# Role of Plasma Janus Kinase 2 in Assessment of Disease Activity in Ulcerative Colitis

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**Abstract**

**Background:** Ulcerative colitis (UC) is a chronic autoimmune disease with increasing incidence worldwide. The pathophysiology of UC is multifactorial, including interplay between immune, gut microbiota, genetic vulnerability, and environment-related factors. Plasma Janus kinase 2 (JAK2) is a protein tyrosine kinase that participates in a group of cytokine receptor signaling pathways.

**Objectives:** Illustrating the role of plasma JAK2 in the assessment of disease activity in UC.

**Patients and methods:** We included 75 subjects; 50 had UC diagnoses and 25 were healthy controls. The enzyme-linked immunosorbent assay (ELISA) was used to determine the plasma JAK2 level. The UC cases were categorized using the Mayo score, Mayo sub-score, and UC Endoscopic Index of Severity (UCEIS) to assess disease activity stages.

**Results:** The median plasma JAK2 level was significantly higher in UC cases (3086.8 pg/ml) than in the control group (2952.0 pg/ml) ($p = 0.049$). Plasma JAK2 value positively correlated with the stages of UC disease activity and the inflammatory marker levels (CRP, albumin, fecal calprotectin).

**Conclusion:** Plasma JAK2 level offers a distinct, trustworthy biomarker that is helpful in tracking the level of mucosal affection in UC.

**Keywords:** Ulcerative colitis; UCEIS; Plasma Janus kinase 2; ELISA.

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## Introduction

Ulcerative colitis (UC) is a chronic debilitating autoimmune disease affecting the colonic mucosa from the rectum to the proximal colon, producing friability, superficial erosions, and bleeding **(Pasvol et al., 2020)**.

The annual documented incidence of UC is between 9 and 20 cases per person. It occurs in 156 to 291 cases per 100,000 people per year **(Danese et al., 2018).**

Uncertainty surrounds the exact pathophysiology of UC; usually it occurs due to interplay between dysregulated immune response, evolving intestinal dysbiosis, underlying genetic susceptibility, and environment-related factors **(Colombel et al., 2020)**.

Common UC symptoms include diarrhea, rectal bleeding, mucorrhea, tenesmus, urgency, and abdominal pain. Severe pancolitis can cause systemic symptoms, so the patient may complain of fatigue, fever, significant dehydration, and weight loss **(Dubinsky et al., 2022)**.

Approximately 27% of UC patients experience extraintestinal manifestations (EIMs), with one-quarter occurring before the diagnosis **(Harbord et al., 2016)**.

IBD diagnosis necessitates combining imaging, histology, endoscopic examination, laboratory markers, and clinical data. The ideal biomarker should be simple and quick, have a reasonable price, and be noninvasive with substantial sensitivity and specificity. Unfortunately, there is no one biomarker harboring all of these characteristics, yet research is ongoing to identify promising new markers **(M'Koma, 2022).**

Stool markers tend to be simple, rapid, and non-invasive techniques frequently used to assess activity in IBD patients. Fecal neopterin, fecal lactoferrin, and fecal calprotectin (FC), which is the most widely employed and available marker **(Colombel et al., 2017)**.

Acute phase reactants such as CRP and ESR are common choices to reflect disease activity **(Sturm et al., 2019)**.

JAKs are considered non-receptor tyrosine protein kinases that employ extracellular signals to achieve control of cellular growth, regulating survival, tissue differentiation, guiding proliferation, and eventually migration. The JAK/STAT system conveys signals from external cytokine stimuli, delivering them to the nucleus **(Salas et al., 2020)**.

JAK activation ultimately results in the phosphorylation of STAT (signal transducers and activators of transcription) proteins, which subsequently dimerize and enter the nucleus. Once in the nucleus, STAT proteins have the ability to affect the transcription of many different genes, including those that are triggered by interferon **(Yamaoka et al., 2004)**.

The JAK/STAT pathway plays a pivotal role in regulating T-cell differentiation. Furthermore, dysregulated JAK/STAT signaling leading to aberrant T cell development as well as flawed regulatory T-cell activity has been proposed as a crucial implementing factor in IBD etiology. Plasma JAK2 protein is a kind of non-receptor tyrosine kinase that has a mass of 130.7 kDa and an amino acid length of 1132 residues. It is known to be displayed in the cytoplasm, nucleus, and membrane of different cells **(Cordes et al., 2020)**.

