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Case report

Exploring the Hematological Risks of Diclofenac Sodium: An Experimental Study in Wistar Rats

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This research study investigated hematological alterations associated with diclofenac sodium injection intraperitoneally at a dosage of 15 mg/kg for six weeks in male Albino Wistar rats. Twelve Albino Wistar rats were divided into two groups randomly: a control group and a diclofenac group, six rats in each group. Blood samples were taken from all rats at the end of the experiment, and a hematological evaluation was performed to measure red blood cell indices, white blood cell counts, and platelet indices. Results show a statistically significant decrease in hemoglobin, red blood cell counts, and hematocrit values in rats that received diclofenac compared to controls, indicating that anemia was potentially developed by the diclofenac treatment. Statistically significant increases in mean corpuscular volume, mean corpuscular hemoglobin, and red cell distribution width (RDW) were found, which indicate macrocytic and anisocytic anemias. Though there was no statistically significant difference in total white blood cell counts, there was a distinct change in differential counts consistent with increased neutrophil percentages and lower levels of lymphocytes, suggesting inflammatory or stress-type activity. The platelet count and mean platelet volume were unchanged, but there was reduced platelet distribution width (PDW) and increased plateletcrit (PCT), suggesting possible changes to platelet turnover or activation. These reports indicated that chronic exposure to diclofenac sodium may induce haematotoxicity characterized by anaemia, immune modulation, and changes to platelet parameters. The study highlights the need to use non-steroidal anti-inflammatory drugs cautiously because they have potential systemic impacts on blood profiles.

Introduction

Non-steroidal anti-inflammatory drugs (NSAIDs) are widely used therapeutic agents with potent analgesic, antipyretic, and anti-inflammatory properties. Among them, diclofenac sodium is one of the most commonly prescribed due to its high efficacy and broad spectrum of action in managing pain and inflammation [1]. Diclofenac sodium functions by non-selectively inhibiting cyclooxygenase (COX) enzymes, particularly COX-1 and COX-2, leading to reduced synthesis of prostaglandins, which mediate inflammation and pain [2]. While this mechanism underlies its therapeutic efficacy, it also contributes to a range of side effects, including gastrointestinal, renal, hepatic, and hematological toxicities [3]. Hematological toxicity, though less frequently highlighted compared to gastrointestinal and renal complications, can be a significant adverse outcome of prolonged NSAID usage. Changes in red blood cell (RBC) indices, hemoglobin levels, and white blood cell (WBC) differentials have been observed following chronic NSAID exposure [4].

Diclofenac's potential to induce anemia has been demonstrated in multiple experimental studies. These effects are thought to be due to bone marrow suppression, hemolysis, or gastrointestinal bleeding induced by mucosal damage [5]. Additionally, NSAIDs like diclofenac can cause oxidative damage to erythrocyte membranes, leading to premature destruction of RBCs [6]. In animal models, diclofenac administration has been associated with a marked reduction in hemoglobin levels, RBC count, and hematocrit, suggesting the onset of anemia. These hematological alterations may also reflect direct toxicity to hematopoietic tissues or indirect effects such as increased oxidative stress [7]. Moreover, diclofenac has been shown to alter red blood cell indices such as mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and red cell distribution width (RDW). These changes indicate disruptions in erythropoiesis or the presence of mixed-type anemia [8].

The impact of diclofenac on leukocytes has also been reported. While total WBC counts may remain unchanged, differential counts often show neutrophilia and lymphocytopenia, suggestive of systemic inflammation or stress responses [9]. These shifts may signal immune modulation or subclinical tissue injury associated with chronic NSAID use [10].

