Research Article

Received: 12 March, 2026
Accepted: 03 August, 2026
Published: 12 August, 2026

Biochemical and Histopathological Evaluation of Banana Peel Extract in CCl4-Induced Liver Toxicity

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Cite this article:

Ahmed, M. A., & Ibrahim, T. A. (2026). Biochemical and Histopathological Evaluation of Banana Peel Extract in CCl_4-Induced Liver Toxicity. Revista Cultura Científica, (24), pp. 1121–1130.

Abstract

This study evaluated the potential protective effect of banana peel extract against carbon tetrachloride (CCl_4)-induced liver toxicity in adult male albino rats. Twenty rats of comparable body weight were randomly divided into five groups, with four animals in each group, following a one-week acclimatization period. Group G1 served as the healthy control, whereas G2 received CCl_4 by subcutaneous injection at a dose of 0.002 mL/g. Groups G3, G4, and G5 received banana peel extract at doses of 0.003, 0.004, and 0.006 mg/kg, respectively, according to the experimental treatment protocol. Biochemical parameters and histopathological changes in liver tissue were subsequently evaluated. Serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels were markedly elevated in G2 (188.90 $\pm$ 40.75 and 141.10 $\pm$ 38.79 IU/L, respectively) compared with G1 (39.00 $\pm$ 1.47 and 51.00 $\pm$ 13.49 IU/L, respectively). Lower AST and ALT values were observed in the banana peel extract-treated groups, with values approaching those of the control group at the higher treatment levels. Gamma-glutamyl transferase (GGT) was also elevated in G2 (73.75 $\pm$ 8.29 IU/L) compared with G1 (52.60 $\pm$ 5.21 IU/L), whereas G3, G4, and G5 showed values of 63.45 $\pm$ 5.34, 48.00 $\pm$ 6.46, and 52.12 $\pm$ 4.22 IU/L, respectively. Lactate dehydrogenase (LDH) showed a variable response among groups, with the highest value observed in G5 (325.75 $\pm$ 30.06 IU/L) and the lowest in G3 (199.00 $\pm$ 11.88 IU/L), with statistically significant differences among groups ($P \leq 0.05$). Histological examination showed a normal hepatic architecture in G1, whereas G2 exhibited vascular congestion, dilation and distortion of the central vein, red blood cell accumulation, and inflammatory changes. The extract-treated groups showed varying degrees of histological improvement, including partial restoration of hepatocyte organization and reduced inflammatory cell infiltration. Overall, the findings suggest that banana peel extract may exert a protective effect against CCl_4-induced hepatic injury, although the response varied across the measured biochemical and histological parameters.

Keywords: banana peel extract, carbon tetrachloride, hepatotoxicity, liver enzymes, histopathology

1. INTRODUCTION

Liver diseases constitute an important and growing global public health concern because the liver plays a central role in metabolism, detoxification, nutrient processing, and the maintenance of physiological homeostasis [1]. As the principal organ responsible for the biotransformation of drugs, chemicals, and environmental toxicants, the liver is particularly susceptible to injury following acute or chronic exposure to hepatotoxic agents. Among the mechanisms involved in hepatic injury, oxidative stress is recognized as an important contributor to the initiation and progression of several liver disorders [2].

Oxidative stress develops when the production of reactive oxygen species (ROS) and other free radicals exceeds the capacity of endogenous antioxidant defense systems [3]. Excessive accumulation of reactive species can damage cellular components, including membrane lipids, proteins, and nucleic acids, thereby promoting inflammation, cellular degeneration, and cell death. Persistent oxidative imbalance has been associated with the pathogenesis and progression of a variety of hepatic disorders, including hepatitis, fibrosis, cirrhosis, and liver failure [4]. Consequently, experimental models that reproduce oxidative liver injury are frequently used to investigate potential hepatoprotective compounds.

