P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)

https://doi.org/10.71375/djvs.2026.043010

# Pathological and Ameliorative Effect of Curcumin Against Toxic Effect of Diclofenac Sodium In The Rabbits

Heba Saleh Shaheed and Ghusoon A.A. Alneamah

[1,2]Department of Pathology and Poultry Diseases, Collage of Veterinary Medicine, Al-Qasim Green University, Babylon Iraq

Corresponding Email: Hebasaleh@vet.uoqasim.edu.iq

ORCID: https://orcid.org/0000-0001-7661-8024

**Important dates:** **Received:** 09-July-2026; **Accepted:** 11- August-2026; **Published**: September-2026

**Abstract:**

**Background:** Diclofenac sodium is a commonly prescribed non-steroidal anti-inflammatory drug (NSAID), valued for its analgesic and anti-inflammatory efficacy. However, its long-term or excessive use has been associated with adverse organ effects, most notably hepatotoxicity, likely mediated through oxidative stress and inflammatory pathways. Curcumin, a natural polyphenolic compound isolated from *Curcuma longa*, has attracted growing scientific interest for its antioxidant, anti-inflammatory, and hepatoprotective properties, positioning it as a promising candidate for mitigating drug-induced tissue injury.

**Aim:** The present study was designed to assess the gross and histopathological alterations induced by diclofenac sodium administration and to evaluate the potential protective role of curcumin against diclofenac-induced toxicity in a rabbit model.

**Results:** Twenty healthy rabbits were randomly divide into four equal groups: control group, diclofenac-treated group, curcumin-treated group, and diclofenac plus curcumin-treated groups. Diclofenac sodium was administered as a single intramuscular dose of 10 mg/kg.bw, while curcumin extract was administered orally at 50 mg/kg daily for five consecutive days beginning on the day of diclofenac administration. Clinical observations and gross pathological examinations of the liver, kidneys, and spleen were performed at the end of the experiment. Diclofenac administration induced marked gross hepatic lesions, including necrosis, hyperemia, and swelling, whereas no obvious pathological alterations were observed in the kidneys or spleen. Curcumin alone produced no detectable lesions, while co-administration of curcumin with diclofenac reduced the severity of hepatic changes. The findings indicate that diclofenac sodium primarily induces hepatic damage in rabbits, while curcumin exhibits a potential protective effect against diclofenac-induced liver injury. These results support the possible use of curcumin as a natural ameliorative agent to reduce NSAID-associated hepatotoxicity.

**Keywords:** Diclofenac, Curcumin, Hepatotoxicity, Nephrotoxicity, Histopathology

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010



## Introduction:

Diclofenac sodium is a non-steroidal anti-inflammatory drug (NSAID) widely used in both human and veterinary medicine for the management of pain, inflammation, and fever. Its therapeutic action is primarily mediated through inhibition of cyclooxygenase (COX) enzymes, which reduces the synthesis of prostaglandins involved in the inflammatory cascade (Dronik and Stasevych, 2025). Despite its clinical efficacy, diclofenac has been associated with adverse effects on several organs, particularly the liver and kidneys, especially when administered at high doses or over prolonged periods (Gan, 2010). Drug induced hepatotoxicity remains a significant concern in both clinical and experimental settings. As diclofenac is metabolized predominantly in the liver, its reactive metabolites can trigger oxidative stress, lipid peroxidation, and subsequent cellular injury. Experimental studies have documented hepatic necrosis, congestion, and inflammatory cell infiltration, along with elevated liver enzyme levels, following diclofenac administration in laboratory animals (Gómez-Lechón *et al.*, 2003). In veterinary practice, diclofenac has additionally been employed in ophthalmic formulations and for the treatment of inflammatory conditions across various animal species. Nevertheless, prolonged use or overdose can result in serious adverse effects affecting the gastrointestinal tract, liver, and kidneys, driven largely by prostaglandin inhibition and oxidative stress mechanisms (Parolini, 2020). Curcumin, the principal bioactive constituent of turmeric (*Curcuma longa*), has drawn considerable scientific attention for its antioxidant, anti-inflammatory, and hepatoprotective properties. It has been shown to scavenge reactive oxygen species, enhance the activity of endogenous antioxidant enzymes, and attenuate tissue damage induced by a range of toxic agents (Hewlings and Kalman, 2017). The liver, kidneys, and spleen play essential roles in metabolism, excretion, detoxification, and immune regulation. Diclofenac toxicity may compromise these organs through oxidative and inflammatory mechanisms, assessment of the resulting gross pathological and histopathological changes can offer valuable insight into organ-specific susceptibility, as well as the potential protective contribution of curcumin (Harirforoosh *et al.*, 2013). Therefore, the present research peruse to evaluate the gross and histopathological effects of diclofenac sodium administration on the liver, kidneys, and spleen in rabbits, and to investigate the potential protective role of curcumin in alleviation diclofenac-induced organ damage

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

## Materials and Methods:

### 1. Experimental Animals:

A total of (20) healthy adult rabbits were used in this study. The animals were housed under standard laboratory conditions with free access to food and water throughout the experimental period. Prior to the start of the experiment, rabbits were allowed a period of acclimatization and were monitored daily for general health status and clinical signs.