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Platelet parameters such as platelet distribution width (PDW), plateletcrit (PCT), and mean platelet volume (MPV) may also be affected by diclofenac administration. These changes might arise from megakaryocyte suppression, platelet activation, or altered turnover, which could influence clotting capacity [11]. In addition, diclofenac affects liver function, which in turn can impact hematopoiesis due to altered synthesis of coagulation factors and reduced metabolism of toxins that affect the bone marrow [12]. The liver's central role in iron metabolism also implicates it in drug-induced anemia [13]. Studies have shown that diclofenac can decrease erythropoietin levels, a hormone crucial for RBC production. This suppression may result from renal toxicity or feedback inhibition due to tissue hypoxia; reduced erythropoietin disrupts RBC maturation and leads to anemia [14].

Several researchers have observed elevated RDW values following diclofenac administration, indicating increased variability in RBC size, which is a hallmark of anisocytosis. This may arise from oxidative damage or stress erythropoiesis [15]. Moreover, chronic diclofenac use has been linked to bone marrow suppression, characterized by a reduction in hematopoietic cell lines. Histological studies in animal models support the evidence of marrow toxicity [16]. Some studies have reported conflicting findings, with no significant hematological changes observed after diclofenac exposure. These discrepancies may be due to variations in species, dosing regimens, administration routes, and experimental duration [17]. This study aimed to investigate the hematological effects of intraperitoneally administered diclofenac sodium (15 mg/kg) in Wistar rats over six weeks. By examining red and white blood cell indices and platelet parameters, this study seeks to clarify the scope and mechanism of diclofenac-induced hematological changes.

Materials and Methods

Animal Model

This study utilized 12 adult male Albino Wistar rats (Sprague Dawley) with a weight range of 200-250g. These rats were accommodated in the controlled laboratory environment at the Pharmacology Department, University of Benghazi. The rats were housed in plastic cages, with a temperature maintained at 25°C and a 12-hour light/dark cycle. They had free access to a standard rat diet and tap water. All experiments were conducted between 9:00 a.m. and 2:00 p.m.

Experimental Design

The rats were randomly assigned to two groups (6 rats per group): the control group and the diclofenac-treated group. The diclofenac-treated group received an intraperitoneal (IP) injection of diclofenac sodium at a dose of 15 mg/kg, while the control group was administered an equal volume of saline solution. Both treatments were administered once daily for a period of 6 weeks.

Blood Collection and Hematological Analysis

At the end of the treatment period, blood samples were collected by decapitation. Blood was drawn directly into EDTA-coated tubes to prevent coagulation for hematological analysis. The samples were dispatched to the Al-Akeed Medical Laboratory for evaluation. Hematological parameters were assessed using an automated hematology analyzer (Mindray BC-5150).

Statistical Analysis

Data were analyzed using SPSS software (version 23). Descriptive statistics were calculated for all groups, and differences between groups were assessed using the independent T-test. Results were expressed as mean ± standard error of mean (SEM). A p-value of less than 0.05 was considered statistically significant.

Results

Administration of diclofenac (15 mg/kg) resulted in a marked reduction in hemoglobin (Hb) parameter levels compared to the control group. The mean Hb value in the diclofenac-treated rats was 5.53 ± 0.176, while the control group exhibited a mean Hb level of 14.41 ± 0.319. Statistical analysis revealed that this difference was highly significant ($p < 0.001$), indicating that diclofenac administration significantly decreases Hb parameter levels (Figure 1).