Carbon tetrachloride (CCl_4) is widely used in experimental animal models to induce hepatic injury because its metabolism generates highly reactive free-radical intermediates that promote oxidative damage and lipid peroxidation [5]. Exposure to CCl_4 can disrupt hepatocyte membrane integrity and alter biochemical indicators of liver function, making it a commonly employed model for evaluating potential hepatoprotective interventions. In parallel, increasing attention has been directed toward plant-derived extracts and natural products as potential sources of antioxidant compounds. Such materials contain phytochemicals, including phenolic compounds, tannins, flavonoids, and vitamins, that may contribute to free-radical scavenging and support endogenous antioxidant defense mechanisms [6].

Banana (*Musa* spp.) is a perennial tropical plant cultivated extensively in many regions because of its nutritional and economic importance [7]. Although the fruit is the principal commercial product, banana processing generates substantial quantities of peel waste. These peels are increasingly recognized as a potential source of biologically active compounds rather than merely an agricultural by-product [8]. Previous studies have reported that banana peels contain phenolic compounds, flavonoids, carotenoids, vitamins, and minerals with potential antioxidant activity. Extracts prepared from banana peels have also demonstrated antioxidant, anti-inflammatory, and antimicrobial properties in experimental studies, suggesting possible applications in biomedical and pharmaceutical research [9, 10].

On this basis, the present study was designed to evaluate the potential hepatoprotective effect of aqueous banana peel extract in albino rats subjected to CCl_4-induced liver injury. The effects of the extract were assessed using biochemical indicators of hepatic function together with histopathological examination of liver tissue. By comparing untreated controls, CCl_4-exposed animals, and animals receiving different concentrations of banana peel extract, the study aimed to determine whether the extract could attenuate the biochemical and structural alterations associated with CCl_4-induced hepatotoxicity.

2. MATERIALS AND METHODS

Chemicals and plant ,aterial. Carbon tetrachloride (CCl_4) used in the experiment was obtained from a commercial source in the United Kingdom. Banana peels were collected from a local market in Baqubah City, Iraq, and used for the preparation of the aqueous extract.

Preparation of the Aqueous banana peel extract. Fresh banana peels were thoroughly washed under running water to remove surface contaminants and were subsequently rinsed with distilled water. The peels were cut into small pieces approximately 1–2 cm in size and dried under sunlight for 48–72 h until a constant weight was obtained. The dried material was then ground using a mortar to produce a fine, homogeneous powder and stored in opaque glass containers until extraction.

For preparation of the aqueous extract, 10 g of banana peel powder was mixed with 50 mL of deionized water. The mixture was maintained on a hot plate for 72 h and was subsequently filtered through Whatman No. 1 filter paper. The filtrate was further clarified by centrifugation at 5000 rpm for 10 min. The resulting extract was transferred to Petri dishes and dried. The dried aqueous extract was then collected and stored in sterile containers under refrigerated conditions until use.

Experimental animals and housing conditions. A total of 20 adult male albino rats weighing approximately 20–39 g were used in the experiment. The animals were maintained at the animal facility of the College of Education for Pure Science, Diyala University. They were housed under a 12-h light/12-h dark cycle and provided with free access to standard laboratory feed and water.

Before the experimental procedures began, the animals were allowed to acclimatize to the laboratory environment for one week. Rats of comparable body weight were then randomly assigned to five experimental groups, with four animals

in each group.

Experimental design. Following acclimatization, the experimental animals were allocated to the following groups:

- **Group 1 (G1):** Healthy control group.
- **Group 2 (G2):** Animals exposed to CCl_4 to induce hepatic injury.
- **Group 3 (G3):** Animals exposed to CCl_4 and subsequently treated with aqueous banana peel extract at a dose of 0.003 mg/kg body weight daily for 30 days.
- **Group 4 (G4):** Animals exposed to CCl_4 and subsequently treated with aqueous banana peel extract at a dose of 0.004 mg/kg body weight daily for 30 days.
- **Group 5 (G5):** Animals exposed to CCl_4 and subsequently treated with aqueous banana peel extract at a dose of 0.006 mg/kg body weight daily for 30 days.

The experimental treatment period lasted 30 days.