### 2. Experimental design:

A total of 20 rabbits were used. The rabbits were randomly divided into four equal groups (n = 5 per group) as follows:

Group I (Control Group): Received no treatment and served as the negative control.

Group II (Diclofenac Group): Received a single intramuscular injection of diclofenac (Diclopara®) at a dose of 10 mg/kg body weight.

The administered volume was approximately 0.8 mL per rabbit.

Group III (Curcumin Group): Received curcumin extract orally at a dose of 50 mg/kg body weight/day for 5 consecutive days.

Group IV (Diclofenac + Curcumin Group): Received a single intramuscular injection of diclofenac (Diclopara®) at 10 mg/kg body weight (approximately 0.8 mL per rabbit). In addition, curcumin extract was administered orally at 50 mg/kg body weight/day for 5 consecutive days, beginning on the same day as diclofenac administration.

### 3. Drug Administration:

Diclofenac sodium (Diclopara® injection) was administered as a single intramuscular (IM) dose to induce toxicity. Curcumin extract was administered orally once daily for five consecutive days using the calculated dose based on body weight.

### 4. Materials Used:

1. Diclopara® Injection (Diclofenac Sodium) used at a dose of 10 mg/kg body weight , administered intramuscularly as a single dose.

2. Curcumin Extract used at a dose of 50 mg/kg body weight/day, administered orally for 5 consecutive days. As showed in the table:

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

| No. | Group name | No. of Rabbits | Treatment |
|---|---|---|---|
| 1 | Control | 5 | No treatment |
| 2 | Diclofenac | 5 | Diclopara® 10 mg/kg IM (single dose) |
| 3 | Curcumin | 5 | Curcumin 50 mg/kg orally for 5 days |
| 4 | Diclofenac + Curcumin | 5 | Diclopara® 10 mg/kg IM + Curcumin 50 mg/kg orally for 5 days |

**Ethical approval**

All experimental procedures involving animals in this study were conducted in accordance with the guidelines for the care and use of laboratory animals and were approved by the Institutional Animal Care and Use Committee (IACUC) / Research Ethics Committee in College of Veterinary Medicine, University of Al-Qasim green university.

**Results:**

**Gross pathological observation:**

Gross pathological examination revealed no visible lesions or abnormalities in the kidneys of rabbits from all experimental groups. Kidney size, shape, color, and external appearance were comparable to those observed in the control group (figure 1). In contrast, the liver of rabbits receiving diclofenac exhibited obvious pathological alterations. Gross examination revealed areas of necrotic lesions accompanied by hyperemia and swelling. The affected livers appeared enlarged and congested compared with those of the control group. These findings were most evident in the diclofenac-treated group.(figure2). Rabbits treated with curcumin alone showed no detectable gross abnormalities in the liver, kidneys, or spleen. In the diclofenac plus curcumin group, liver lesions appeared less pronounced than those observed in the diclofenac-only group, suggesting a possible protective effect of curcumin against diclofenac-induced hepatic injury. No visible gross lesions were observed in the spleen of any experimental group. Splenic size, consistency, and coloration remained within normal limits throughout the study period(figure3).

