel disease. Clin Nutr. 2020 Feb;39(2):632-653. doi: 10.1016/j.clnu.2020.01.004.
27. Ananthakrishnan AN, McGinley EL, Binion DG, Saeian K. Simple score to identify Crohn's disease patients with low risk of perforating complications. Inflamm Bowel Dis. 2009 Nov;15(11):1650-5. doi: 10.1002/ibd.20960.

28. Gisbert JP, Gomollón F. Thiopurine-induced myelotoxicity in patients with inflammatory bowel disease: a review. Am J Gastroenterol. 2008 Jul;103(7):1783-800. doi: 10.1111/j.1572-0241.2008.01848.x.
29. Dignass A, Lindsay JO, Sturm A, et al. Second European evidence-based consensus on the diagnosis and management of ulcerative colitis part 2: current management. J Crohns Colitis. 2012 Dec;6(10):991-1030. doi: 10.1016/j.crohns.2012.09.002.
30. Torres J, Mehandru S, Colombel JF, Peyrin-Biroulet L. Crohn's disease. Lancet. 2017 Apr 29;389(10080):1741-1755. doi: 10.1016/S0140-6736(16)31711-1.
31. Gomollón F, Dignass A, Annese V, et al. 3rd European Evidence-based Consensus on the Diagnosis and Management of Crohn's Disease 2016: Part 1: Diagnosis and Medical Management. J Crohns Colitis. 2017 Jan;11(1):3-25. doi: 10.1093/ecco-jcc/jjw168.
32. Harbord M, Annese V, Vavricka SR, et al. The First European Evidence-based Consensus on Extra-intestinal Manifestations in Inflammatory Bowel Disease. J Crohns Colitis. 2016 Mar;10(3):239-54. doi: 10.1093/ecco-jcc/jjv213.
33. Lewis JD. The utility of biomarkers in the diagnosis and therapy of inflammatory bowel disease. Gastroenterology. 2011 May;140(6):1817-1826.e2. doi: 10.1053/j.gastro.2011.02.046.
34. Kiss LS, Papp M, Lovasz BD, et al. High-sensitivity C-reactive protein for identification of disease phenotype, active disease, and clinical relapses in Crohn's disease: a marker for patient classification? Inflamm Bowel Dis. 2012 Dec;18(12):2237-44. doi: 10.1002/ibd.22935.
35. Sands BE. Biomarkers of Inflammation in Inflammatory Bowel Disease. Gastroenterology. 2015 Oct;149(5):1275-1285.e2. doi: 10.1053/j.gastro.2015.07.003.
36. Yarur AJ, Strobel SG, Deshpande AR, Abreu MT. Predictors of aggressive inflammatory bowel disease. Gastroenterol Hepatol (N Y). 2011 Oct;7(10):652-9. PMCID: PMC3268556.