Blood collection and Serum preparation. At the end of the experimental period, the animals were weighed and anesthetized according to the experimental protocol. Blood samples were collected by cardiac puncture using sterile syringes and transferred into sterile tubes without anticoagulant.

The blood samples were centrifuged at 3000 rpm for 15 min to separate the serum. The resulting serum was transferred into clean, labeled Eppendorf tubes and stored at -20°C until biochemical analysis.

Biochemical analysis. Serum biochemical parameters were measured using commercially available diagnostic kits and standard enzymatic colorimetric methods. The parameters evaluated included total bilirubin, direct bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), and gamma-glutamyl transferase (GGT).

These biochemical indicators were used to assess changes in hepatic function and cellular injury among the experimental groups.

Histopathological examination. Following blood collection, the animals were dissected and the livers were carefully removed. Surrounding adipose and connective tissues were removed, and the liver samples were washed with distilled water to eliminate residual blood. The tissues were gently dried on filter paper and weighed.

Liver specimens were subsequently fixed in 10% formalin solution for histopathological examination. Following routine tissue processing and embedding, sections approximately 5 μm thick were prepared and stained with hematoxylin and eosin (H&E). Histopathological alterations were evaluated by a pathologist who was blinded to the experimental groups [11].

3. RESULTS AND DISCUSSION

The biochemical and histopathological findings were evaluated comparatively across the five experimental groups in order to determine the extent of liver injury produced by CCl_4 and to assess the response associated with banana peel extract treatment. The biochemical results are summarized in Tables 1 and 2, whereas representative histological findings are presented in Figures 1–4. Taken together, the results show a clear biochemical disturbance in the CCl_4-exposed group and varying degrees of recovery in the extract-treated groups. However, the response was not uniform across all measured parameters, particularly LDH, and the histological findings therefore provide an important complementary assessment of tissue-level changes.

3.1. BIOCHEMICAL FINDINGS

Table 1 presents the serum activities of AST, ALT, GGT, and LDH in the five experimental groups. Marked differences were observed among the groups. The highest AST and ALT values were recorded in the CCl_4-treated group (G2), where AST reached 188.90 ± 40.75 IU/L and ALT reached 141.10 ± 38.79 IU/L. These values were substantially higher than those of the healthy control group (G1), in which AST and ALT were 39.00 ± 1.47 IU/L and 51.00 ± 13.49 IU/L, respectively. The increase in both enzymes after CCl_4 exposure is consistent with hepatocellular injury and loss of membrane integrity, which can permit intracellular enzymes to enter the circulation.

The extract-treated groups showed lower AST and ALT values than G2. In G3, AST and ALT were 106.42 ± 6.90 IU/L and 89.07 ± 20.11 IU/L, respectively; in G4, the corresponding values were 69.00 ± 14.04 IU/L and 68.75 ± 12.46

IU/L; and in G5, they were 49.50 ± 7.93 IU/L and 48.25 ± 10.75 IU/L. Thus, the numerical pattern in Table 1 shows progressive movement of AST and ALT toward the control values across the treated groups, with G5 exhibiting the closest values to G1 for these two enzymes.

GGT also differed among the groups. The CCl_4 group (G2) showed the highest GGT value, 73.75 ± 8.29 IU/L, compared with 52.60 ± 5.21 IU/L in G1. The values in G3, G4, and G5 were 63.45 ± 5.34, 48.00 ± 6.46, and 52.12 ± 4.22 IU/L, respectively. In contrast to AST, ALT, and GGT, LDH did not display a simple dose-related recovery pattern. The lowest LDH value occurred in G3 (199.00 ± 11.88 IU/L), whereas G4 and G5 showed higher values of 272.50 ± 14.40 and 325.75 ± 30.06 IU/L, respectively. Notably, the LDH value in G5 exceeded that of both G1 (252.50 ± 24.78 IU/L) and G2 (221.75 ± 5.75 IU/L). This divergence indicates that the biochemical response to the extract was parameter-dependent and that the LDH findings should not be interpreted as showing the same recovery pattern observed for AST and ALT. The reported statistical analysis indicated significant differences among the groups, as summarized by the P-values and letter-based comparisons in Table 1.