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

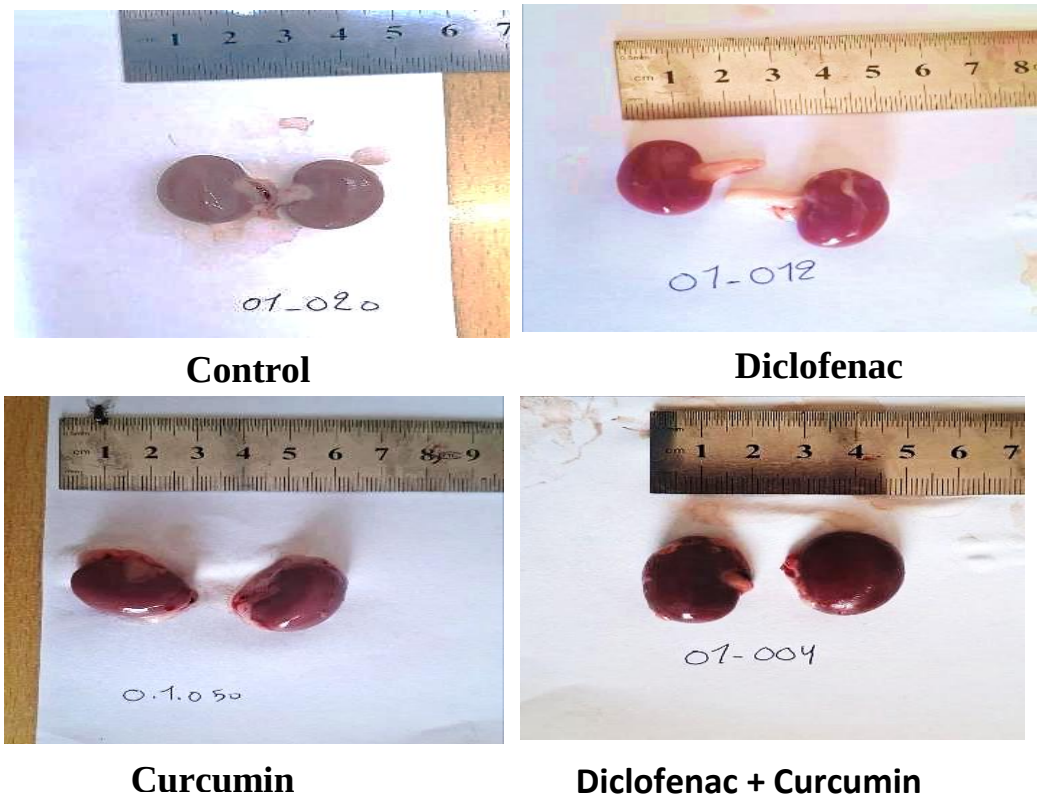



**Figure (1):** Gross image of rabbit kidney showing the different size of the kidney between groups.

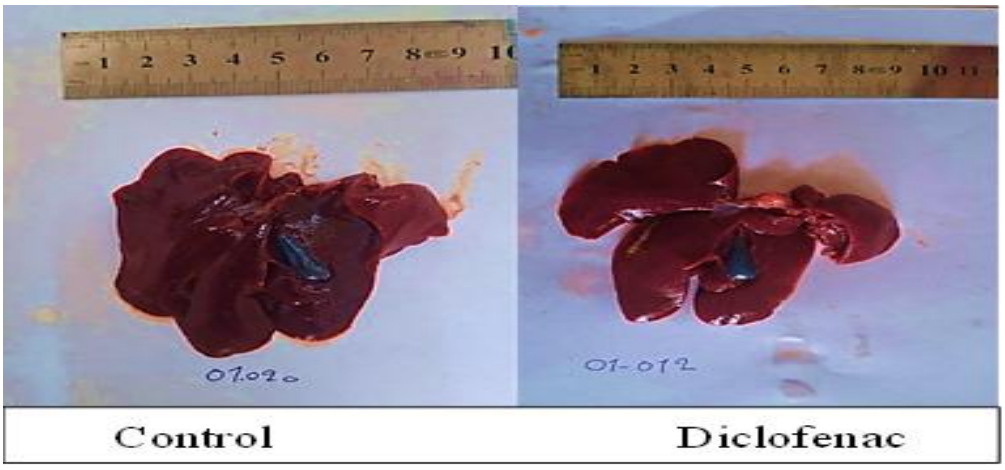



**Figure (2):** Gross image of rabbit liver showing enlargement and congestion in the liver in group treated with Diclofenac.

