ISSN: 2755-6468

Research Article

Open Access Journal of Clinical Pathology Research

Platelet Insights in Ulcerative Colitis: Exploring Novel Perspectives from an Indian Context

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Received: June 10, 2025; **Accepted:** June 20, 2025; **Published:** June 27, 2025

ABSTRACT

Aim: Platelets play a crucial role in exacerbations and thromboembolic complications of Ulcerative Colitis (UC) by contributing to inflammation and coagulation. This study aimed at correlating platelet indices (PI) with UC disease activity.

Methods: A case-control study was conducted including 200 adult UC patients (18–65 years;106 females, 94 males) and 100 controls, incorporating all clinically, endoscopically, and histopathologically diagnosed UC cases. Patients were categorized by disease activity as active (symptomatic) and quiescent (asymptomatic) and additionally classified using Endoscopic Mayo Scores. Platelet (PLT) parameters such as 1) PLT count (PLC), 2) Mean PLT Volume (MPV), 3) Plateletcrit (PCT) 4) PLT Distribution width (PDW); were assessed before treatment initiation and compared across groups using Student's t-test.

Results: Majority of cases (30%) were aged 31–40 years, with a male-to-female ratio of 0.9:1. Of 200 cases, 89 (44%) were in the active phase. The mean values of PLC, MPV, PDW, and PCT in Active UC were 394.5 × 10^9/L, 8.2 fL, 14.0%, and 0.32%, respectively; in Quiescent UC, 228.6 × 10^9/L, 9.1 fL, 16.5%, and 0.21%; and in controls, 196.5 × 10^9/L, 9.1 fL, 16.8%, and 0.17%. Additionally, a significant trend (p10< 0.05) of increased PLC and decreased MPV correlated with higher Endoscopic Mayo scores.

Conclusion: Platelet indices significantly correlated with UC disease activity, highlighting the potential for targeting the inflammation-coagulation interface with prophylactic anti-platelet agents to reduce UC-related mortality and morbidity.

Keywords: Disease Activity, Inflammation, Platelet Indices, Platelets, Ulcerative Colitis

Introduction

Ulcerative colitis (UC) is a chronic inflammatory bowel disease (IBD), primarily affecting the gastrointestinal tract. The immune system essentially contributes to the multifactorial pathogenesis of UC, followed by genetic and environmental factors. A growing number of studies have highlighted the importance of non- immune cells like platelets (PLTs), in UC inflammatory cascades [1].

Apart from their long-established role in hemostasis, PLTs also display proinflammatory properties. Activated PLTs produce and release a variety of proinflammatory mediators that not only influence the inflammatory response but also induce a hypercoagulable state in UC. This in turn initiates a vicious cycle of two interdependent processes, inflammation and coagulation, with platelets being the crucial link between the two [2].

A variety of morphological alterations of platelets have been associated with UC, such as, mean PLT volume (MPV), PLT distribution width (PDW), plateletcrit (PCT) and reactive

Citation: Gargi J Padalkar, Anjali D Amarapurkar, Meghraj Ingale. Platelet Insights in Ulcerative Colitis: Exploring Novel Perspectives from an Indian Context. Open Access J Clin Path Res. 2025. 1(1): 1-6. DOI: doi.org/10.61440/OAJCPR.2025.v1.06

thrombocytosis, which are all linked to PLT activation induced by the ongoing inflammation [3].

An exclusive feature of spontaneous PLT aggregation in the mesenteric circulation has been encountered in UC patients. [4,5] As a consequence, the hypercoagulable state is established in UC which leads to an increased risk of thromboembolic (TE) events.

Evidence of PLT aggregates in colonic biopsies of UC patients in remission and their absence in healthy controls, prove their existence to be independent of disease activity [6]. The prevalence of TE events in UC varies between 1.3% to 6.0%, although it is interesting to note that one third of these events occur during clinical remission, implying a persistent activated state of PLTs in UC [7,8].

Anti–PLT agents have been recognized as secondary prevention therapy for TE events in high-risk cardiovascular diseases [9]. Furthermore, it has been observed that a single intra-colonic administration of clopidogrel significantly reduced the inflammatory markers and resolved exacerbation symptoms of experimentally induced IBD in rats [10].