Table 1. *Liver enzyme levels in the studied groups.*

Groups	AST (IU/L)	ALT (IU/L)	GGT (IU/L)	LDH (IU/L)
G1	39.00 ± 1.47 c	51.00 ± 13.49 b	52.60 ± 5.21 b	252.50 ± 24.78 bc
G2	188.90 ± 40.75 a	141.10 ± 38.79 a	73.75 ± 8.29 a	221.75 ± 5.75 bc
G3	106.42 ± 6.90 b	89.07 ± 20.11 ab	63.45 ± 5.34 ab	199.00 ± 11.88 c
G4	69.00 ± 14.04 bc	68.75 ± 12.46 b	48.00 ± 6.46 b	272.50 ± 14.40 ab
G5	49.50 ± 7.93 bc	48.25 ± 10.75 b	52.12 ± 4.22 b	325.75 ± 30.06 a
L.S.D.	59.29 **	65.52 *	18.28 *	58.75 **
P-value	0.0005	0.0496	0.0499	0.0035

Means with different letters within the same column indicate significant differences; *$P \leq 0.05$, **$P \leq 0.01$.

The pronounced increases in AST, ALT, and GGT in G2 relative to G1 are consistent with the hepatotoxic effect of CCl_4. Following metabolic activation, CCl_4 can generate reactive intermediates capable of initiating lipid peroxidation and damaging hepatocyte membranes. Loss of membrane integrity facilitates the release of intracellular enzymes into the circulation, thereby increasing serum enzyme activities. This interpretation is consistent with previous reports describing CCl_4 as a potent experimental hepatotoxic agent [12, 13].

The lower AST, ALT, and GGT values observed in the extract-treated groups may reflect partial preservation or recovery of hepatocyte membrane integrity. Banana peel contains phenolic compounds and flavonoids that have been associated with antioxidant and free-radical-scavenging activity. These constituents may contribute to limiting membrane injury and, consequently, reducing leakage of hepatic enzymes into the blood. The observed biochemical pattern is therefore compatible with the hepatoprotective potential reported for banana-derived preparations [14]. Nevertheless, the elevated LDH in G5 indicates that the response should not be generalized across all biochemical markers and emphasizes the need to interpret individual parameters separately.

Table 2 summarizes the total and direct bilirubin concentrations. The highest bilirubin values were observed in G2, with total bilirubin of 30.25 ± 3.77 mg/dL and direct bilirubin of 6.82 ± 1.04 mg/dL. These values were higher than those of G1, which showed total bilirubin of 16.55 ± 0.48 mg/dL and direct bilirubin of 4.10 ± 0.28 mg/dL. The elevation in G2 is consistent with impaired hepatic handling of bilirubin after CCl_4-induced liver injury.

The extract-treated groups showed lower bilirubin concentrations than G2. Total bilirubin was 15.82 ± 1.24 mg/dL in G3, 16.15 ± 1.28 mg/dL in G4, and 13.93 ± 3.72 mg/dL in G5. Direct bilirubin values were 4.22 ± 0.37, 4.00 ± 0.58, and 3.36 ± 0.64 mg/dL in G3, G4, and G5, respectively. As shown in Table 2, all three extract-treated groups had values closer to the control group than to G2. The reported P-values were 0.0022 for total bilirubin and 0.0152 for direct bilirubin, indicating statistically significant differences among the experimental groups according to the analysis used in the study.

Table 2. *Effect of the experimental treatments on total and direct bilirubin levels (mean ± SE, mg/dL).*

Group / Concentration	Total Bilirubin (mg/dL)	Direct Bilirubin (mg/dL)
G1	16.55 ± 0.48 b	4.10 ± 0.28 b
G2	30.25 ± 3.77 a	6.82 ± 1.04 a
G3	15.82 ± 1.24 b	4.22 ± 0.37 b
G4	16.15 ± 1.28 b	4.00 ± 0.58 b
G5	13.93 ± 3.72 b	3.36 ± 0.64 b
L.S.D.	7.562 **	1.931 *
P-value	0.0022	0.0152