## Patients and methods

### *Subjects*

The study included 75 subjects, 50 patients diagnosed with UC and further subdivided according to different stages of disease activity using the Mayo score, Mayo subscore, and UCEIS, and 25 healthy control patients. Patients were recruited from the outpatient clinic, scheduled for colonoscopy, or admitted to the internal medicine department at Alexandria Main University Hospital wards. Patients aged 18-60 year were enrolled in the study. We excluded patients

taking Jak inhibitors or other biological therapy, cases with gastrointestinal malignancies, type I DM, chronic kidney or liver disease, other autoimmune diseases, and pregnant females.

Written informed consent was taken from the subjects either by themselves or by their guardians.

**Ethical approval code:** 0201847, where the study was conducted in a way that respects the rights and dignity of the included patients. All procedures carried out in the study involving human subjects followed the ethical standards of the institutional research committee (Medical Research Ethics Committee of Alexandria Faculty of Medicine, Egypt).

***Clinical procedures***

All patients were subjected to the following:

1. A detailed medical history is taken, with specific emphasis on symptoms related to gastrointestinal diseases such as loose stool, weight loss, bleeding per rectum, and abdominal pain. This is followed by a full systemic physical examination, which looks for abdominal tenderness, palpable organs, and EIM.
2. Laboratory investigations as:

A. Erythrocyte sedimentation rate (ESR), quantitative C-reactive protein (CRP), serum albumin, ALT, AST, urea, and creatinine.

B. Fecal markers: quantitative assessment of FC by ELISA, EDI™ Quantitative Fecal Calprotectin ELISA Kit, KT-849, San Diego, United States, sensitivity / LLOD 2.5 ng /mL, cutoff value 120 ng/mL or 43.2 µg/g, the overall CV% 15.04%.

C. Determination of circulating plasma JAK2 concentration using Sandwich enzyme-linked immunoassay (ELISA), kit (Catalogue No. In-Hu4220) provided by (Innova Biotech, Co.) London, United Kingdom LTD., the sensitivity is 5 pg/ml, with a CV% of 10% within the assay and 12% between the assays.

3. Imaging: CT Entero-colonography if needed.
4. Ileocolonoscopy was performed on all patients, endoscopic lesions were recorded, endoscopic indices of activity were assessed, and tissues were obtained for histopathology in order to verify the diagnosis and assess the disease's activity.
5. Assessment of disease activity clinically by the Mayo score and endoscopically by the UC endoscopic index of severity and Mayo subscore **(Ruscio et al., 2022)**

**Statistical analysis**

Data were processed with IBM SPSS software version 20.0. (Armonk, NY: IBM Corporation) Qualitative data were described using numbers and percentages. The normality of the distribution was verified using the Kolmogorov-Smirnov test. Quantitative data were described as range (minimum and maximum), mean, standard deviation, median, and interquartile range (IQR). The significance of the acquired results was evaluated at the 5% level. The chi-square test was used for comparing categorical data. Fisher's Exact correction was used if 20% of the cells have expected counts less than 5. A student t-test was used for normally distributed quantitative variables. Mann-Whitney test for abnormally distributed quantitative variables. Correlation was done by the Spearman coefficient (r). Receiver operating characteristic curve (ROC) is generated to assess the accuracy of JAK2 in discriminating UC cases from healthy controls. The area under the curve (AUC) denotes the diagnostic performance of the test.

**Results**

***Demographic data of the studied groups***

No statistically significant difference exists between UC cases and controls regarding both age and gender (p = 0.991)

(p = 0.187), respectively (**Table.1**). No statistically significant difference exists between cases with different stages of disease activity with (p = 0.735) for age and (p = 0.239) for gender, (**Table. 2**).

**Table 1. Comparison between the two studied groups according to demographic data**

| Variables | Cases (n = 50) | | Control (n = 25) | | Test of Sig. | p |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | | |
| **Gender** | | | | | | |
| Male | 24 | 48.0 | 8 | 32.0 | $\chi^2$= 1.744 | 0.187 |
| Female | 26 | 52.0 | 17 | 68.0 | | |
| **Age (years)** | | | | | | |
| Min. – Max. | 21.0 – 56.0 | | 22.0 – 60.0 | | U= 624.00 | 0.991 |
| Mean ± SD. | 36.30 ± 9.58 | | 36.64 ± 10.92 | | | |
| Median (IQR) | 36.0 (28.0 – 44.0) | | 32.0 (28.0 – 45.0) | | | |

IQR: Inter quartile range; SD: Standard deviation; U: Mann-Whitney test; $\chi^2$: Chi-square test.