In addition, the current UC regimes of 5-amino salicylic acid, azathioprine and 6 mercaptopurine are reported to inhibit PLT activation markers and PLT aggregation in IBD patients. [11,12] Moreover, low molecular weight heparin has been widely accepted as a prophylactically and therapeutically beneficial treatment option in UC due to its broad spectrum of anticoagulant and anti-inflammatory properties [13].

Based on this evidence and the high procoagulant and proinflammatory properties of PLTs, they can be considered ambitious targets for future UC remedies.

The current study aimed at documenting and correlating the changes in platelet indices with disease activity in Ulcerative Colitis patients by analyzing quantitative changes in platelet parameters, comparing these indices between Ulcerative Colitis patients and healthy controls, and among different UC disease activity groups.

Methods

This prospective observational case-control study, from Nov 2022–Feb 2024, included adult UC patients of both genders, selected from the Gastroenterology outpatient department of a tertiary care hospital in Mumbai. The study encompassed newly diagnosed UC cases, patients with disease flare-ups, and those in remission attending follow-ups.

Age- and gender-matched 100 healthy blood donors from general community served as the control group.

Ethical clearance was obtained from the Institutional Ethics Committee, and written informed consent was collected from all participating patients.

Inclusion Criteria

Patients above 18 years of age who were clinically, endoscopically, and histopathologically diagnosed with ulcerative colitis were included in this study.

Clinical presentation included mucoid bloody diarrhea with increased stool frequency, abdominal pain, abdominal distention, weight loss, and fever. Patients were categorized by gastroenterologist as having mild, moderate, or severe disease activity; or being in remission, based on their symptoms and the Modified Truelove and Witts criteria [14].

Patients with newly diagnosed UC, active flare-ups, or in remission underwent flexible sigmoidoscopy to evaluate mucosal healing. Endoscopic features indicative of ulcerative colitis comprised erythema, granularity, friability and loss of vascularity. All UC patients were evaluated and categorized by the gastroenterologists according to their Endoscopic Mayo score, a four-point scoring system that classifies disease severity from 0 (inactive) to 3 (severe) [15].

Histologically, UC was diagnosed by an inflammatory infiltrate in the lamina propria, consisting of predominantly lymphocytes, plasma cells, variable eosinophils, neutrophils, and lymphoid aggregates. Other features include basal lymphoplasmacytosis, goblet cell depletion, crypt destruction, cryptitis, crypt abscess and crypt shortening.

Exclusion Criteria

Patients younger than 18 years of age and those without a diagnosis of UC were excluded from this study.

Blood collection

Venous blood samples from the antecubital vein were collected in dipotassium EDTA anticoagulant tubes for all cases, including follow-ups and controls, while for newly diagnosed and flare-up cases, sampling was done before treatment initiation. The samples were analysed using a NABL-accredited 5-part haematology analyser which displayed detailed PLT indices. The tests were performed under strict quality control measures.

Study Groups

The present study compared PLT indices across different disease groups, categorized both clinically and endoscopically:

Comparison of PLT Indices Between UC Disease Activity and Healthy Controls

An initial comparison of PLT indices was performed between UC cases and healthy controls. Subsequently, UC cases, classified as active or quiescent by gastroenterologists using the modified Truelove and Witts criteria, were compared for their platelet indices both within these groups and against healthy controls.

Comparison of PLT Indices Among Different Endoscopic Mayo Scores

During flexible sigmoidoscopy, patients were stratified based on their Endoscopic Mayo Scores. Scores ranged from zero (normal mucosa or inactive disease) to three (severe disease). PLT indices among patients with Mayo scores of 1, 2, and 3 were compared.

The following PLT indices were selected for comparison: PLT count (PLC), Mean PLT Volume (MPV), PLT Distribution Width (PDW), and Plateletcrit (PCT). Normal PLT count ranges from 150-450 x 10^9/L [16]. MPV, an average size measure of PLTs, ranges from 7.2-11.7 fL [17]. PDW, indicating platelet size

variability, ranges from 8.3% to 56.6% [17]. PCT, representing the percentage of blood volume occupied by platelets, has a normal range of 0.22–0.24% [17].

