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Full length article

Cypermethrin–Induced Nephrotoxicity in Rabbits: A Histopathological Study

Ateeq M.J. Al-Arami , Abdulkarem A.Y. Al-Rezaki

Department of Biology, Faculty of Education, University of Thamar, Yemen
Corresponding Author: histo.a2015@gmail.com

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ABSTRACT

Cypermethrin is an important type II pyrethroid pesticide widely used to protect crops against pests and insect infestations, but it is also associated with environmental pollution and health hazards. The present study was designed to investigate histological changes in the kidneys of male rabbits following exposure to cypermethrin. A total of 18 adult male rabbits were used. The rabbits were randomly divided into three groups of six animals each. The first group(G1) served as the control and received normal saline daily. The second group(G2) was administered cypermethrin orally at a dose equivalent to 1/10 of the LD_{50} (66.5 mg/kg body weight) once daily for 21 consecutive days. The third group(G3) received a higher oral dose equivalent to 1/5 of the LD_{50} (133 mg/kg body weight) under the same schedule. Histological examination of the treated groups revealed multiple pathological alterations compared to control group, Cypermethrin caused degeneration of renal corpuscles, hydropic changes, necrosis, inflammation, fatty degeneration, amyloidosis, dilation of renal tubules, congestion, fibrosis, steatosis, and tubular degeneration. In conclusion, cypermethrin produced significant histological damage in the kidneys of male rabbits. Further research is required to elucidate the molecular and cellular mechanisms underlying cypermethrin-induced nephrotoxicity.

Keywords: Cypermethrin, Histopathological**,** Kidney, Toxicity, Rabbits.

INTRODUCTION

Pyrethroids, synthetic derivatives of natural pyrethrins, represent the most widely used class of pesticides globally due to their high efficacy, relatively low mammalian toxicity, and rapid biodegradation in the environment (Ahmad et al., 2009). They are generally classified into two groups: Type I (without an alpha-cyano group) and Type II (with an alpha-cyano group), which differ in their structural features and the behavioral effects they produce (Spencer et al., 2005; Wolansky et al., 2006; Saka et al., 2011). Despite their widespread use, pyrethroid pesticides, including cypermethrin, are recognized as major environmental pollutants. Cypermethrin toxicity has been documented in numerous experimental and clinical studies (Idris et al., 2012; USEPA, 2009; Singh et al., 2012).

Cypermethrin, a Type II pyrethroid, was first synthesized in 1974 and introduced commercially in 1979 (WHO, 1989). It has a high potential to accumulate within food chains, thereby contributing to toxic effects in exposed organisms (Muthuviveganandavel et al., 2011; Sangha et al., 2013). In humans, cypermethrin poisoning manifests as facial burning and tingling (paraesthesia), dizziness, headaches, nausea, anorexia, fatigue, and urinary incontinence. With higher exposure, symptoms may progress to muscle twitching, drowsiness, coma, and seizures (Chakravarthi et al., 2007; Gheshlaghi et al., 2011). In laboratory animals, toxic signs include pawing, burrowing, salivation, tremors, writhing, and seizures (Ullah et al., 2006; Grewal et al., 2010).

Kidney function can be assessed through the measurement of metabolic waste products such as urea

and creatinine, which are normally excreted via the renal system (Garba et al., 2007). Elevated levels of these metabolites in blood indicate cellular damage and impaired renal clearance (Aslam et al., 2010). Thus, urea and creatinine serve as sensitive biochemical markers for diagnosing renal injury. Reduced serum protein levels may result from renal or intestinal protein loss, hemorrhage, malabsorption, or liver dysfunction (Khan, 2008).

Exposure to cypermethrin has been linked to a wide range of adverse effects, including anemia, impaired blood coagulation, neurotoxicity, paralysis, jaundice, hepatic fibrosis, renal dysfunction, cancer, genetic abnormalities, birth defects, and reproductive disorders such as impotence and infertility (Yousef et al., 2003a,b). Long-term exposure has also been associated with dopaminergic neurodegeneration in adult rats (Singh et al., 2012). Several studies have demonstrated that cypermethrin induces diverse physiological, biochemical, toxicological, and histological alterations in experimental animals (Kumari et al., 2002; Hussain et al., 2009).

Although extensive research have addressed hepato-renal pathology in pyrethroid-exposed animals, humans, and environmental systems, the present study aims to provide a detailed description of kidney pathology in animals exposed to cypermethrin under local environmental conditions.

MATERIALS AND METHODS

This experimental study was conducted in the Department of Biology, Faculty of Education, University of Thamar, Yemen.

Experimental Animals

Eighteen mature healthy male rabbits (*Oryctolagus cuniculus*) weighing between 900–1200 g, were obtained from the local market of Dhamar, Yemen.

