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Simultaneous Hepatic and Renal Biochemical Toxicity Following Chronic Petroleum Hydrocarbon Exposure in Chickens

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Abstract

Petroleum hydrocarbons are persistent environmental contaminants capable of inducing multi-organ toxicity following chronic exposure. This study evaluated simultaneous hepatic and renal biochemical toxicity in chickens chronically exposed to a petroleum hydrocarbon–contaminated environment. Exposed chickens (n = 12; 6- and 12-month exposure groups) were compared with unexposed controls (n = 6). Serum hepatic enzymes, protein indices, bilirubin fractions, renal biomarkers, and electrolyte profiles were determined using standard biochemical methods, and data were analyzed at $p < 0.05$. Significant hepatic dysfunction was observed in exposed chickens, characterized by elevated serum AST (43.92 ± 15.14 vs 13.00 ± 4.52 IU/L), ALT (25.42 ± 22.15 vs 8.17 ± 6.46 IU/L), and ALP (52.67 ± 24.37 vs 25.00 ± 13.84 IU/L) activities relative to controls. Serum albumin (53.42 ± 24.23 vs 95.50 ± 14.73 g/L) and total protein (59.00 ± 23.61 vs 91.50 ± 15.60 g/L) concentrations were significantly reduced, whereas total bilirubin levels increased (11.08 ± 4.46 vs 3.40 ± 1.67 µmol/L). Renal dysfunction was evidenced by elevated serum urea (13.40 ± 9.40 vs 2.42 ± 1.89 mmol/L) and creatinine (3.21 ± 3.34 vs 1.52 ± 1.94 mmol/L), accompanied by significant electrolyte imbalance. Chickens exposed for 12 months exhibited more severe biochemical alterations than those exposed for 6 months, indicating progressive and time-dependent toxicity. The findings demonstrate that chronic petroleum hydrocarbon exposure induces significant simultaneous hepatorenal biochemical toxicity in chickens and highlight the importance of combined hepatic and renal biomarker assessment in environmental toxicology studies.

Keywords: *Petroleum hydrocarbons, hepatotoxicity, nephrotoxicity, biochemical markers, chickens, environmental toxicity, Nigeria.*

INTRODUCTION

Petroleum hydrocarbons are among the most important environmental contaminants generated through crude oil exploration, transportation, refining, industrial discharge, and accidental spills [1]. In oil-producing communities, continuous contamination of soil and water creates long-term exposure risks for humans and animals [2]. Organisms inhabiting polluted environments may absorb hydrocarbons through ingestion of contaminated feed and water, inhalation of volatile compounds, or dermal exposure [3]. Chronic exposure to petroleum hydrocarbons has been linked with toxic effects involving several organ systems, especially the liver and kidneys because of their critical functions in metabolism, detoxification, and excretion of xenobiotics [4].

The liver is highly susceptible to petroleum hydrocarbon toxicity because it is the principal organ responsible for the metabolism of foreign compounds [5]. Petroleum hydrocarbons include aliphatic compounds, aromatic hydrocarbons, cycloalkanes, and polycyclic aromatic hydrocarbons, many of which are capable of generating reactive intermediates during metabolism [6].

These metabolites may induce oxidative stress, lipid peroxidation, mitochondrial dysfunction, and membrane instability within hepatocytes, leading to hepatocellular injury and altered liver function [7]. Such hepatic damage is commonly reflected by elevated serum aminotransferases and alkaline phosphatase activities, together with disturbances in albumin synthesis, total protein concentration, and bilirubin metabolism [8].

The kidneys are also vulnerable to hydrocarbon toxicity because they participate in the elimination of water-soluble metabolites derived from petroleum hydrocarbons [9]. Exposure to these compounds may impair glomerular filtration and tubular function, resulting in elevated serum urea and creatinine concentrations as well as electrolyte imbalance [10]. Persistent renal injury may further contribute to systemic metabolic disturbances and altered physiological homeostasis [11].

Interactions between hepatic and renal dysfunction are increasingly recognized in toxicological and clinical studies. Damage to the liver may increase circulating toxic metabolites capable of aggravating renal injury, while impaired renal clearance may prolong systemic retention of hepatotoxic compounds [12,13].

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Despite this relationship, many studies continue to assess hepatic and renal toxicity separately, thereby limiting understanding of the overall systemic effects of petroleum hydrocarbon exposure.

Chickens are considered valuable sentinel organisms in environmental toxicology because they are continuously exposed to environmental contaminants through soil, water, and feed interactions [14]. In addition, chickens are economically important food animals, making environmental toxicant exposure a public health concern [15]. Although previous studies have investigated petroleum hydrocarbon toxicity in poultry, information regarding simultaneous hepatic and renal biochemical responses during chronic exposure remains limited.