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

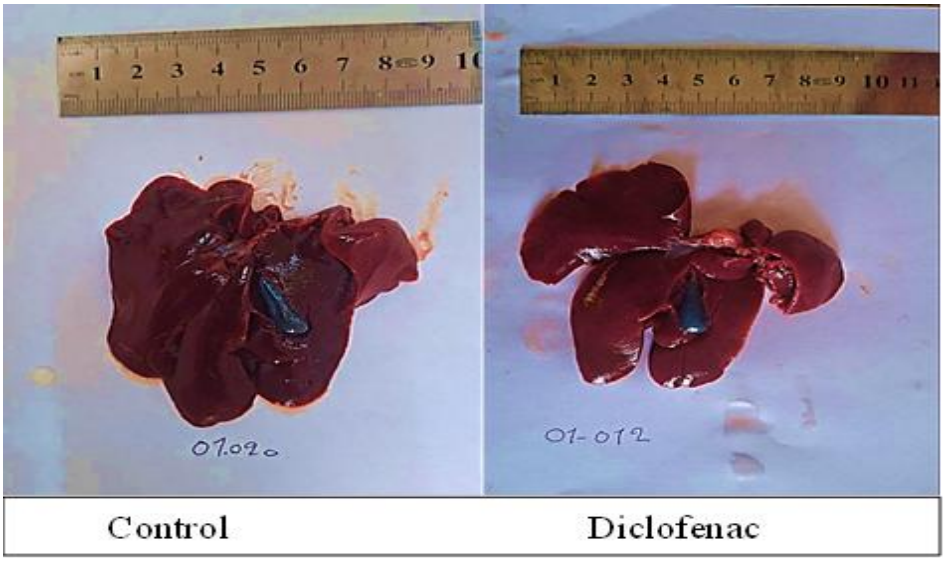



**Figure (3):** gross image of rabbit spleen showing no differentiation in the splenic size.

## Histopathological examination:

Histopathological examination of the liver, kidney, and spleen was performed to assess the hepatic, renal, and splenic toxicity of diclofenac and the potential protective and immunomodulatory effects of curcumin in a rabbit model. Liver sections for control group showed normal hepatic architectures (figure 4:I) . The diclofenac-treated group revealed marked hepatocellular vacuolation, congestion of the central vein, and dilation with congestion of the hepatic sinusoids, indicating diclofenac-induced hepatotoxic injury (figure 4:II). In contrast, the curcumin-treated group showed comparatively milder alterations(figure4:III). One section demonstrated only mild dilation of the hepatic sinusoids accompanied by increased Kupffer cell infiltration, while another section from the group treated with curcumin and diclofenac showed marked mononuclear cell aggregation adjacent to the central vein along with activated Kupffer cells, suggesting an active immune/inflammatory response rather than degenerative injury(figure 4:IV).The kidney section for control group displayed normal renal histological architecture (figure5:I), The diclofenac-treated group exhibited pronounced nephrotoxic changes, including enlargement of the glomerular tufts due to vacuolation of glomerular cells, sloughing of epithelial cells lining the renal tubules, interstitial hemorrhage, and inflammatory cell infiltration, consistent with acute drug-induced renal injury(figure 5:II).The curcumin-treated group showed no clear renal lesion (Figure 5:III), indicating a favorable renal tolerability profile. Notably, the group co-treated with diclofenac and curcumin showed regeneration of the glomerular tufts and renal tubules (Figure5: IV), suggesting that curcumin exerts a nephroprotective, reparative effect against diclofenac-induced renal damage. The section of spleen for control group showed a normal red and white pulp architecture (Figure 6:I) .The diclofenac-treated group displayed congestion of the red pulp together with depletion of the white pulp (Figure 6:II), reflecting a diclofenac-induced

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

immunosuppressive and vascular effect on splenic tissue. By comparison, curcumin-treated groups exhibited a stimulatory effect on splenic lymphoid tissue: one section showed mild white pulp hyperplasia (Figure 6:III), while in group treated with curcumin and diclofenac showed marked white pulp hyperplasia attributable to increased lymphocyte proliferation (Figure 6: IV).

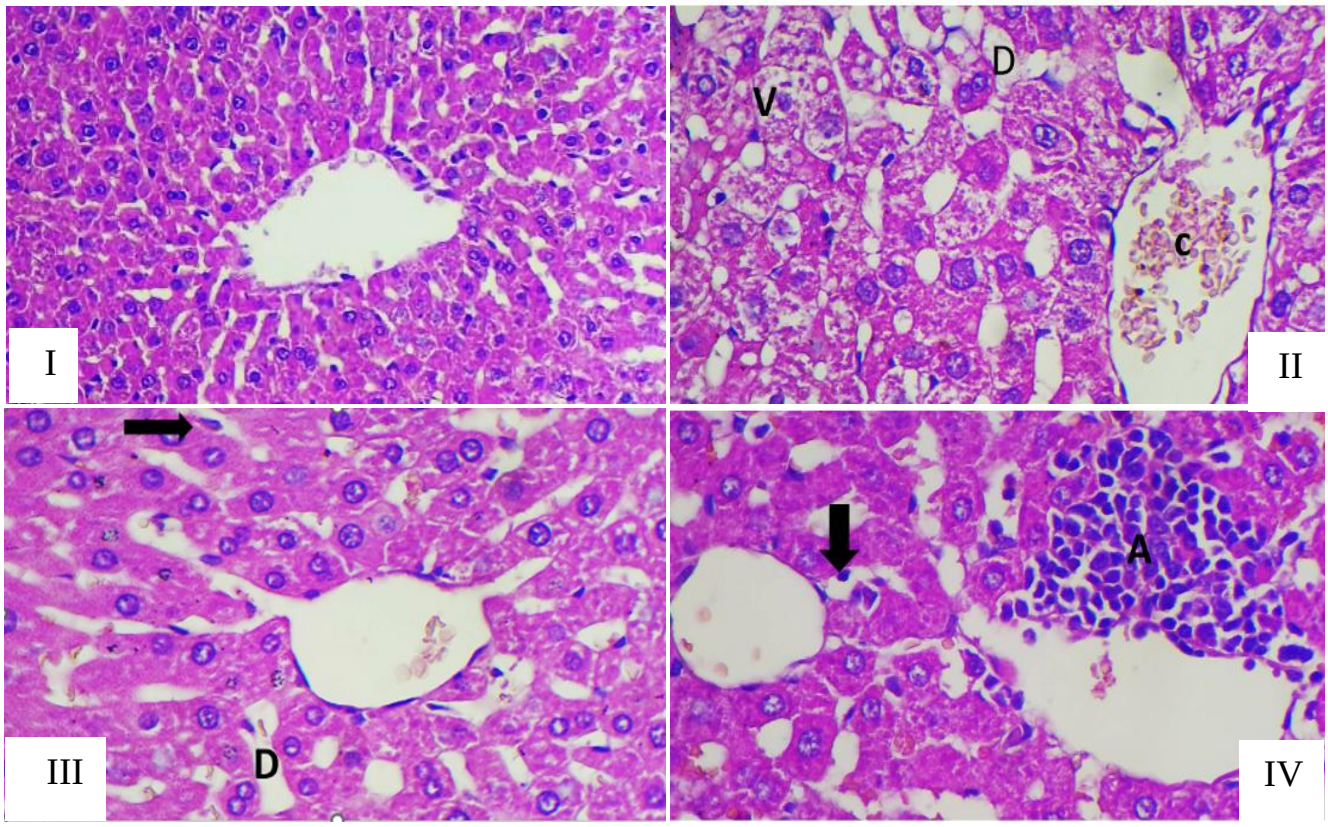



**Figure 4:** Histopathological section of rabbit liver **I:** control group showing normal histological architectures (H&E, stains 200x) **II**: diclofenac treated group showing vacuolation of hepatocyte (V) congestion of central vein(C), dilation and congestion of hepatic sinusoids (D). **III:** curcumin treated group showing mild dilation of hepatic sinusoids (D) with increase Kupfer cells infiltration ( arrow). **IV**: curcumin and diclofenac's treated group showing marked mononuclear cells aggregation beside central vein (A), with active Kupfer cells also seen (arrow) (H&E, stains, 400x).