The marked bilirubin elevation in G2 may reflect reduced hepatic capacity for bilirubin uptake, conjugation, and

excretion after toxic injury. Increased circulating bilirubin has been associated with impaired hepatocellular function and, in some settings, altered biliary excretion following CCl_4 exposure [15]. By comparison, the lower total and direct bilirubin values in G3–G5 suggest partial restoration of these hepatic functions. The findings are also consistent with previous observations that banana-derived extracts may support recovery of liver tissue and biochemical function [16]. Because the biochemical outcomes were not uniform for all enzymes, however, the bilirubin results are most appropriately considered together with the histopathological observations described below.

3.2. HISTOLOGICAL STUDY

Representative histological sections provide tissue-level evidence that complements the biochemical findings. Figure 1 shows the liver architecture of the healthy control group (G1). The section demonstrates a generally preserved hepatic organization, with a central vein surrounded by cords of polygonal hepatocytes and intervening sinusoidal spaces. Hepatocyte nuclei are visible, and the overall lobular pattern is consistent with the expected appearance of control liver tissue. This image provides the morphological reference against which the CCl_4-exposed and extract-treated groups were compared.

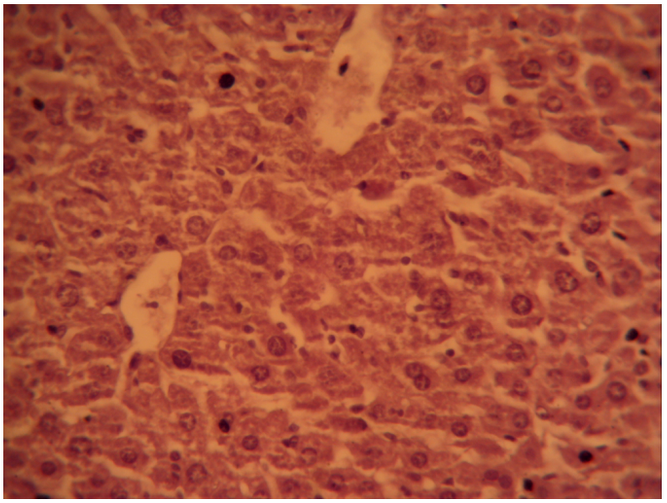

Figure 1. *Representative liver section from the control group (G1), showing preserved hepatic architecture with the central vein (CV), hepatocytes (HC), nuclei (N), binucleated hepatocytes (BN), and sinusoids (S) (H&E stain, 40×).*

In contrast, Figure 2 shows representative histological changes after CCl_4 exposure in G2. The left portion of the image demonstrates prominent vascular congestion around the central vein and accumulation of red blood cells (RBC), whereas the right portion highlights enlarged hepatic sinusoids (ES) and infiltration of inflammatory cells (IC). Relative to the preserved architecture in Figure 1, the CCl_4-exposed tissue therefore shows clear structural disturbance involving the vascular and parenchymal compartments.