Therefore, this study was designed to evaluate concurrent hepatic and renal biochemical toxicity in chickens chronically exposed to petroleum hydrocarbon contamination. The study assessed liver enzymes, bilirubin fractions, serum proteins, renal biomarkers, and electrolyte profiles, while also examining the influence of exposure duration on toxicological outcomes.

MATERIALS AND METHODS

Study design

This study adopted a comparative experimental design to investigate hepatic and renal biochemical toxicity in chickens exposed to a petroleum hydrocarbon–contaminated environment. Biochemical findings obtained from exposed chickens were compared with those of unexposed control birds. The study also evaluated the influence of exposure duration on the severity of hepatorenal toxicity.

Experimental animals and grouping

A total of eighteen chickens were used for the study. Twelve chickens were obtained from an environment characterized by chronic petroleum hydrocarbon contamination associated with prolonged hydrocarbon-related activities. Six chickens obtained from a non-contaminated environment served as controls.

The exposed chickens were grouped according to exposure duration. Six chickens were evaluated after 6 months of exposure, while another six chickens were evaluated after 12 months of exposure. Control birds were similarly categorized according to age and duration. All chickens were maintained under similar feeding and husbandry conditions throughout the study period to minimize environmental and nutritional variation.

Blood sample collection and serum preparation

Blood samples were collected aseptically by venipuncture into clean dry tubes without anticoagulant. The samples were allowed to clot at room temperature and were centrifuged at 3000 rpm for 10 minutes to separate serum. Serum samples were transferred into properly labeled containers and stored at −20 °C pending biochemical analysis. All analyses were completed within 72 hours of sample collection.

Biochemical analysis

Serum AST and ALT activities were determined using the Reitman–Frankel method, while ALP activity was analyzed using the p-nitrophenyl phosphate kinetic method. Serum albumin concentration was measured using the bromocresol green method, and total protein was determined using the Biuret technique. Total and conjugated bilirubin concentrations were estimated using the diazo reaction method.

Renal function was assessed by measuring serum urea and creatinine concentrations using the urease–Berthelot and Jaffe alkaline picrate methods, respectively. Electrolyte analysis included sodium and potassium determination by flame photometry, while chloride and bicarbonate concentrations were measured using standard colorimetric procedures.

Commercially available diagnostic kits were used for all biochemical analyses according to the manufacturers' instructions. Absorbance readings were obtained using a visible spectrophotometer (Model S23A, HELMREASINN, China).

To ensure analytical reliability, all assays were performed in duplicate, and internal quality control sera were included during each analytical run.

Statistical analysis

Data were analyzed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Results were expressed as mean ± standard deviation. Comparisons between exposed and control groups were performed using independent-sample t-tests, while one-way analysis of variance (ANOVA) followed by Tukey's post hoc test was used to assess differences associated with exposure duration. Statistical significance was accepted at $p < 0.05$.

Ethical considerations

All experimental procedures involving animals were carried out in accordance with the National Research Council Guide for the Care and Use of Laboratory Animals (8th edition). Measures were implemented to minimize animal discomfort during handling and sample collection.

RESULTS

Hepatic biochemical alterations

The hepatic biochemical parameters of exposed and control chickens are presented in Table 1. Chickens exposed to petroleum hydrocarbon contamination showed significant hepatic dysfunction when compared with controls ($p < 0.05$) . Serum AST, ALT, and ALP activities were significantly elevated in exposed birds, findings consistent with previous reports of petroleum hydrocarbon–induced hepatocellular injury and membrane destabilization in exposed animals and poultry species [16, 17]. In contrast, serum albumin and total protein concentrations were significantly lower in exposed chickens, suggesting impaired hepatic synthetic function under chronic toxic stress. Total and conjugated bilirubin concentrations were significantly increased following chronic hydrocarbon exposure, indicating possible disruption of bilirubin metabolism and hepatobiliary function [18].

Renal biochemical alterations

The renal and electrolyte findings are presented in Table 2. Exposed chickens demonstrated significant increases in serum urea and creatinine concentrations relative to controls ($p < 0.05$), indicating compromised renal filtration and excretory dysfunction associated with hydrocarbon toxicity [24]. Significant electrolyte imbalance was also observed, including reduced sodium, chloride, and bicarbonate concentrations together with elevated potassium levels. These electrolyte disturbances are consistent with impaired renal tubular handling and altered acid–base regulation reported in chronic renal injury conditions [20, 21].