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

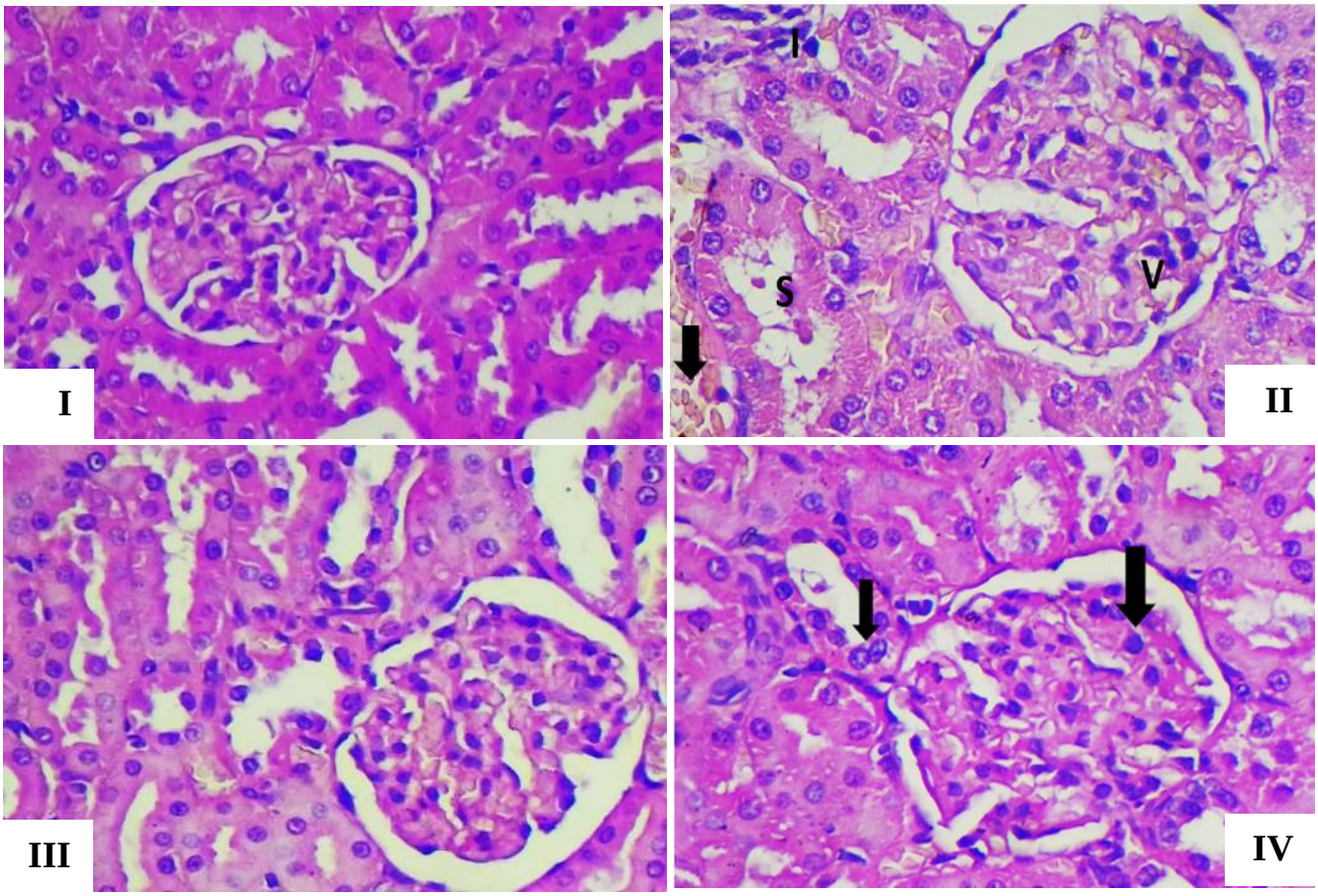
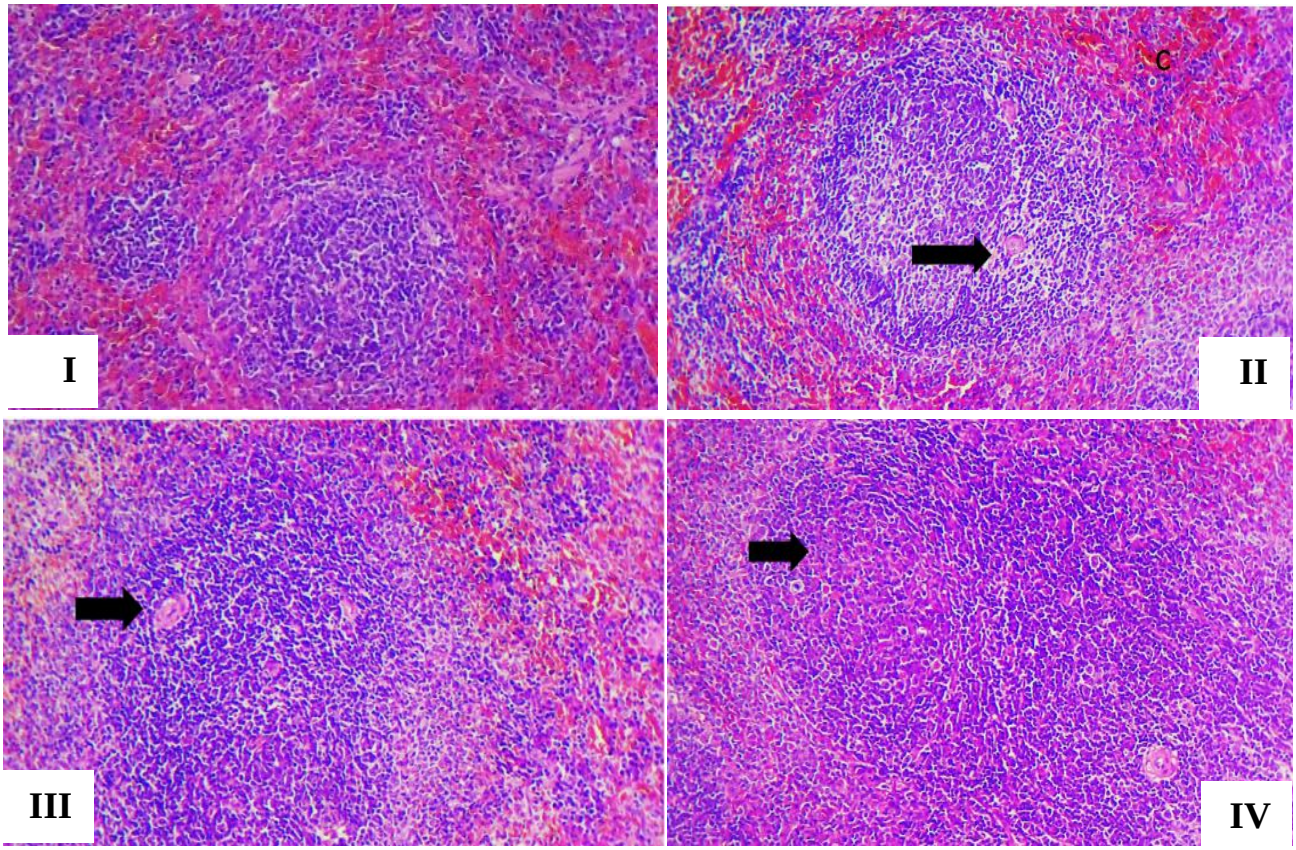