**Table 2.Comparison between the studied subgroups according to demographic data**

| Variables | Mayo score | | | | | | Test of Sig. | p |
|---|---|---|---|---|---|---|---|---|
| | Mild (n = 20) | | Moderate (n = 19) | | Severe (n = 11) | | | |
| | No. | % | No. | % | No. | % | | |
| **Gender** | | | | | | | | |
| Male | 8 | 40.0 | 12 | 63.2 | 4 | 36.4 | $\chi^2$= 2.859 | 0.239 |
| Female | 12 | 60.0 | 7 | 36.8 | 7 | 63.6 | | |
| **Age (years)** | | | | | | | | |
| Min. – Max. | 23.0 – 56.0 | | 22.0 – 50.0 | | 21.0 – 50.0 | | H= 0.617 | 0.735 |
| Mean ± SD. | 37.60 ± 10.85 | | 35.21 ± 8.55 | | 35.82 ± 9.39 | | | |
| Median (IQR) | 37.5(28.50– 44.50) | | 35.0 (28.0 – 41.0) | | 39.0(29.50 – 41.50) | | | |

IQR: Inter quartile range; SD: Standard deviation; χ2: Chi-square test; H: H for Kruskal Wallis test

(**Table .3)** displays the cases' distribution into different stages of disease activity based on the Mayo score, Mayo subscore and UCEIS in group I (patients with UC) as recommended by British Society of Gastroenterology consensus guidelines on the management of IBD in adults.

**Table 3.Distribution of UC cases according to Mayo score, Mayo sub-score, and UCEIS**

| Variables | No. | % |
|---|---|---|
| **Mayo score** | | |
| Mild | 20 | 40.0 |
| Moderate | 19 | 38.0 |
| Severe | 11 | 22.0 |
| Min. – Max. | 2.0 – 9.0 | |
| Mean ± SD. | 5.12 ± 2.07 | |
| Median (IQR) | 5.0 (4.0 – 6.0) | |
| **Mayo sub-score** | | |
| Mild | 20 | 40.0 |
| Moderate | 19 | 38.0 |

| | | |
|---|---|---|
| Severe | 11 | 22.0 |
| Min. – Max. | 1.0 – 3.0 | |
| Mean ± SD. | 1.82 ± 0.77 | |
| Median (IQR) | 2.0 (1.0 – 2.0) | |
| **UCEIS** | | |
| Mild | 20 | 40.0 |
| Moderate | 19 | 38.0 |
| Severe | 11 | 22.0 |
| Min. – Max. | 2.0 – 8.0 | |
| Mean ± SD. | 5.0 ± 1.92 | |
| Median (IQR) | 5.0 (4.0 – 6.0) | |

IQR: Inter quartile range; SD: Standard deviation

The UC cases had significantly higher CRP and ESR in the 1st hour, 2nd hour, and FC levels than in the control group, whereas serum albumin level was significantly lower in the UC group ($p < 0.001$). The plasma JAK2 was significantly higher in UC cases with a median of 3086.8 pg/ml, while it was 2952.0 pg/ml in the control group ($p = 0.049$), (**Table.4).**