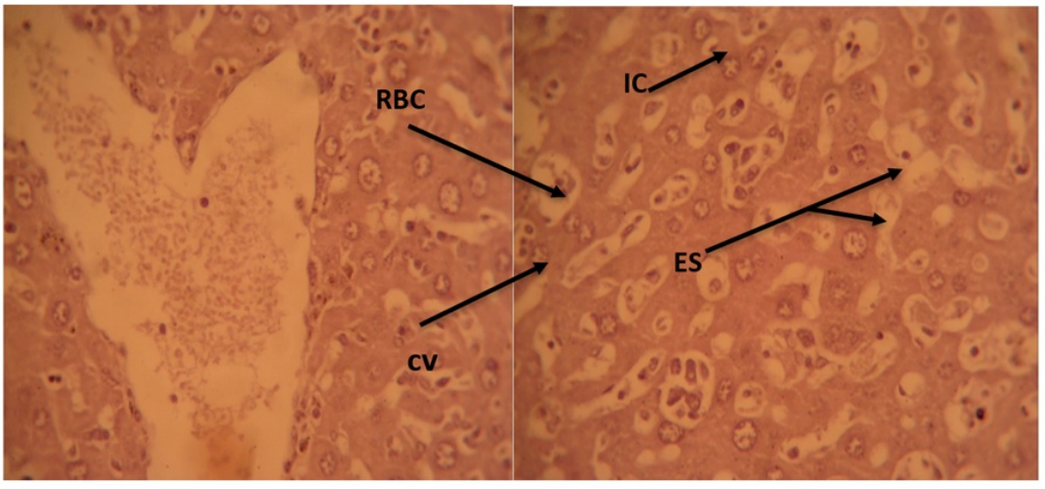

Figure 2. *Representative histological changes in the liver after CCl_4 exposure (G2), showing vascular congestion around the central vein (CV), red blood cell accumulation (RBC), enlargement of hepatic sinusoids (ES), and inflammatory cell infiltration (IC).*

The alterations visible in Figure 2 are compatible with the recognized hepatotoxic effects of CCl_4. Histological injury

in CCl_4 models is commonly associated with vascular congestion, disruption of hepatic cords, hepatocyte degeneration, inflammatory changes, and disturbances in sinusoidal organization. The mechanism is strongly linked to the metabolic formation of reactive trichloromethyl and peroxytrichloromethyl radicals, which initiate lipid peroxidation and promote membrane and cellular injury [17]. Similar patterns of necrotic and degenerative liver injury following CCl_4 exposure have been reported by El Sayed et al. [18]. Reactive intermediates generated from CCl_4 can damage cellular membranes, disturb enzyme activity, impair mitochondrial processes, and contribute to progressive hepatocellular injury.

Additional mechanisms associated with CCl_4 toxicity include disruption of oxidative phosphorylation, depletion of sulfhydryl-containing cellular defenses, increased lipid peroxidation, altered calcium homeostasis, and damage to nucleic acids [19, 20]. These mechanisms provide a plausible biological explanation for the simultaneous biochemical and histological abnormalities observed in G2. The substantial increases in AST, ALT, and GGT shown in Table 1, together with the congestion, sinusoidal enlargement, and inflammatory infiltration visible in Figure 2, indicate that the CCl_4 exposure protocol produced measurable hepatic injury in the experimental animals.

3.3. TREATMENT WITH AQUEOUS BANANA PEEL EXTRACT AT 0.003 MG/KG

Histological examination of the 0.003 mg/kg treatment condition showed that the lowest extract dose was associated with only limited morphological improvement. As illustrated in Figure 3, the liver tissue continued to display structural abnormalities after treatment. The left image shows residual red blood cell accumulation within the tissue, while the right image highlights a region of degenerative cellular change (DC). The hepatocyte arrangement remained disrupted, and evidence of inflammatory-cell infiltration and nuclear alterations was still present.

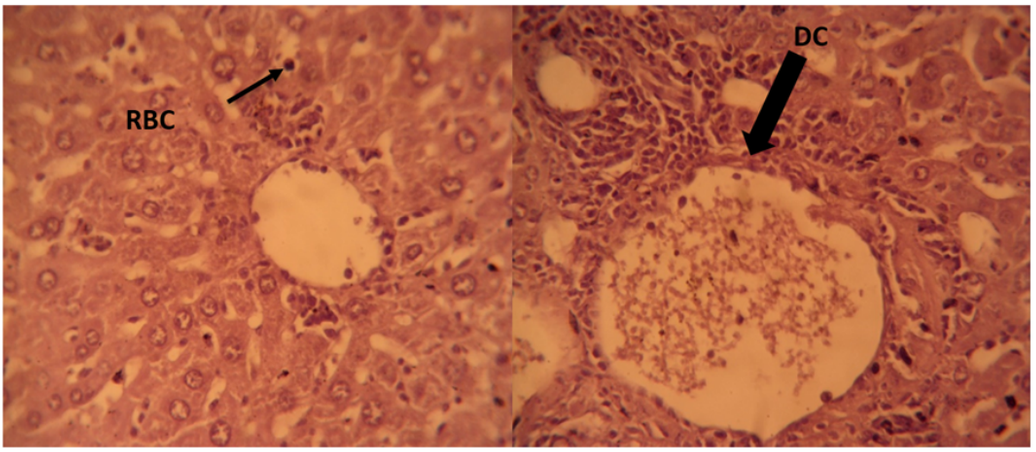

Figure 3. *Representative histological appearance after treatment with aqueous banana peel extract at 0.003 mg/kg, showing persistent red blood cell accumulation (RBC) and degenerative cellular changes (DC).*