Effect of exposure duration

The influence of exposure duration on hepatic and renal biochemical parameters is presented in Table 3. Chickens exposed for 12 months exhibited more pronounced biochemical abnormalities than those exposed for 6 months. Hepatic enzymes, bilirubin concentrations, urea, creatinine, and potassium levels were higher after prolonged exposure, whereas albumin, total protein, sodium, chloride, and bicarbonate concentrations were further reduced. These findings indicate progressive hepatorenal toxicity associated with chronic petroleum hydrocarbon exposure and support previous observations that prolonged toxicant exposure may lead to cumulative biochemical and metabolic deterioration in exposed animals [23, 25].

DISCUSSION

The findings of this study demonstrate that chronic exposure to petroleum hydrocarbon contamination induces significant hepatic and renal biochemical dysfunction in chickens. Exposed birds showed marked elevations in liver enzymes together with impaired renal biomarkers and electrolyte imbalance, indicating simultaneous hepatorenal toxicity.

The significant increases observed in AST, ALT, and ALP activities suggest hepatocellular injury and disruption of hepatic membrane integrity. Elevated serum aminotransferases are widely recognized indicators of liver cell damage because injured hepatocytes release intracellular enzymes into circulation [18]. Similar findings have been reported in experimental and environmental petroleum hydrocarbon exposure studies and are commonly associated with oxidative stress, lipid peroxidation, and mitochondrial dysfunction within hepatic tissues [22].

The reductions in albumin and total protein concentrations observed in exposed chickens indicate impairment of hepatic synthetic function. Chronic toxic exposure may interfere with protein biosynthesis and hepatic metabolic activity, thereby reducing serum protein levels. Increased total and conjugated bilirubin concentrations further indicate compromised bilirubin conjugation and excretion, suggesting hepatobiliary dysfunction.

Renal dysfunction was evidenced by elevated serum urea and creatinine concentrations in exposed birds. These biomarkers are commonly used indicators of impaired glomerular filtration and reduced renal clearance. Petroleum hydrocarbons and their metabolites may accumulate within renal tissues and induce oxidative and inflammatory injury, leading to nephrotoxicity [19].

Electrolyte abnormalities observed in this study also support renal tubular dysfunction. Reduced sodium, chloride, and bicarbonate concentrations together with increased potassium levels indicate impaired electrolyte handling and acid–base imbalance associated with chronic renal injury [20, 21].

An important observation in this study was the progressive increase in toxicity with prolonged exposure duration. Chickens exposed for 12 months exhibited more severe biochemical disturbances than those exposed for 6 months, indicating cumulative toxic effects resulting from continuous environmental exposure. Similar time-dependent toxic responses have been documented in studies involving chronic toxicant exposure in poultry and experimental animals [23, 25].

The present findings are consistent with earlier reports demonstrating petroleum hydrocarbon-induced hepatic and renal injury in chickens [26]. Reported elevated hepatic enzymes in environmentally exposed chickens, while documented altered renal biochemical parameters following chronic hydrocarbon exposure. [27] observed evidence of impaired kidney function in chickens exposed to polluted environments. However, unlike many previous investigations that focused on single-organ toxicity, the present study simultaneously evaluated hepatic and renal biochemical responses, thereby providing a broader assessment of systemic petroleum hydrocarbon toxicity.

The findings suggest that chronic petroleum hydrocarbon exposure produces coordinated dysfunction involving both hepatic and renal systems. Simultaneous assessment of liver and kidney biomarkers may therefore provide a more comprehensive evaluation of environmental toxicant exposure in poultry and related biological systems.

Table 1: Liver function parameters of chickens exposed to a petroleum hydrocarbon–contaminated environment after 6/12 months of exposure

Parameter	Exposed chickens (n = 12) Mean ± SD	Control chickens (n = 6) Mean ± SD
AST (IU/L)	43.92 ± 15.14	13.00 ± 4.52
ALT (IU/L)	25.42 ± 22.15	8.17 ± 6.46
ALP (IU/L)	52.67 ± 24.37	25.00 ± 13.84
Albumin (g/L)	53.42 ± 24.23	95.50 ± 14.73
Total protein (g/L)	59.00 ± 23.61	91.50 ± 15.60
Total bilirubin (μmol/L)	11.08 ± 4.46	3.40 ± 1.67
Conjugated bilirubin (μmol/L)	3.06 ± 2.01	0.85 ± 0.76

Values are expressed as mean ± SD. Differences between exposed and control chickens were statistically significant for all parameters ($p < 0.05$). AST, ALT, ALP, total bilirubin, and conjugated bilirubin were increased, while albumin and total protein were decreased in exposed chickens relative to controls.