**Table 4. Laboratory investigations' comparison between the two studied groups**

| Variables | UC Cases (n = 50) | Control (n = 25) | Test of Sig. | p-value |
|---|---|---|---|---|
| **CRP (mg/dl)** | | | 201.50* | <0.001* |
| Min. – Max. | 0.60 – 122.0 | 0.60 – 7.0 | | |
| Median (IQR) | 16.0 (3.50 – 30.0) | 2.0 (2.0 – 4.0) | | |
| **ESR 1st hour (ml/h)** | | | U=428.50* | 0.027* |
| Min. – Max. | 6.0 – 48.0 | 11.0 – 17.0 | | |
| Median (IQR) | 16.0 (11.0 – 27.0) | 13.0 (12.0 – 14.0) | | |
| **ESR 2nd hour (ml/h)** | | | U=294.50* | <0.001* |
| Min. – Max. | 14.0 – 110.0 | 17.0 – 25.0 | | |
| Median (IQR) | 33.50 (20.0 – 50.0) | 21.0 (19.0 – 22.0) | | |
| **FC (mg/dl)** | | | U=127.00* | <0.001* |
| Min. – Max. | 25.0 – 1300.0 | 16.0 – 98.0 | | |
| Median (IQR) | 315.0 (88.0 – 750.0) | 44.0 (25.0 – 62.0) | | |
| **Albumin (mg/dl)** | | | U= 283.50* | <0.001* |
| Min. – Max. | 2.80 – 4.10 | 3.80 – 4.20 | | |
| Mean ± SD. | 3.61 ± 0.38 | 3.96 ± 0.13 | | |
| Median (IQR) | 3.70 (3.20 – 4.0) | 4.0 (3.80 – 4.0) | | |
| **Plasma Janus kinase 2** | | | | |
| Min. – Max. | 2655.0 – 16005.0 | 1972.5 – 3318.0 | U = 450.00* | 0.049* |
| Mean ± SD. | 4035.5 ± 3055.7 | 2867.2 ± 385.4 | | |
| Median (IQR) | 3086.8 (2840.5 – 3232.5) | 2952.0 (2758.0 – 3087.0) | | |

*: significant; IQR: Inter quartile range; SD: Standard deviation; t: Student t-test; U: Mann-Whitney test.

There was a significant association between increased inflammatory marker levels (CRP, ESR, and FC) in cases and disease severity. However, plasma JAK2 among cases showed a statistically significant difference between mild and severe cases ($p2 = 0.044$); there was no statistically significant distinction between moderate and severe instances ($p3 = 0.371$) or even between mild and moderate cases ($p1 = 0.191$), **(Table.5 , Fig.1)**.

**Table 5. Comparison of different laboratory markers between the studied UC subgroups**

| Laboratory markers | Mayo score | | | | | | Test of Sig. | p-value |
|---|---|---|---|---|---|---|---|---|
| | Mild (n = 20) | | Moderate (n = 19) | | Severe (n = 11) | | | |
| | No. | % | No. | % | No. | % | | |
| **CRP (mg/dl)** | | | | | | | | |
| Min. – Max. | 0.60 – 12.0 | | 12.0 – 35.0 | | 30.0 – 122.0 | | H= 41.700* | <0.001* |
| Median (IQR) | 3.0 (2.0 – 5.50) | | 19.0 (16.0 – 23.50) | | 60.0 (43.0 – 77.50) | | | |
| **ESR 1st hour (ml/h)** | | | | | | | | |
| Min. – Max. | 6.0 – 25.0 | | 12.0 – 35.0 | | 21.0 – 48.0 | | H= 29.162* | <0.001* |
| Median (IQR) | 10.50 (9.50 – 15.0) | | 17.0 (14.50 – 27.0) | | 30.0 (25.50 – 40.0) | | | |
| Sig. bet. grps. | $p_1 = 0.001^*$, $p_2 < 0.001^*$, $p_3 = 0.021^*$ | | | | | | | |
| **ESR 2nd hour (ml/h)** | | | | | | | | |
| Min. – Max. | 14.0 – 70.0 | | 26.0 – 70.0 | | 40.0 – 110.0 | | | |
| Median (IQR) | 18.0 (16.50 – 26.0) | | 40.0 (30.0 – 47.50) | | 60.0 (55.0 – 80.0) | | | |
| Sig. bet. grps. | $p_1<0.001^*$, $p_2<0.001^*$, $p_3=0.018^*$ | | | | | | | |
| **FC (mg/dl)** | | | | | | | H=37.846* | <0.001* |
| Min. – Max. | 25.0 – 250.0 | | 220.0 – 1100.0 | | 555.0 – 1300.0 | | | |
| Median (IQR) | 75.0 (45.0 – 115.0) | | 450.0 (315.0 –765.0) | | 950.0 (695.0 –960.0) | | | |
| **Albumin (mg/dl)** | | | | | | | | |
| Min. – Max. | 3.50 – 4.10 | | 3.0 – 3.90 | | 2.80 – 3.60 | | H=30.535* | <0.001* |
| Mean ± SD. | 3.93 ± 0.17 | | 3.53 ± 0.30 | | 3.18 ± 0.24 | | | |
| Median (IQR) | 4.0 (3.90 – 4.0) | | 3.60 (3.20 – 3.80) | | 3.20 (3.05 – 3.20) | | | |
| **Plasma Janus kinase 2** | | | | | | | | |
| Min. – Max. | 2820.5 – 3235.0 | | 2746.0 – 4316.5 | | 2655.0 – 16005.0 | | 8.209* | 0.042* |
| Mean ± SD. | 2992.6 ± 147.5 | | 3150.8 ± 352.7 | | 7460.0 ± 5378.2 | | | |
| Median (IQR) | 2954.0 (2835.5 – 3143.5) | | 3117.0 (2935.3 – 3219.3) | | 5745.0 (2867.5–10500.0) | | | |
| Sig. bet. grps. | $p_1=0.191$, $p_2=0.044^*$, $p_3=0.371$ | | | | | | | |