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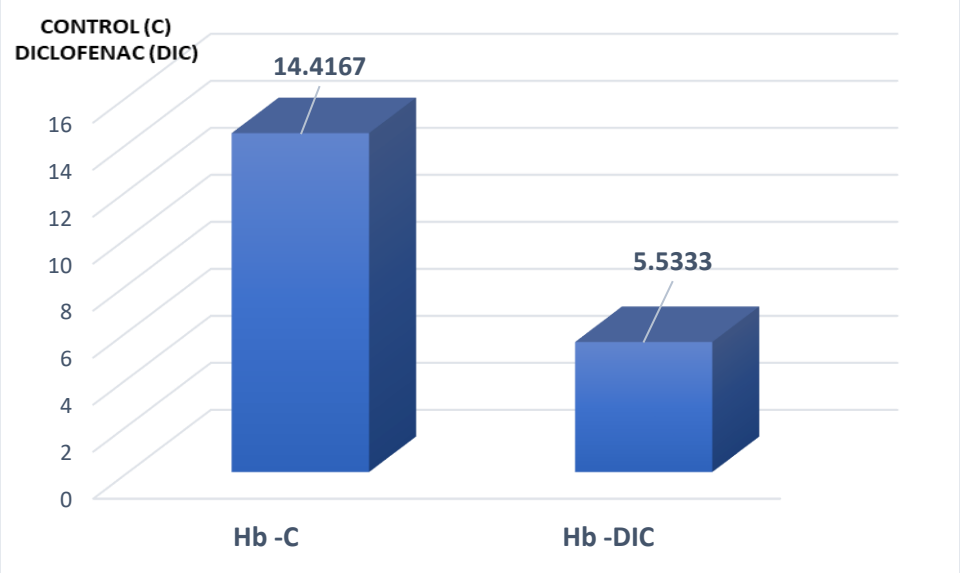

Figure 1: Effect of Diclofenac sodium (15mg/kg) on hemoglobin (Hb) parameter levels. Values were expressed as mean±standard error of mean.

Diclofenac administration significantly affected several red blood cell (RBC) parameters compared to the control group. RBC count and hematocrit (HTC) were notably reduced in the diclofenac group (2.51±0.027 and 15.08±0.524, respectively) compared to controls (7.43±0.267 and 36.73±1.014), showing a very significant difference ($p < 0.001$). Mean corpuscular volume (MCV) increased significantly from 49.31±0.469 to 77.66±0.721 ($p < 0.001$). Mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) were also significantly altered, increasing from 19.73±0.28 to 22.05±0.895 ($p < 0.05$) and decreasing from 39.65±0.212 to 36.66±1.004 ($p < 0.05$), respectively. Red cell distribution width–coefficient of variation (RDW-CV) and standard deviation (RDW-SD) increased significantly from 16.15±0.204 to 26.75±2.547 ($p < 0.01$) and 29.8±0.481 to 56.71±5.465 ($p < 0.01$), respectively (Figure 2).