**Figure5:** Histopathological section of rabbit kidney **I:** control group showing normal histological architectures **II:** diclofenac treated group showing enlargement of glomerular tufts due to vacuolation in their cells (V),sloughing of epithelial cells lining the renal tubules (S), interstitial hemorrhage(arrow) and inflammatory cells infiltration (I) . **III.** curcumin treated group showing no clear lesion. **IV:** diclofenac and curcumin treated group showing regeneration of glomerular tuft and renal tubules( arrow) (H&E, stains 400x)

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010




**Figur6:** Histopathological section of rabbit spleen **I:** control treated group showing normal red and white plub. **II**: diclofenac treated group showing congestion of the red plub (C)with depletion in the white plub(arrow).**III:** curcumin treated group showing mild hyperplasia of white plub( arrow). **IV:** curcumin treated group showing marked hyperplasia in white plub due to increase in lymphocyte proliferation (arrow) (H&E, stains 200x).

## Discussion:

The present study demonstrated that administration of diclofenac sodium at a dose of 10 mg/kg b.w. produced gross pathological changes in the liver characterized by necrosis, hyperemia, and swelling. These findings are consistent with previous reports by (Gómez-Lechón *et al*., 2003) indicating that diclofenac may induce hepatotoxicity through oxidative stress, mitochondrial dysfunction, and the formation of reactive metabolites capable of damaging hepatocytes . The absence of gross renal lesions in the current study may be attributed to the relatively short duration of the experiment and the use of a single diclofenac dose. Previous investigations by (Harirforoosh *et al*., 2013) was showed that NSAID-induced renal injury may initially occur at the microscopic level before progressing to grossly visible lesions, particularly with prolonged exposure . No gross pathological changes were detected in the spleen, suggesting that the administered dose and study duration were insufficient to induce visible splenic alterations. Similar findings have been reported in experimental studies where diclofenac primarily targeted hepatic tissue, with minimal splenic involvement (Akinwumi *et al.*, 2018). The apparent reduction in hepatic lesion severity among rabbits co-treated with curcumin and diclofenac

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

may be attributed to curcumin's antioxidant and anti-inflammatory properties. Curcumin has been reported to reduce lipid peroxidation, suppress inflammatory mediators, and enhance antioxidant defense systems, thereby protecting tissues against oxidative damage induced by toxic compounds (Hewlings and Kalman, 2017).