*: significant; IQR: Inter quartile range; SD: Standard deviation; $\chi^2$: Chi square test; FET.$p_1$: comparing between mild and moderate; $p_2$: comparing between mild and severe; $p_3$: comparing between moderate and severe.

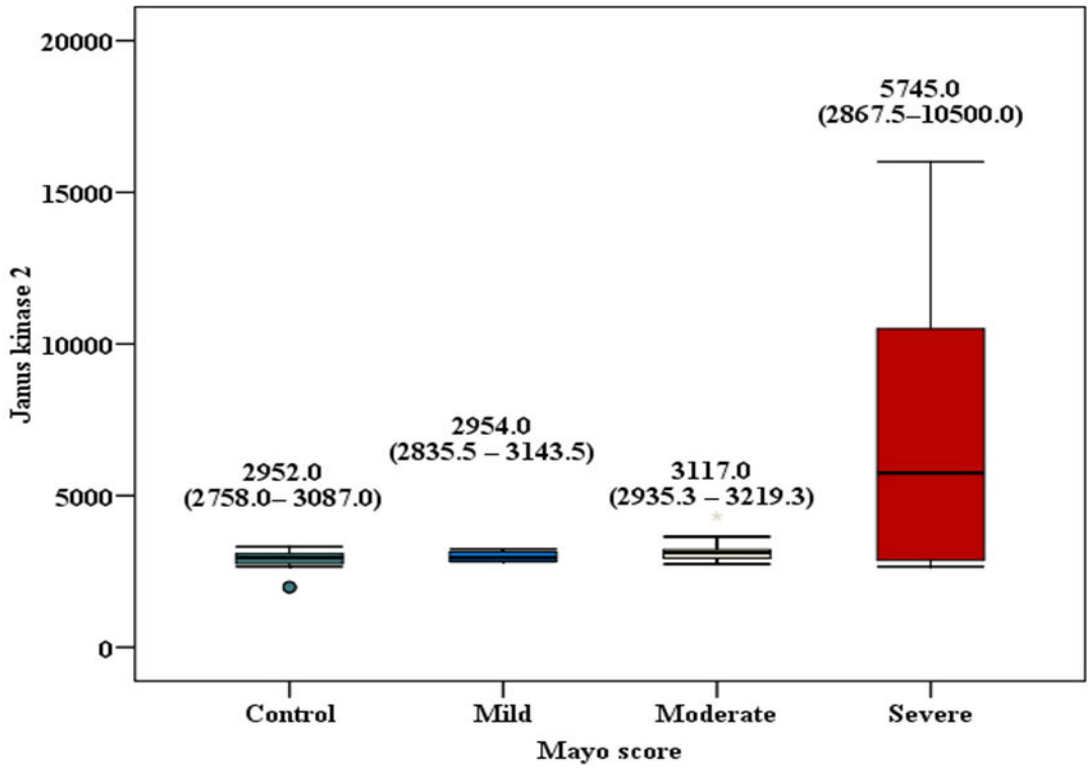


**Fig. 1. Plasma Janus kinase 2 levels in the studied groups. (**The upper and lower lines represent the 75th and 25th percentiles, respectively. The lines in the middle of the boxes stand for the median. The upper and lower error lines designate the 90th and 10th percentiles, respectively. Dots denote data outside the 10th and 90th percentiles.)