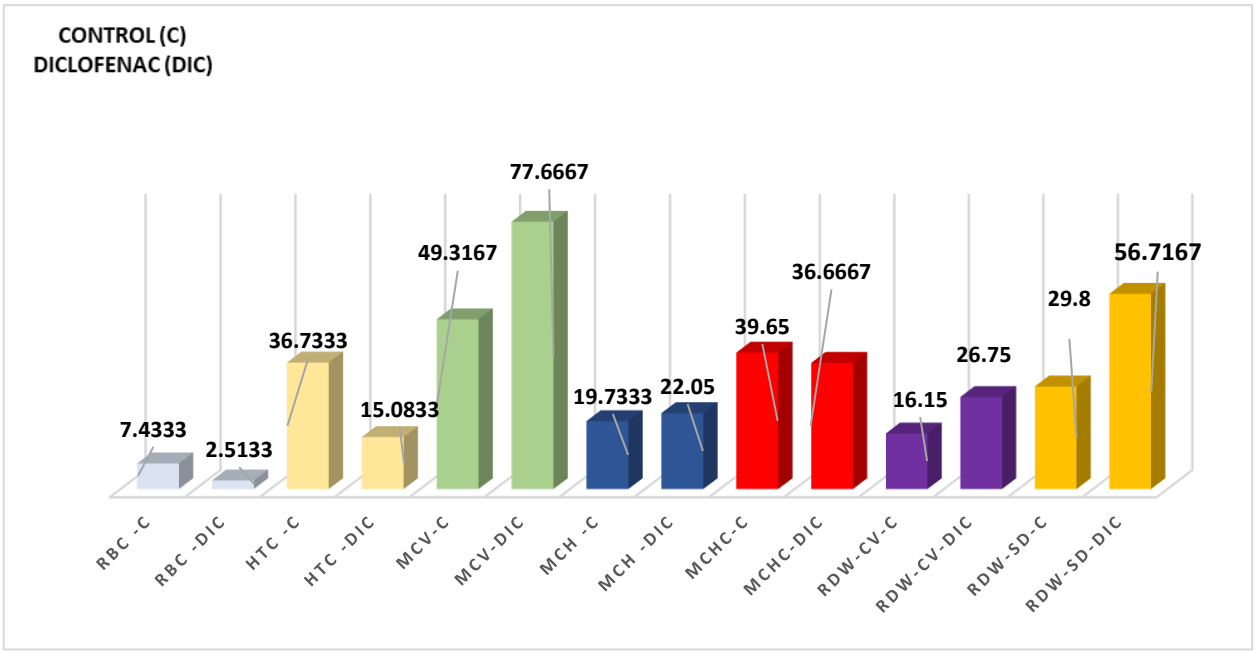

Figure 2: Effect of Diclofenac sodium (15mg/kg) on red blood cell count (RBC), hematocrit (HTC), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red cell distribution width-coefficient of variation (RDW-CV), and red cell distribution width-standard deviation (RDW-SD). Values were expressed as mean±standard error of mean.

Diclofenac treatment did not significantly alter total white blood cell (WBC) count, with similar means observed between the control group (7.83±0.396) and the treated group (7.68±0.368). However, a highly significant increase in neutrophil percentage was observed in the diclofenac group (51.08±1.959) compared to

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the control group (38.13±1.57) with a p-value < 0.001. Lymphocyte percentage significantly decreased from 51.35±1.025 in controls to 39±1.932 in the treated group (p < 0.001), as shown in Figure 3.