Chemicals

The synthetic Cypermethrin Pyrethroid (α-Cyano-3-phenoxy benzyl 3-(2, 2-dichloro vinyl)-2,2-dimethyl cyclopropane carboxylate). Cypermethrin(10% EC) produced by Vapco company- Amman Jordan and purchased from authorized dealers in local market at Dhamar, Yemen**.**

Study Design

The experimental animals were housed under uniform management conditions in the animal facility of the Biology Department, Faculty of Education, University of Thamar, Yemen. All animals were healthy and acclimatized for 14 days prior to the experiment.

The rabbits were randomly divided into three groups of six animals each. The first group(G1) served as the control and received normal saline daily via disposable syringe. The second group(G2) was administered cypermethrin orally at a dose equivalent to 1/10 of the LD50 (66.5 mg/kg body weight) once daily for 21 consecutive days. The third group(G3) received a higher oral dose equivalent to 1/5 of the LD50 (133 mg/kg body weight) under the same schedule. Cypermethrin was dissolved and diluted in distilled water to achieve the required concentrations.

Throughout the study, animals were maintained under daily observation in well-ventilated housing at room temperature (25–27°C), relative humidity (50 ± 15%), and a 12-hour light/dark cycle. They were provided with rabbit chow and water *ad libitum*, ensuring equal quantities of food and water across all groups.

2.5. Specimen processing and Histolopathological examination

At the end of the experiment, all rabbits were subjected to necropsy. Kidneys were excised, rinsed in normal saline, and fixed in 10% formalin for 24 hours. Following fixation, tissues were washed in tap water, dehydrated through ascending grades of ethanol, cleared in xylene, and embedded in paraffin wax (melting point 50–56°C). Paraffin blocks were sectioned at 6 µm thickness using a rotary microtome. The sections were stained with Harris hematoxylin and eosin, examined under a light microscope, and photographed using an automated photomicrographic system according the techniques described by Jaber and Al-Bakri (2018); Al-Hamawandy and Al-Bakri (2020); Jaber et al. (2020).

RESULTS

The findings of this study can be summarized as follows: Kidney sections from rabbits in the control group, which received physiological saline, displayed normal renal tubules and glomeruli (Figure 1). In the group treated with cypermethrin at a dose of 66.5 mg/kg body weight for 21 days, several pathological alterations were observed, including degeneration of renal corpuscles, hydropic changes, necrosis, inflammation, fatty degeneration, amyloidosis, dilation of renal tubules, congestion, and fibrosis (Figures 2–6). In contrast, rabbits exposed to the higher dose of cypermethrin (133 mg/kg body weight for 21 days) exhibited marked histological changes such as steatosis, inflammation, degeneration of kidney tubules, and amyloidosis (Figures 7–8).

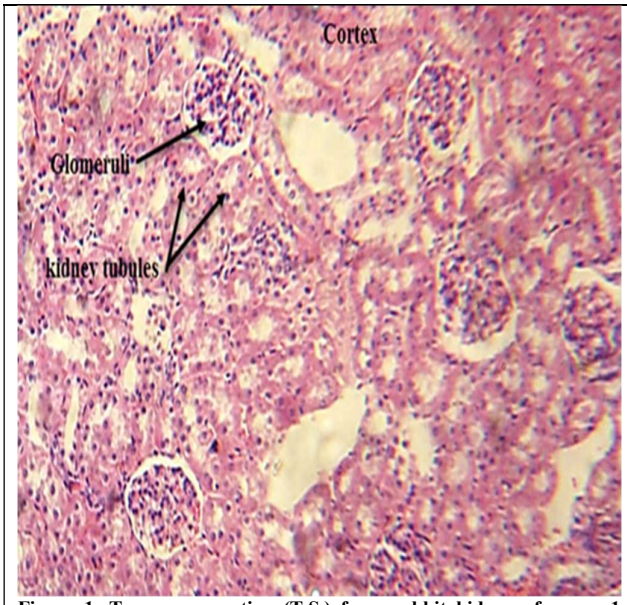

Figure 1. Transverse section (T.S.) from rabbit kidney of group 1 (control) treatment with physiological solution for 21 days shows: normal renal glomeruli and renal tubules (RT) (H&E Stain, 100X).