Positive correlation between plasma JAK2 and CRP, FC, and UCEIS. In contrast to its negative correlation with albumin (**Table.6**, **Fig.2).**

**Table 6. Correlation between plasma Janus kinase 2 with different parameters in the cases group**

| Variables | Plasma Janus kinase 2 | |
|---|---|---|
| | $r_s$ | P |
| CRP | 0.451* | 0.001* |
| Fecal calprotectin | 0.537* | <0.001* |
| Albumin | -0.449* | 0.001* |
| UCEIS | 0.550* | <0.001* |

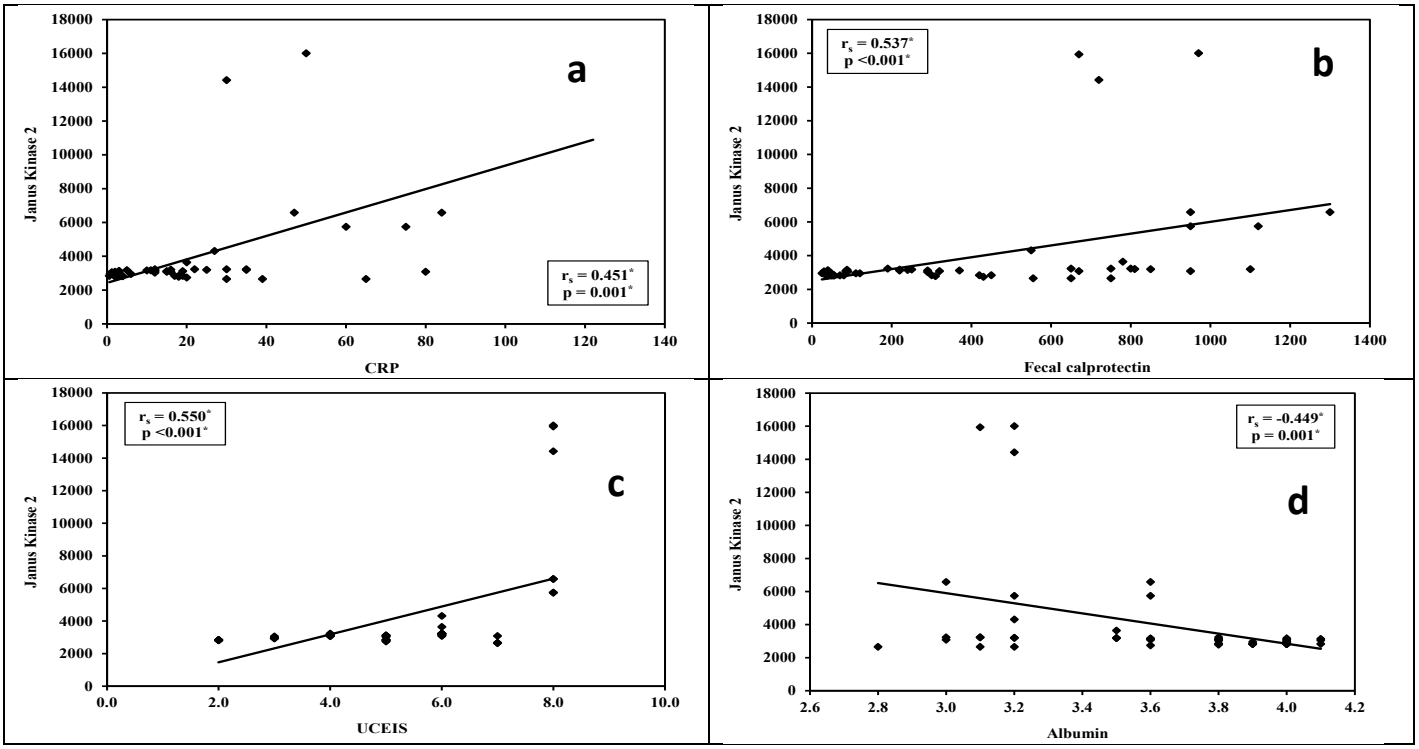


**Fig.2. Correlation between plasma Janus kinase 2 with different parameters in the UC cases group; (a) CRP, (b) FC, (c) UCEIS, (d) albumin**