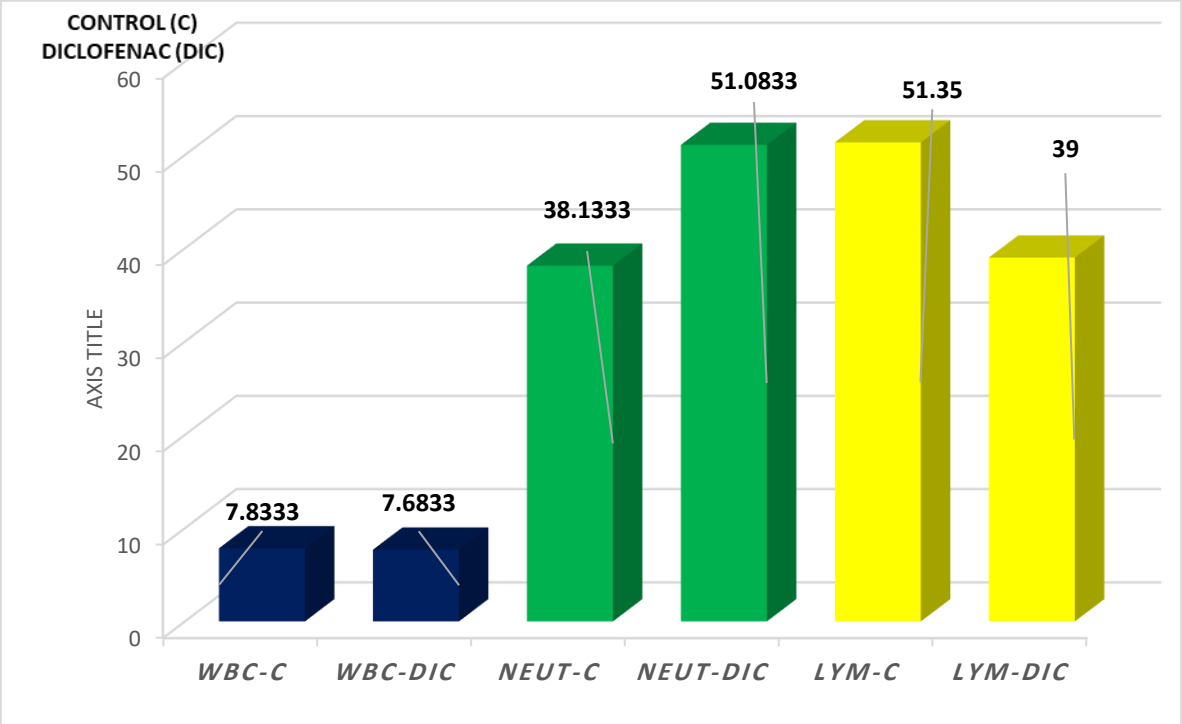

Figure 3: Effect of Diclofenac sodium (15mg/kg) on total white blood cell count (WBC), neutrophil (NEUT) percentage lymphocyte (LYM) percentage. Values were expressed as mean±standard error mean.

Diclofenac administration did not significantly affect total platelet count, with platelet means of 723.83±18.73 (control) and 748.33±4.773 (treated) as shown in Figure 4.

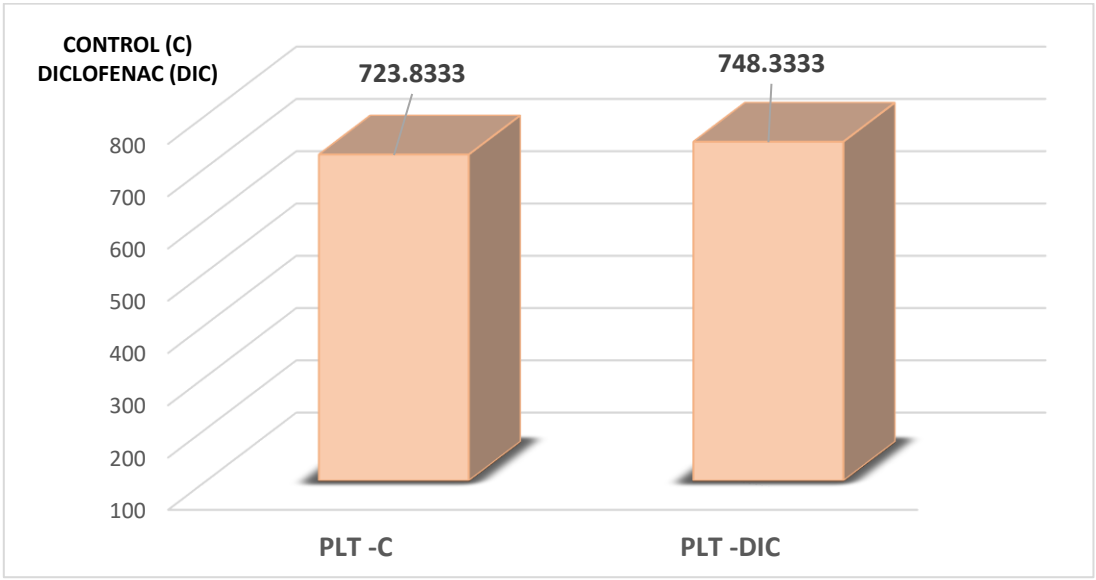

Figure 4: Effect of Diclofenac sodium (15mg/kg) on total platelet count (PLT). Values were expressed as mean±standard error of mean.

Diclofenac administration did not significantly affect MPV values, with MPV means of 7.03±0.128 (control) and 7.78±0.047 (treated). However, platelet distribution width (PDW) significantly decreased in the treated group (12.48±0.047) compared to the control (15.33±0.12) with a p-value < 0.01. Furthermore, plateletcrit (PCT) showed a very significant increase from 0.47±0.008 in the control group to 0.58±0.006 in the diclofenac group (p < 0.001), as shown in Figure 5.