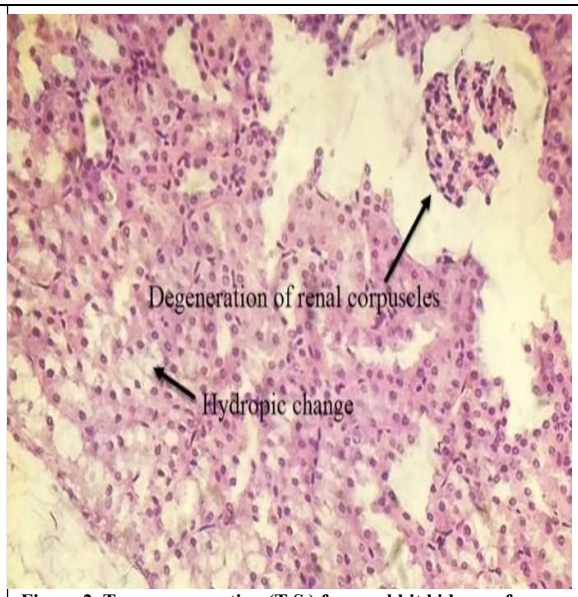

Figure 2. Transverse section (T.S.) from rabbit kidney, of group 2, treated with 66.5 mg/ kg-^{1}.body.weight/ day Cypermethrin for 21 day shows: Degeneration of Renal Corpuscles and Hydropic Change (H&E Stain, 100x)

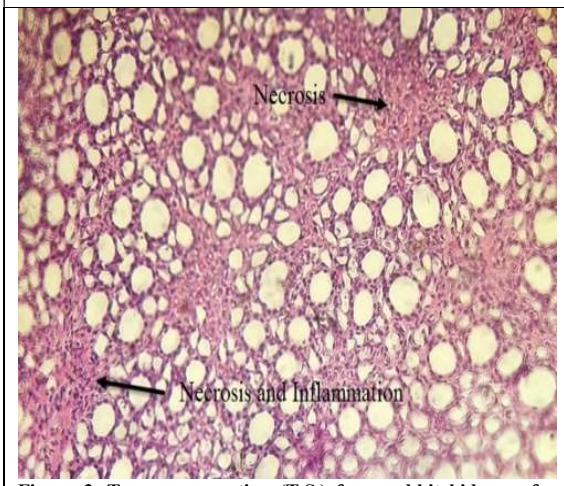

Figure 3. Transverse section (T.S.) from rabbit kidney, of group 2, treated with 66.5 mg/ kg-^{1}.body.weight/ day Cypermethrin for 21 day shows : Necrosis and Inflammation (H&E Stain, 100x) .

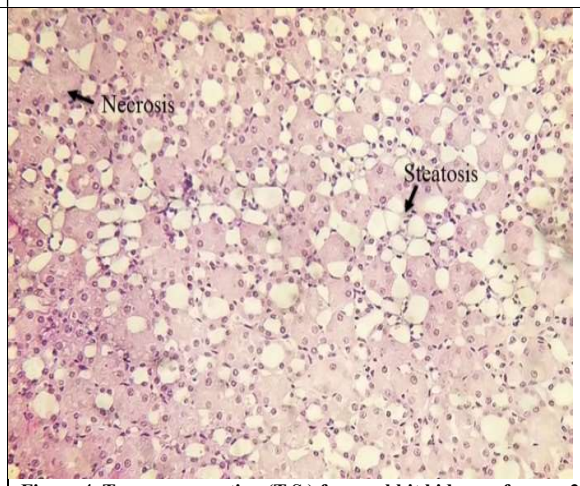

Figure 4. Transverse section (T.S.) from rabbit kidney, of group 2, treated with 66.5 mg/ kg-^{1}.body.weight/ day Cypermethrin for 21 day shows :Steatosis and Necrosis (H&E Stain, 100x)

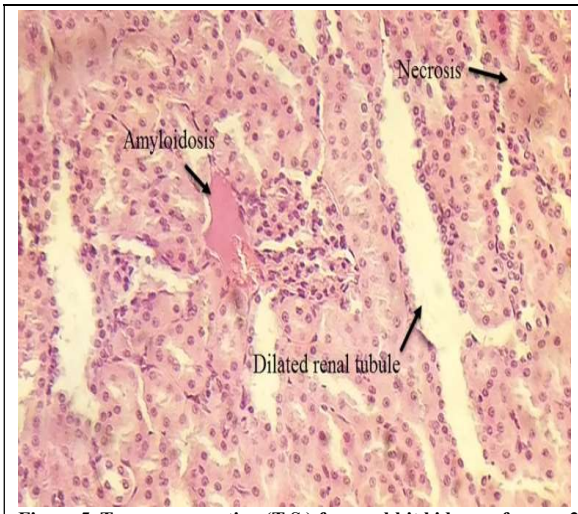

Figure 5. Transverse section (T.S.) from rabbit kidney, of group 2, treated with 66.5 mg/ kg-^{1}.body.weight/ day Cypermethrin for 21 day shows : Dilated Renal Tubule, Necrosis and amyloidosis (H&E Stain, 400x) .