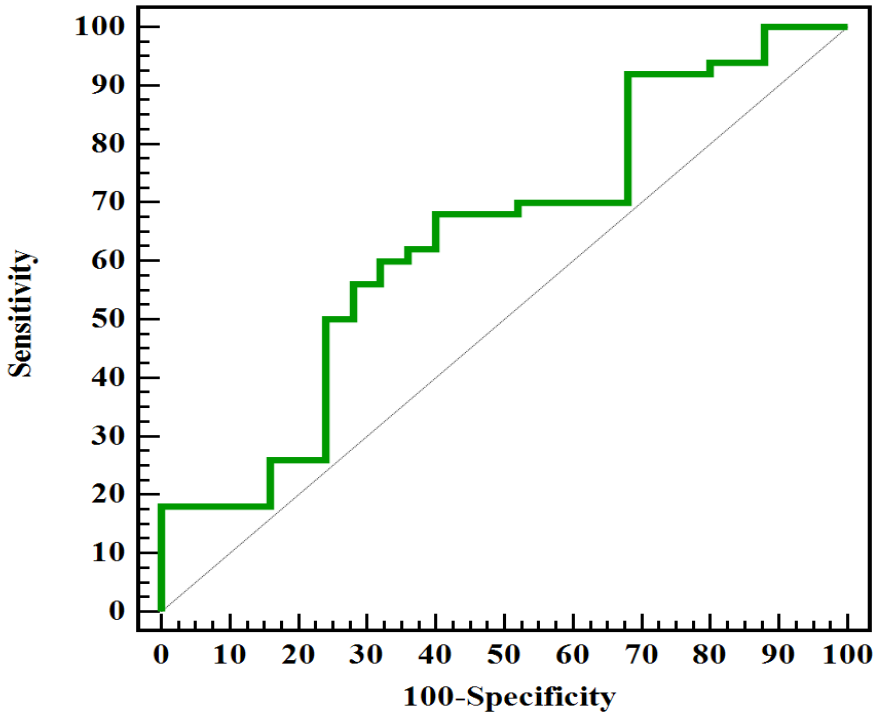


**Fig. 3.ROC curve for plasma Janus kinase 2 to discriminate UC cases from control**

**(Table.7)** describes data plotted in the ROC curve **(Hajian-Tilaki, K. et al., 2013)** revealing that plasma JAK2 can discriminate patients with UC from the control group at a cutoff value of > 2953 pg/ml. The sensitivity, specificity, PPV, and NPV were 68%, 60%, 77.3%, and 48.4%, respectively (p = 0.039) as shown in **(Fig. 3) .**

**Table 7. Diagnostic performance for plasma Janus kinase 2 to discriminate UC cases from control**

| | AUC | P-value | 95% CI | Cutoff# | Sensitivity | Specificity | PPV | NPV |
|---|---|---|---|---|---|---|---|---|
| **Plasma Janus kinase 2** | 0.640 | 0.049* | 0.505 – 0.775 | > 2953 pg/ml | 68.0 | 60.0 | 77.3 | 48.4 |

*: significant; AUC: Area Under a Curve; p value: Probability value; CI: Confidence Intervals; NPV: Negative predictive value; PPV: Positive predictive value; #Cut off was choose according to Youden index.

## Discussion

Inflammatory bowel diseases (IBD) are recognized as a type of chronic inflammatory condition striking the gastrointestinal tract. IBD comprises two basic types: Crohn's disease (CD) and UC. Despite the fact that the exact cause of IBD is unknown, numerous epidemiologic and genetic studies indicate that it results from a complex interaction between environmental variables, immune dysregulation, and genetics **(Eldman et al., 2015)**.

JAK steers the signaling pathway for several proinflammatory cytokines. Many components of the known interleukin-23 receptor (IL23R) pathway, especially IL-23, JAK2, signal transducer and activator of transcription-3 (STAT3), and tyrosine kinase-2 (Tyk2), which play crucial roles in intestinal immune response, have been detected in CD and UC susceptibility loci observed in western GWA studies **(Cohen, 2021)**.

The aim of the study is to determine the significance of circulating plasma JAK2 in assessing disease activity in UC patients.