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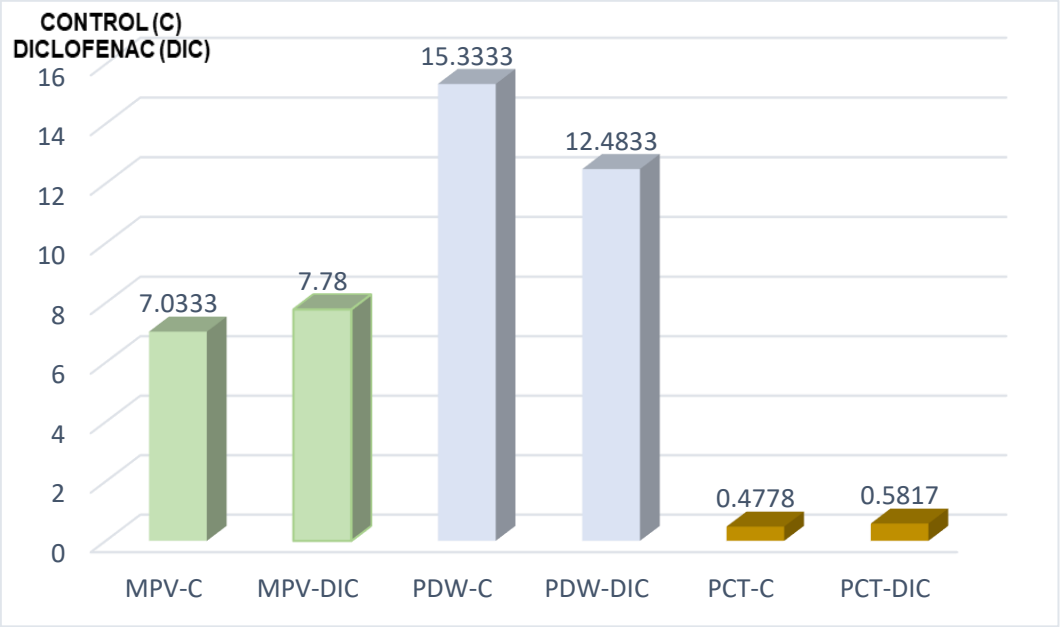

Figure 5: Effect of Diclofenac sodium (15mg/kg) on platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT). Values were expressed as mean±standard error of mean.

Discussion

The main aim of the study was to assess the effects of diclofenac sodium given intraperitoneally at a dose of 15 mg/kg for six weeks in rats on hematological parameters. The results indicate significant differences in several parameters, indicating possible hematotoxic effects of diclofenac sodium. The present study revealed a significant ($p < 0.001$) decrease in hemoglobin (HB) levels in the diclofenac-treated rats, indicating the presence of anemia, primarily due to a reduction in erythropoiesis for RBC formation or destruction of RBC. Our results are similar to previous studies [18,19]. In line with our results, diclofenac has been shown to cause oxidative damage to erythrocytes, leading to hemolysis and decreased RBC [20]. In our study, the RBC count and Hematocrit (HTC) values were both significantly ($p < 0.001$) lower in treated rats compared to controls. These results support the contention that diclofenac disrupts erythropoiesis or enhances RBC destruction. In contrast, other findings reported no significant change in HB or RBC count following diclofenac exposure and suggested that dose, route, and duration of exposure were important factors in the hematological impact [21].

The significant increases in MCV ($p < 0.001$) after diclofenac treatment suggest macrocytic anemia may be present, which can occur due to impaired DNA synthesis of erythroid precursors. MCH also increased significantly ($p < 0.05$) while MCHC decreased ($p < 0.05$), indicating a lower concentration of hemoglobin per unit volume of RBCs. These findings are consistent with those of earlier research [22]. Conversely, some researchers observed no significant changes in the MCH and MCV levels after diclofenac treatment, and suggested strain differences or compensatory effects could modulate the hematological response [23].

RDW-CV and RDW-SD both significantly ($p < 0.01$) increased across our samples, suggesting that anisocytosis and variability in RBC size may increase. An elevated RDW is typically a result of mixed anemia or regenerating responses [24], and our findings are in agreement with those reporting similar changes after diclofenac exposure [25]. Despite total WBC count showing no statistical difference, the neutrophil percentage increased ($p < 0.001$), alongside a decrease in lymphocytes ($p < 0.001$). These changes conform to an acute inflammatory or stress response. This response may relate to tissue injury or immune challenge upon impact [26,27]. In contrast, other studies found no significant differences in WBC differentials, suggesting immune variability in response to NSAIDs [28].