Histological examination of the diclofenac-treated group revealed clear pathological alterations, including hepatocyte vacuolation, congestion of the central vein, and dilation of hepatic sinusoids findings consistent with hepatotoxicity likely mediated by oxidative stress and inflammatory responses this finding also reported in study by Alharbi, 2025. In the diclofenac + curcumin group showed, marked mononuclear cell aggregation near the central vein and active Kupffer cells were evident, this may be reflecting an organized inflammatory response and potential tissue repair, and highlighting curcumin's protective and immunomodulatory role against diclofenac-induced injury (Aboubakr *et al*., 2021). While the group treated with curcumin-only exhibited only mild sinusoidal dilation accompanied by increased Kupffer cell infiltration, suggesting that curcumin enhances phagocytic activity and immune surveillance while limiting structural damage (Aljuhani *et al*., 2019; Teschke, 2018).

In the diclofenac plus curcumin group, evidence of regeneration in glomerular tufts and renal tubules supported curcumin's role in promoting tissue repair and mitigating diclofenac-induced nephrotoxicity (Tabari *et al*., 2024). In contrast, the diclofenac-treated group displayed severe lesions, including glomerular tuft enlargement due to vacuolation, sloughing of tubular epithelial cells, interstitial hemorrhage, and inflammatory infiltration changes confirming diclofenac's nephrotoxic effects (Saxena and Meshram, 2018). The group treated with curcumin only showed no obvious lesions, with intact glomeruli and tubules, indicating a protective effect of curcumin against renal damage and preservation of normal renal histology (Hassan *et al*., 2026).

The diclofenac-treated group exhibited congestion of the red pulp and depletion of the white pulp, this result may due to immunosuppression or lymphocyte depletion (Yoo *et al*., 2025). The mild white pulp hyperplasia in curcumin-treated group indicating stimulation of lymphoid proliferation and immune activity this result agreement with (Vyas *et al*., 2019), while the group with marked changes displayed pronounced white pulp hyperplasia due to increased lymphocyte proliferation, confirming curcumin's immunostimulatory effect, Abari *et al*., 2024 also reach to same result.

## Conclusion

Administered of Diclofenac Sodium at 10 mg/kg b.w. induced marked hepatic damage in rabbits, characterized by necrosis, congestion, and cellular swelling, confirming the liver as the principal target organ. Curcumin alone showed no clear lesion in the tissues , and when co-administered with diclofenac, it reduced the severity of hepatic lesions. All, these findings underscore diclofenac's hepatotoxic potential and curcumin's partial protective role, with diclofenac administration inducing significant histopathological damage across the liver, kidney, and spleen consistent with its known toxicological profile. Curcumin treatment demonstrated protective and restorative properties, reducing lesion severity, enhancing immune cell activity, and promoting tissue regeneration. The findings

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

support the hypothesis that curcumin acts as an antioxidant and immunomodulator, mitigating diclofenac-induced toxicity and improving organ histology.

**Acknowledgment:**

The authors would like to acknowledge the support provided by the Department of Pathology and Poultry Diseases, College of Veterinary Medicine, Al-Qasim Green University, for facilitating the completion of this experimental study.

**Conflict of Interest:**

The authors declare that there are no conflicts of interest associated with this study.

**Funding Sources:**

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors**.**

**Authors Contributions:**

H.S.S. and G.A.A.A. contributed to the conception and design of the study. H.S.S. performed the experimental procedures, data collection, and laboratory investigations. G.A.A.A. contributed to the pathological evaluation, interpretation of results, and scientific supervision. Both authors participated in data analysis, manuscript preparation, critical revision of the manuscript, and approved the final version for publication.

**References**

Aboubakr, M., Abdelkader, A., Habotta, O.A., Adel, N., Emam, M.A., Abdelhiee, E.Y., Shanab, O., Shoghy, K., Elnoury, H., Soliman, M.M. and Ibrahim, S.F. (2021). Cefepime and diclofenac sodium combined treatment-potentiated multiple organ injury: role of oxidative damage and disrupted lipid metabolism. Journal of Biochemical and Molecular Toxicology, 35(12), e22929. https://doi.org/10.1002/jbt.22929

Akinwumi, I.A., Adeniyi, O.S. and Omotuyi, I.O. (2018). Evaluation of diclofenac-induced toxicity and associated pathological changes in experimental animals. Toxicology Reports, 5, 592–598.

Alharbi, F. (2025). Silymarin alleviates diclofenac-induced hepatotoxicity in male Wistar rats: Histopathological investigation. Open Veterinary Journal, 15(4), 1790–1797. https://doi.org/10.5455/OVJ.2025.v15.i4.31

Aljuhani, N., Elkablawy, M.A., Elbadawy, H.M., Alahmadi, A.M., Aloufi, A.M., Farsi, S.H., Alhubayshi, B.S., Alhejaili, S.S., Alhejaili, J.M. and Abdel-Halim, O.B. (2019). Protective effects of Ajwa date extract against tissue damage induced by acute diclofenac toxicity.