The persistence of tissue abnormalities in Figure 3 indicates that the 0.003 mg/kg treatment did not completely reverse the histological injury produced by CCl_4. Although some biochemical improvement was observed in G3, particularly relative to G2, the histological appearance suggests that recovery at the tissue level was incomplete. This distinction is important because normalization of selected serum markers does not necessarily imply complete restoration of hepatic architecture.

The limited histological response at the lower extract concentration may be related to an insufficient amount of biologically active constituents reaching the damaged tissue. Banana peel is reported to contain phenolic compounds and flavonoids with antioxidant properties, but the biological effect of an extract can depend on dose, composition, extraction efficiency, and bioavailability. At a relatively low treatment level, the quantity of active compounds may therefore have been insufficient to counter the continuing oxidative and inflammatory processes initiated by CCl_4 [23]. The findings in Figure 3 are consequently consistent with a partial, rather than complete, protective response at the lowest tested concentration.

3.4. TREATMENT WITH AQUEOUS BANANA PEEL EXTRACT AT 0.006 MG/KG

A different histological pattern was observed in the tissue presented for the 0.006 mg/kg treatment. Figure 4 shows a comparatively more preserved hepatic appearance than the CCl_4-only tissue shown in Figure 2. Hepatocytes (HC), their nuclei (N), a central vein (CV), and sinusoidal spaces (S) are readily identifiable. The organization of the hepatic parenchyma appears more regular, although the sinusoidal spaces remain conspicuous and complete normalization cannot be inferred from a representative image alone.

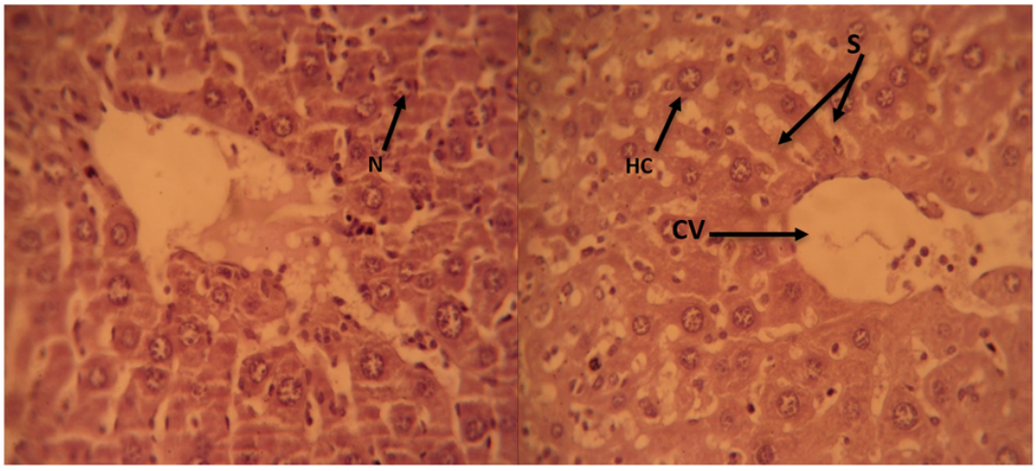

Figure 4. *Representative histological appearance after treatment with aqueous banana peel extract at 0.006 mg/kg, showing hepatocytes (HC), nuclei (N), the central vein (CV), and sinusoidal spaces (S).*

The appearance in Figure 4, when considered together with the lower AST, ALT, GGT, and bilirubin values reported for the higher-dose treatment group in Tables 1 and 2, suggests a degree of structural and functional recovery relative to the CCl_4-only group. In particular, the preservation of recognizable hepatocyte organization and central-vein architecture contrasts with the marked congestion and inflammatory changes illustrated in Figure 2. Nevertheless, the elevated LDH value in G5 shown in Table 1 demonstrates that the biochemical response was not uniformly normalized, and this finding should be considered when interpreting the overall protective effect.