Table 2: Renal function and electrolyte parameters of chickens exposed to a petroleum hydrocarbon–contaminated environment after 6/12 months of exposure

Parameter	Exposed chickens (n = 12) Mean ± SD	Control chickens (n = 6) Mean ± SD
Sodium (mmol/L)	113.33 ± 38.97	159.50 ± 37.82
Potassium (mmol/L)	3.54 ± 1.23	1.98 ± 1.61
Bicarbonate (mmol/L)	37.08 ± 10.37	68.83 ± 11.94
Chloride (mmol/L)	83.58 ± 25.28	134.00 ± 40.54
Urea (mmol/L)	13.40 ± 9.40	2.42 ± 1.89
Creatinine (mmol/L)	3.21 ± 3.34	1.52 ± 1.94

Values are expressed as mean ± SD. All parameters differed significantly between exposed and control chickens ($p < 0.05$). Exposed chickens showed increased potassium, urea, and creatinine levels, with reduced sodium, chloride, and bicarbonate concentrations, indicating renal dysfunction and electrolyte imbalance.

Table 3: Effect of Exposure Duration (6 vs. 12 Months) on Hepatic and Renal Biochemical Parameters in Petroleum Hydrocarbon–Exposed Chickens

Parameter	6 Months (n = 6) Mean ± SD	12 Months (n = 6) Mean ± SD
Hepatic Parameters		
AST (IU/L)	32.00 ± 10.50	55.84 ± 14.20
ALT (IU/L)	18.00 ± 12.30	32.84 ± 18.70
ALP (IU/L)	40.00 ± 15.20	65.34 ± 20.10
Albumin (g/L)	65.00 ± 20.10	41.84 ± 18.30
Total protein (g/L)	70.00 ± 18.40	48.00 ± 20.20
Total bilirubin (μmol/L)	8.50 ± 3.20	13.66 ± 4.10
Conjugated bilirubin (μmol/L)	2.10 ± 1.40	4.02 ± 1.80
Renal Parameters		
Urea (mmol/L)	8.50 ± 5.10	18.30 ± 7.80
Creatinine (mmol/L)	2.10 ± 2.20	4.32 ± 3.10
Sodium (mmol/L)	130.00 ± 25.40	96.66 ± 28.10
Potassium (mmol/L)	2.80 ± 0.90	4.28 ± 1.10
Chloride (mmol/L)	100.00 ± 20.30	67.16 ± 22.50
Bicarbonate (mmol/L)	45.00 ± 9.20	29.16 ± 8.80

Values are expressed as mean ± SD. Significant differences were observed between chickens exposed for 6 months and those exposed for 12 months ($p < 0.05$). Prolonged exposure was associated with increased AST, ALT, ALP, bilirubin, urea, creatinine, and potassium levels, while albumin, total protein, sodium, chloride, and bicarbonate concentrations decreased, indicating progressive hepatorenal toxicity following chronic petroleum hydrocarbon exposure.

CONCLUSION

Chronic exposure to petroleum hydrocarbon contamination produced significant hepatic and renal biochemical alterations in chickens. Elevated liver enzymes and bilirubin concentrations, together with reduced albumin and total protein levels, demonstrated impaired hepatic function. Increased serum urea and creatinine concentrations accompanied by electrolyte imbalance indicated significant renal dysfunction.

The severity of these abnormalities increased with prolonged exposure duration, demonstrating progressive hepatorenal toxicity associated with chronic petroleum hydrocarbon exposure.

The findings emphasize the importance of evaluating both hepatic and renal biomarkers simultaneously when assessing systemic toxicity resulting from environmental petroleum hydrocarbon contamination.

LIMITATIONS AND FUTURE DIRECTIONS

This study was limited by the relatively small sample size, which may reduce the generalizability of the findings. In addition, the environmental exposure conditions did not permit precise quantification of petroleum hydrocarbon concentrations or identification of individual hydrocarbon constituents responsible for toxicity. The investigation relied primarily on serum biochemical parameters without histopathological or molecular evaluation of hepatic and renal tissues. Consequently, structural tissue alterations and mechanistic pathways could not be directly assessed. Future studies should incorporate larger sample populations, controlled exposure experiments, histopathological evaluation, and molecular investigations to better characterize the mechanisms underlying petroleum hydrocarbon-induced hepatorenal toxicity. Assessment of oxidative stress pathways, inflammatory responses, and reversibility following cessation of exposure would further improve understanding of chronic hydrocarbon toxicity in poultry.

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