There were no significant differences in platelet count or MPV following diclofenac administration, suggesting no apparent impairment of primary platelet production. However, PDW was significantly decreased ($p< 0.01$), and plateletcrit (PCT) was increased ($p < 0.001$), which may indicate altered platelet activation or turnover. Decreased PDW may reflect reduced anisocytosis of platelets, possibly due to changes in megakaryocyte differentiation [29]. While some researchers noted changes in platelet aggregation post-diclofenac exposure [30], others reported no significant alterations in platelet indices [31]. These findings may suggest a dose-dependent or systemic modulation of platelet-related parameters.

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Conclusion

The present study shows that administration of diclofenac sodium intraperitoneally at a dose of 15 mg/kg for 6 weeks produces significant blood changes, most importantly anemia through the decreased HB, RBC counts, and decreased HTC, changes in the leukogram characterizing potential inflammation, and changes in platelet function. These changes are consistent with previous reports and indicate the potential for hematotoxicity after prolonged NSAID administration. It is advised that additional toxicological research be done on diclofenac sodium in various animal species to establish the drug's safe therapeutic dosages in those species.

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الملخص

هدفت هذه الدراسة إلى التحقيق في التأثيرات الدموية لدواء ديكلوفيناك الصوديوم عند إعطائه داخل الصفاق بجرعة 15 ملغم/كغم لمدة ستة أسابيع في ذكور جرذان ويستر البيضاء. تم تقسيم 12 جرذًا عشوائيًا إلى مجموعتين: مجموعة ضابطة ومجموعة معالجة بالديكلوفيناك، بواقع ستة حيوانات في كل مجموعة. بعد فترة المعالجة، تم جمع عينات الدم وإجراء تحليل دموي شامل لتقييم مجموعة من المعايير، بما في ذلك مؤشرات خلايا الدم الحمراء، وعدد خلايا الدم البيضاء، ومؤشرات الصفائح الدموية. أظهرت النتائج انخفاضًا كبيرًا في تركيز الهيموغلوبين وعدد خلايا الدم الحمراء وقيم الهيماتوكريت في مجموعة الديكلوفيناك مقارنةً بالمجموعة الضابطة، مما يشير إلى تطور فقر الدم، ويُحتمل أن يكون ذلك نتيجة ضعف تكوين خلايا الدم الحمراء أو زيادة تدميرها. كما لوحظت زيادات ملحوظة في الحجم الكروي الوسيط (MCV) والهيموغلوبين الكروي الوسيط (MCH) وتوزيع حجم خلايا الدم الحمراء(RDW-CV) و(RDW-SD)، مما يشير إلى وجود فقر دم كبير الحجم ومتفاوت الحجم. على الرغم من أن العدد الكلي لخلايا الدم البيضاء لم يختلف بشكل ملحوظ، إلا أنه تم تسجيل تغير ملحوظ في نسب الخلايا التفريقية، بزيادة في نسبة العدلات ونقص في اللمفاويات، مما يدل على استجابة التهابية أو مرتبطة بالإجهاد. على الرغملم تتغير أعداد الصفائح الدموية ومتوسط حجمها، إلا أن توزيع حجم الصفائح (PDW) انخفض، بينما زادت نسبة الصفائح الدموية (PCT) بشكل كبير، مما يشير إلى تغيرات محتملة في تنشيط الصفائح أو دورانها. تشير هذه النتائج إلى أن التعرض الطويل الأمد للديكلوفيناك الصوديوم قد يسبب سمية دموية تتميز بفقر الدم وتعديلات مناعية وتغيرات في معايير الصفائح الدموية. وتبرز الدراسة أهمية الحذر عند استخدام مضادات الالتهاب غير الستيرويدية بسبب تأثيراتها الجهازية المحتملة على مكونات الدم.

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