The apparent improvement at the higher extract concentration is biologically plausible in view of the antioxidant constituents reported in banana-derived materials. Arhoghro and Kpomah [24] described antioxidant-related effects of banana plant material in an experimental model of hepatic oxidative stress. Phenolic and flavonoid constituents can act as electron or hydrogen donors and may interrupt free-radical chain reactions. Such compounds have also been associated with modulation of endogenous antioxidant systems, including superoxide dismutase, catalase, and glutathione-related defenses [25]. In the present study, these mechanisms were not measured directly; therefore, they should be regarded as possible explanations for the observed biochemical and histological changes rather than as demonstrated mechanisms.

Related experimental work has also reported hepatoprotective effects of banana-derived preparations in CCl_4-associated liver injury. Elhassaneen et al. [26] investigated banana peel-containing preparations in male rats with experimentally induced hepatotoxicity and reported improvements in biochemical and tissue-related outcomes. Banana peel contains several classes of bioactive compounds, including polyphenols and flavonoids, that may contribute to antioxidant and anti-inflammatory activity [27]. These findings provide contextual support for the present observations, although differences in extract preparation, administered dose, treatment duration, and experimental design should be considered when comparing studies.

Overall, the results demonstrate a consistent pattern of pronounced liver injury in the CCl_4-exposed group, as evidenced by the enzyme and bilirubin changes in Tables 1 and 2 and by the histological abnormalities in Figure 2. The control morphology shown in Figure 1 provides a clear reference for these alterations. Treatment with banana peel extract was associated with lower AST, ALT, GGT, and bilirubin values and with varying degrees of histological improvement. Figure 3 indicates that the lowest extract concentration was associated with persistent structural abnormalities, whereas Figure 4 shows a more preserved tissue organization at the higher concentration. At the same time, the LDH pattern in Table 1 did not parallel the other liver enzymes, indicating that the protective response was not uniform across all measured endpoints. These combined biochemical and histological findings support a cautious interpretation that aqueous banana peel extract may attenuate some features of CCl_4-induced hepatic injury, while further work is required to establish the magnitude, reproducibility, and mechanism of the effect.

4. CONCLUSION

The findings of the present study indicate that exposure to CCl_4 produced marked biochemical and histopathological alterations in the liver, including elevated serum liver enzyme activities, increased bilirubin levels, vascular congestion, inflammatory-cell infiltration, and disruption of normal hepatic architecture. Treatment with aqueous banana peel extract was associated with varying degrees of improvement in several of these parameters, particularly reductions in AST, ALT, GGT, and bilirubin levels, together with partial restoration of liver tissue organization at the higher treatment concentration.

The histopathological findings further suggest that the protective response was concentration-dependent. The lowest

tested concentration was associated with persistent structural abnormalities, whereas the higher concentration showed comparatively better preservation of hepatocyte organization and vascular architecture. However, the biochemical response was not uniform across all measured markers, as LDH did not follow the same recovery pattern observed for AST, ALT, and GGT. Therefore, the overall findings support a cautious interpretation that banana peel extract may exert a hepatoprotective effect against CCl_4-induced liver injury rather than demonstrating complete biochemical and histological normalization.

Because direct oxidative-stress biomarkers were not measured in the present experiment, the antioxidant mechanism proposed for banana peel extract should be regarded as a plausible explanation supported by previous literature rather than as a mechanism directly demonstrated in this study. Further investigations should include larger sample sizes, standardized extract characterization, direct measurement of oxidative-stress and antioxidant biomarkers, and systematic histopathological scoring. Future studies should also evaluate a broader range of doses and treatment durations and identify the specific bioactive compounds responsible for the observed protective effects.

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