Statistical Analysis

Data was collected and analysed using MS Excel 2021 (Version 16.0) for Windows. Statistical differences between groups were assessed using the unpaired Student's t- test. Results were presented as mean and standard deviation, with a p-value of <0.05 considered statistically significant.

Result

This prospective observational study included 200 adult Ulcerative Colitis (UC) patients and 100 healthy controls. UC diagnosis was confirmed by clinical presentation, endoscopy and biopsy findings.

The majority of UC patients were aged 31–40 years (30%), with a mean age of 37.5 ± 12 years. Females made up 53% of cases. Clinically, 56% had quiescent UC, while 44% had active UC. Among active cases, rectal bleeding was the most common symptom (46%). Endoscopic Mayo Score showed 66% had mild disease (Mayo1), 24% moderate (Mayo2), and 10% severe (Mayo3).

Table 1: Comparison of platelet parameters between UC cases and Control group

Parameters	n	PLC ($x10^9$/L)	PCT (%)	MPV (fL)	PDW (%)
UC	200	302.4 ± 93.2	0.26 ± 0.06	8.7 ± 0.6	15.4 ± 1.5
Control	100	196.5 ± 68.8	0.17 ± 0.06	9.2 ± 0.5	16.8 ± 0.1

Data represented as Mean ± standard deviation

Platelet parameters showed significant differences between UC patients and controls. UC patients exhibited a higher platelet count of 302.4 × 10^9/L and a higher plateletcrit of 0.26% compared to controls, which had a platelet count of 196.5 × 10^9/L and a plateletcrit of 0.17%.

Similarly, UC patients had a lower mean platelet volume of 8.7 fL compared to 9.1 fL in controls and a lower platelet distribution width of 15.4% compared to 16.8% in controls. All differences were statistically significant with $p < 0.05$. (Table 1).

Further comparisons between active UC, quiescent UC, and controls demonstrated a significantly higher platelet count and plateletcrit, and a significantly lower mean platelet volume and platelet distribution width in active UC compared to the other groups.

Table 2: Comparison of platelet parameters between Active UC, Quiescent UC and Control group

Parameters	n	PLC ($x10^9$/L)	PCT (%)	MPV (fL)	PDW (%)
Active UC	89	394.5 ± 49.2	0.32 ± 0.03	8.2 ± 0.2	14.0 ± 1.1
Quiescent UC	111	228.6 ± 37.7	0.21 ± 0.03	9.1 ± 0.5	16.5 ± 0.3
Control	100	196.5 ± 68.8	0.17 ± 0.06	9.2 ± 0.5	16.8 ± 0.1

Data represented as Mean ± standard deviation

Active UC patients had a mean PLC of 394.5 x 10^9/L, mean PCT of 0.32%, mean MPV of 8.2 fL, and mean PDW of 14.0%. Quiescent UC patients had a mean PLC of 228.6 x 10^9/L, mean PCT of 0.21%, mean MPV of 9.1 fL, and mean PDW of 16.5%. Controls had a mean PLC of 196.5 x 10^9/L, mean PCT of 0.17%, mean MPV of 9.2 fL, and mean PDW of 16.8% ($p < 0.05$ for all comparisons) (Table. 2).

Table 3: Comparison of platelet parameters between different Endoscopic Mayo Scores

Parameters	n	PLC ($x10^9$/L)	PCT (%)	MPV (fL)	PDW (%)
Mayo 1	132	248.9 ± 59.2	0.22 ± 0.04	9.0 ± 0.6	16.4 ± 0.4
Mayo 2	48	388.4 ± 20.5	0.32 ± 0.01	8.2 ± 0.1	13.6 ± 0.7
Mayo 3	20	448.8 ± 68.2	0.36 ± 0.04	8.1 ± 0.2	13.1 ± 0.3

Data represented as Mean ± standard deviation

Analysis by Endoscopic Mayo Score revealed similar trends, with higher platelet count and plateletcrit, and lower mean platelet volume and platelet distribution width associated with higher disease severity. For Mayo 3, the values were a mean PLC of 448.8 x 10^9/L, mean PLC of 0.36%, mean MPV of 8.1 fL, and mean PDW of 13.1%.