The present findings revealed that CRP level in group I was dramatically higher than in the control group. There was a substantial statistical difference between the three groups and these findings are consistent with **Rivière et al. (2023)** which showed that CRP increase is an effective surrogate sign for the emergence of deep ulcers in UC, and this is guiding for picking medication of choice for acute severe UC.

According to the current results, patients with UC had significantly higher ESRs for the first and second hours than the control group with a noticeable statistical difference between subgroups with different disease activity, and this is coinciding with **D'Incà and Sturniolo (2023)**. Highlighting that in UC, ESR has been correlated to both the clinical activity and the severity of the illness.

According to the current study, FC level was normal among control group II, but high in group I cases showing appreciable statistical difference; also, there was a considerable statistical difference between the studied groups regarding FC. **Mahdipour et al. (2019)** reported a substantial correlation between the severity of the disease and FC based on both Mayo scores ($p = 0.007$) and Montreal classification ($p = 0.001$).

The current investigations showed that serum albumin level was low in group I cases specially in severe cases than moderate cases, while mild cases had normal levels. In agreement with the current study **Yagi et al. (2021)** exhibited that serum albumin level harbored a substantial positive correlation with

mucosal healing in Japanese UC patients. Making it an informative marker in mucosal healing in people with brief UC duration.

The current study displayed distribution of the subgroups based on assessment of disease activity according to the Mayo score, Mayo sub score, and UCEIS in group I (patients with UC). **Bacsur et al. (2024)** pointed out that combining disease extent and severity in these scoring systems promotes the accuracy of predicting long-term outcomes in UC and recognizes patients who are at risk of aggressive disease progression.

JAK2 phosphorylates STATfamily transcription factors and relays pertinent immune system signals via corresponding cytokine receptors. Immunosuppressive treatment for IBD commonly targets JAK family kinases **(Cardinale et al., 2020)**.

Addressing its function in the immunological response, **Betts et al. (2011)** clarified that several inflammatory cytokines, including IL-6, connect to receptors via JAK2, which eventually phosphorylates signal transducer and activator of transcription 3 (STAT3). JAK2 is a guide for T-cell signaling in response to proinflammatory cytokines such as IL-6, IL-12, and IL-23. These cytokines are a key for the growth and further expansion of Th1 cells, employing IL-12 and Th17 cells, which require IL-6 and IL-23.

In the current study, plasma JAK2 in UC cases was significantly higher than in controls ($p = 0.049$), with mild cases showing a lower increase than moderate cases. Severe cases had the highest level, but no significant differences between mild or moderate cases. JAK2 can reflect severe disease activity, stratifying patients into active and inactive, but there is no clear demarcation between mild, moderate, or severe cases. Plasma JAK2 is positively correlated with UCEIS, fecal calprotectin, and CRP, as opposed to its inverse relationship with albumin; these findings empower its role in assessing disease activity and mucosal healing.

The present findings are coinciding with **Hedl et al. (2016)**, which stated that the rs10758669 risk locus is strictly located in the JAK2 region and was shown to be linked to Crohn's disease and UC. The exact implication of the C risk allele on other immunological processes is uncertain; more intestinal permeability, however, is linked to it.

Also, **Ong Shin et al. (2018)** depicted that in the Chinese population, the JAK2 rs10974944 polymorphism was found to be closely associated with the onset of CD; more research may be done to determine its effect on CD.

In contrast to **Buran et al. (2023)**, who found that most UC cases with thrombotic symptoms did not have a connection to the JAK2 V617F mutation, and the V617F mutation was rare in UC patients.

The main limitation of the study is the small sample size which affected the statistical power to properly use JAK2 as a diagnostic marker or provide clear distinction between different stages of disease activity. therefore, more research on a larger patient cohort is advised to demonstrate its diagnostic utility.

**Conclusion**

In UC, Jannus kinase 2 can be utilized as a marker of disease activity that can reflect mucosal healing as it correlates well with different inflammatory markers.

**List of abbreviations:**

CD: Crohn’s Disease

CRP: C-Reactive protein

ELISA: enzyme-linked immunosorbent assay

ESR: Erythrocyte sedimentation rate.

FC: Fecal Calprotectin

IBD: inflammatory bowel disease

JAK2: Janus kinase

STAT: signal transducers and activators of transcription

UC: Ulcerative Colitis

UCEIS: Ulcerative Colitis Endoscopic Index of Severity.

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

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