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

Journal of Taibah University Medical Sciences, 14(6), 553–559. https://doi.org/10.1016/j.jtumed.2019.10.002

Amalraj, A.; Pius, A.; Gopi, S. and Gopi, S. (2017). Biological activities of curcuminoids and curcumin. Journal of Traditional and Complementary Medicine, 7(2), 205–233. https://doi.org/10.1016/j.jtcme.2016.05.005

Beevers, C.S. and Huang, S. (2011). Pharmacological and clinical properties of curcumin. Botanics: Targets and Therapy, 1, 5–18. https://doi.org/10.2147/BTAT.S17244

Dronik, M. and Stasevych M. (2025). Expanding Horizons: Opportunities for Diclofenac Beyond Traditional Use – A Review. Scientia Pharmaceutica, 93(3), 31, 1–28. https://doi.org/10.3390/scipharm93030031

Gan, T.J. (2010). Diclofenac: an update on its mechanism of action and safety profile. Current Medical Research and Opinion, 26(7), 1715–1731. https://doi.org/10.1185/03007995.2010.486301

Gómez-Lechón, M.J.; Ponsoda, X. and O'Connor, E. (2003). Diclofenac induces apoptosis in hepatocytes through oxidative stress and mitochondrial dysfunction. Toxicology in Vitro, 17(5–6), 675–685. https://doi.org/10.1016/S0887-2333(03)00105-X

Harirforoosh, S.; Asghar, W. and Jamali, F. (2013). Adverse effects of nonsteroidal anti-inflammatory drugs: an update of gastrointestinal, cardiovascular and renal complications. Journal of Pharmacy and Pharmaceutical Sciences, 16(5), 821–847. https://doi.org/10.18433/j3vw2f

Hassan, R.M., El Shafei, A.A. and Mohamed, D.H. (2026). Protective effect of curcumin nanoparticles versus curcumin on gentamicin-induced nephrotoxicity in albino rats: histological and immunohistochemical study. Beni-Suef University Journal of Basic and Applied Sciences, 15(10), 10–13. https://doi.org/10.1186/s43088-025-00734-7

Hewlings, S.J. and Kalman, D.S. (2017). Curcumin: A Review of Its Effects on Human Health. Foods, 6(10), 92. https://doi.org/10.3390/foods6100092

Kang, S.J.; Chung, H. and Yoo, J.M. (2000). Inhibition of diclofenac formulated in hyaluronan on angiogenesis in vitro and its intraocular tolerance in the rabbit eye. Graefe's Archive for Clinical and Experimental Ophthalmology, 238(4), 273–282. https://doi.org/10.1007/s004170050353

Kunnumakkara, A.B.; Bordoloi, D. and Harsha, C. (2017). Curcumin mediates anticancer effects by modulating multiple cell signaling pathways. Clinical Science, 131(15), 1781–1799. https://doi.org/10.1042/CS20160935

P-ISSN: 2410-8863
E-ISSN:2958-6178
https://djvs.uodiyala.edu.iq
Pages: 132-144

Diyala Journal for Veterinary Sciences
Vol. 4 No.3 September (2026)
https://doi.org/10.71375/djvs.2026.043010

Parolini, M. (2020). Toxicity of the Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) acetylsalicylic acid, paracetamol, diclofenac, ibuprofen and naproxen towards freshwater invertebrates: A review. Science of the Total Environment, 740, 140043, 1–9. https://doi.org/10.1016/j.scitotenv.2020.140043

Saxena, A. and Meshram, S.V. (2018). Predictors of Mortality in Acute Kidney Injury Patients Admitted to Medicine Intensive Care Unit in a Rural Tertiary Care Hospital. Indian Journal of Critical Care Medicine: Peer-reviewed, Official Publication of Indian Society of Critical Care Medicine, 22(4), 231–237. https://doi.org/10.4103/ijccm.IJCCM_462_17

Sharifi-Rad, J.; Rayess, Y.E. and Rizk, A.A. (2020). Turmeric and its major compound curcumin on health: bioactive effects and safety profiles. Frontiers in Pharmacology, 11, 01021. https://doi.org/10.3389/fphar.2020.01021

Tabari, M.A., Houshyar, M., Araghi, A., Mirzakhani, N., Crescenzo, G., Cardone, R. and Zizzadoro, C. (2024). Nephroprotective and hepatoprotective effects of lemongrass essential oil and citral on diclofenac-induced toxicity in mice. Biomedicine & Pharmacotherapy, 180, 117541. https://doi.org/10.1016/j.biopha.2024.117541

Teschke, R. (2018). Alcoholic liver disease: Alcohol metabolism, cascade of molecular mechanisms, cellular targets, and clinical aspects. Biomedicines, 6(4), 106. https://doi.org/10.3390/biomedicines6040106

Vyas, A.; Purohit, A. and Ram, H. (2019). Assessment of dose-dependent reproductive toxicity of diclofenac sodium in male rats. Drug and Chemical Toxicology, 42, 478–486. https://doi.org/10.1080/01480545.2017.1421659

Yoo, S., Noh, J.H., Lee, H.S., Lee, S.H., Choi, E., Kim, D.I., Min, S.E., Han, K.H. and Kim, S.K. (2025). Toxicity of diclofenac sodium salt after two weeks of daily intramuscular administration in cynomolgus monkeys. Toxicological Research, 41(3), 279–290. https://doi.org/10.1007/s43188-025-00281-4

Zhang, L.; Fang, Y. and Xu, Y. (2023). Pharmacological mechanisms and clinical applications of curcumin: Update. Aging and Disease, 14(3), 716–749. https://doi.org/10.14336/AD.2022.1101