For Mayo 2, the values were a mean PLC of 388.4 x 10^9/L, mean PCT of 0.32%, mean MPV of 8.2 fL, and mean PDW of 13.6%. For Mayo 1, the values were a mean PLC of 248.9 x 10^9/L, mean PCT of 0.22%, mean MPV of 9.0 fL, and mean PDW of 16.4% ($p < 0.05$ for all comparisons) (Table. 3)

Discussion

The relationship between platelet activity and haemostasis is well established, but the role of platelets in regulating inflammation has been acknowledged recently [3].

Acting as key players in inflammation, platelets utilize their broad array of surface receptors to interact with various immune cells. These interactions are active processes that involve recruiting and modulating leukocytes, such as monocytes and neutrophils; by releasing biologically active substances like growth factors, chemokines, and prostaglandins [18].

Furthermore, platelets actively participate in inflammation through the release of soluble mediators such as cytokines and chemokines. These mediators have diverse effects on the immune system, including influencing cell migration, differentiation, and function. Platelets also interact with vascular endothelial cells, increasing vascular permeability and facilitating the entry of

inflammatory cells and inflammatory factors into tissues, thus exacerbating the inflammatory response [18].

Numerous inflammatory conditions have been associated with morphological alterations in platelet (PLT) parameters [4].

Platelet count (PLT) serves as a crucial marker of inflammation and varies across different diseases. Reactive thrombocytosis, is commonly seen in conditions such as rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), chronic obstructive pulmonary disease (COPD), tuberculosis (TB), and cardiovascular diseases (CVDs) due to increased platelet production in response to inflammation [19-23].

This occurs due to abnormal bone marrow thrombopoiesis driven by inflammatory mediators, alongside a reduced platelet lifespan caused by increased activation and consumption at inflammation sites.

Studies have shown, in sepsis, PLT levels may initially rise as a reaction to infection, but as the condition worsens, excessive platelet consumption leads to thrombocytopenia. [24] In diabetes mellitus, increased platelet count has been reported in a few studies, which contributes to a higher risk of thrombotic complications [25,26]. Some studies have reported an increase in PLT count in early stages of chronic kidney disease (CKD) due to inflammatory stimulation and decrease in advanced stages due to impaired platelet production [27].

Recent research indicates elevated Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) in CVDs, diabetes, and COPD, reflecting increased platelet activity and turnover, which contribute to a higher thrombotic risk. In RA and SLE, few studies have reported that low MPV and increased PDW are associated with ongoing platelet activation and consumption, resulting in smaller, less functional platelets [19-23,25,26]

In TB, some studies have noted reduced MPV and increased PDW, linked to chronic platelet activation and microthrombi formation, correlating with disease severity. [22] Research on sepsis has demonstrated a decrease in MPV and Plateletcrit (PCT), with an initial rise in PCT followed by a drop due to excessive platelet consumption [24]. Elevated PCT has also been reported in COPD and TB [22], indicating increased platelet count and activation due to chronic inflammation [21].

Ulcerative colitis (UC) has emerged as a significant public health concern in India, with an incidence rate of 6.02 per 100,000, which is notably higher than in other Asian countries. Given its increasing prevalence, UC necessitates focused research to better understand its pathophysiology and improve disease management [28].

An intricate link between inflammation and coagulation has been documented in UC.[3] Research indicates that inflammatory mediators and their signaling pathways induce a hypercoagulable state by reducing natural anticoagulant activity and impairing the fibrinolytic system, thereby increasing thrombotic risk. [28] Conversely, coagulation pathways also influence inflammation, with factors like thrombin and tissue factor amplifying the inflammatory response, while anticoagulants such as activated protein C and heparin exert anti-inflammatory effects [29].

This study highlights the significant role of platelets, as reflected in PLT parameters, in understanding ulcerative colitis within an Indian context. In the present study, we conducted a detailed comparison of platelet parameters in UC cases with those reported in other studies.

Studying four platelet parameters i.e PLC, MPV, PCT, and PDW gives a complete picture of how platelets behave in UC. PLC reflects inflammation driven platelet increase, MPV indicates platelet size and activation, PCT represents total platelet mass, and PDW highlights variability in platelet size. Assessing all four offers a clearer understanding of platelet involvement in UC, as a single parameter may not fully reflect the disease's impact.

Ulcerative colitis has a bimodal onset, with peaks between 15-30 and 50-70 years, and an average diagnosis age between 30-40 years [30]. In this study, the mean age of participants was 37.5 ± 12 years, similar to Ozturk ZA et al. (37.6 ± 13.3 years) and Malik Galijašević et al. (34.7 ± 9.8 years) [31,32]. The age range of 18-65 years aligns with findings from Gawrońska B et al. (19-80 years) and Polińska B et al. (18-60 years) [33,34].

The current study found highest platelet count (PLC) in active UC (394.5 × 10^9/L), lower in inactive UC (228.6 × 10^9/L), and lowest in controls (196.5 × 10^9/L), indicating inflammation-driven platelet production. This trend was also reported by Malik Galijašević et al., with PLC values of 336.5 × 10^9/L in active UC, 278.7 × 10^9/L in inactive UC, and 253.2 × 10^9/L in controls [32]. Mean platelet volume (MPV) was lowest in active UC (8.2fL), slightly higher in inactive UC (9.1fL), and highest in controls (9.2fL), indicating larger platelets are consumed in inflammation sites, consistent with Ozturk ZA (8.1fL in active UC). Plateletcrit (PCT) was highest in active UC (0.32%), lower in inactive UC (0.21%), and lowest in controls (0.17%), reflecting increased platelet activity during inflammation, as shown by Malik Galijašević et al. and Ozturk ZA [31,32].

Platelet distribution width (PDW) was lower in active UC (14.0%) compared to inactive UC (16.5%) and controls (16.8%), suggesting reduced platelet size variation in active disease. This was also observed by Ozturk ZA (15.2% in active UC) [31]. Inactive UC cases showed intermediate platelet patterns, suggesting persistent platelet abnormalities despite reduced inflammation.

Overall, inactive UC cases showed platelet patterns that were intermediate between active UC and controls, suggesting that while inflammation decreases in the quiescent phase, platelet abnormalities persist to some extent. In contrast, active UC cases consistently demonstrated the most significant platelet alterations, emphasizing the role of thrombocytosis and platelet activation in ongoing inflammation.

The comparison of PLT parameters across Endoscopic Mayo Score groups revealed a trend: as disease severity increased, PLC and PCT rose, while MPV and PDW decreased. Mayo 3 showed the highest PLC (448.8 × 10^9/L) and PCT (0.36%), and the lowest MPV (8.1 fL) and PDW (13.1%), with statistically significant differences ($p < 0.05$). This is the first study to compare PLT parameters with Endoscopic Mayo Scores.

The present study aligns with existing literature, highlighting platelet parameters, especially PLC and MPV, as valuable markers for UC activity and severity. The lower MPV in active UC supports the hypothesis of platelet consumption at inflammation sites. Elevated PCT in active UC suggests increased platelet activity, while lower PDW points to a more uniform platelet size distribution, likely due to increased platelet turnover during inflammation.

This underscores the role of platelet dynamics in UC pathogenesis, suggesting that platelet parameters can serve as both biomarkers for UC monitoring and potential therapeutic targets. Targeting platelet activity could help manage UC and reduce thromboembolic complications.

While these findings may not directly apply to tertiary care centres, they offer a reference for further studies. Larger, multi-center studies are needed to enhance external validity.

Conclusion

In conclusion, platelets, beyond their traditional role in thrombosis and hemostasis, are key regulators in inflammatory disorders. Altered PLT parameters in active Ulcerative Colitis reflect a reciprocal relationship between coagulation and inflammation, increasing thromboembolic event risk. This study is the first in Indian literature to link UC disease activity with PLT indices, contributing to the growing evidence of platelet involvement in inflammation. The significant correlation between UC disease activity and PLT indices not only establishes these indices as reliable biomarkers for disease activity but also raises the possibility of targeting the inflammation-coagulation interface with anti-platelet agents to reduce the mortality and morbidity associated with UC and its complications.

Funding: NIL.

Conflicts of Interest: There are no conflicts of interest.

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