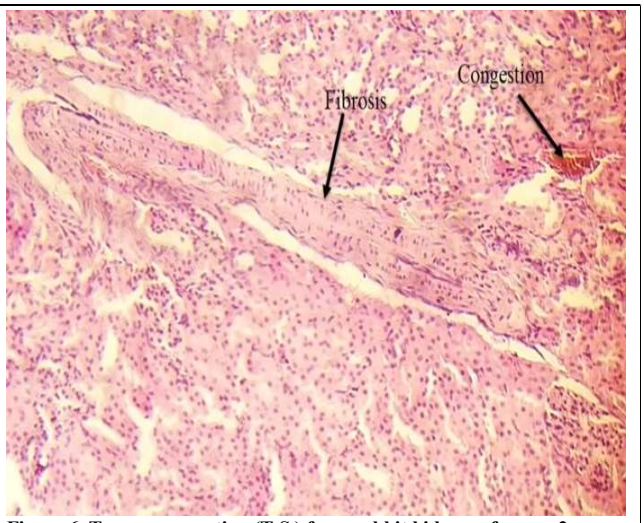

Figure 6. Transverse section (T.S.) from rabbit kidney, of group 2, treated with 66.5 mg/ kg-^{1}.body.weight/ day Cypermethrin for 21 day shows : Congestion and Fibrosis (H&E Stain, 400x) .

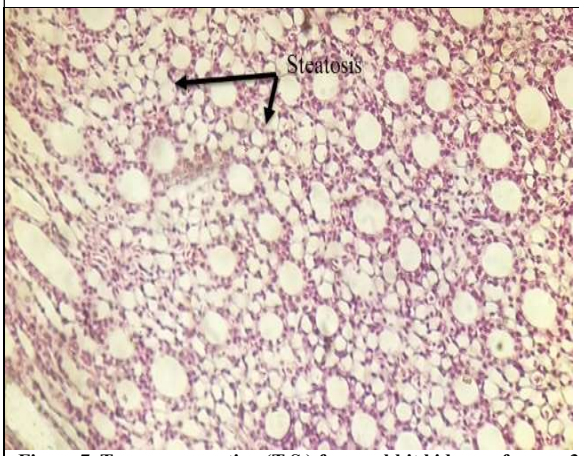

Figure 7. Transverse section (T.S.) from rabbit kidney, of group 3, treated with 133 mg/ kg-^{1}.body.weight/ day Cypermethrin for 21 day shows : Steatosis (H&E Stain, 100x).

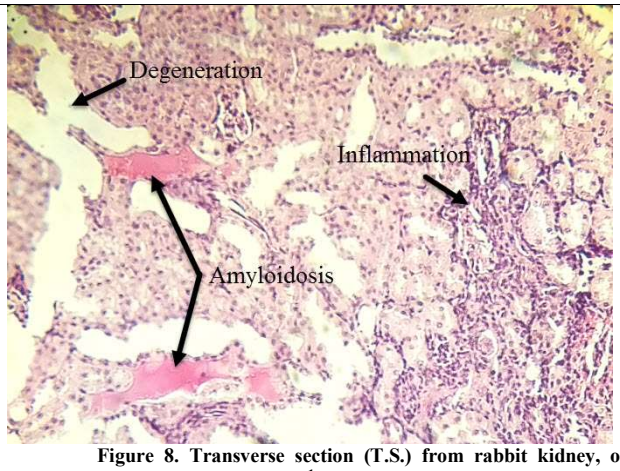

Figure 8. Transverse section (T.S.) from rabbit kidney, of group 3, treated with 133 mg/ kg-^{1}.body.weight/ day Cypermethrin for 21 day shows : Inflammation, Degeneration and Amyloidosis (H&E Stain, 400x).

DISCUSSION

The present investigation revealed marked histopathological alterations in renal tissues following cypermethrin exposure. These changes were consistent with the observations of Shuklan et al. (2023). Similar findings were reported by Manna et al. (2004) and Hussien et al. (2013), who noted that the lipophilic nature of cypermethrin enables its penetration through cellular membranes, resulting in structural damage and leakage of intracellular enzymes. Grewal et al. (2010) further demonstrated that cypermethrin intoxication disrupts renal architecture, leading to glomerular shrinkage, tubular necrosis, hemorrhage, and epithelial sloughing in the convoluted tubules. Elevated plasma levels of urea and creatinine, biomarkers of renal dysfunction, were also associated with these histopathological changes (Grewal et al., 2009).

Comparable toxic effects were observed in the cypermethrin-treated groups of the current study. Shivanoor and David (2014) reported that deltamethrin exposure caused proximal tubular dilation, inflammation, cellular infiltration, and necrosis due to epithelial desquamation. Likewise, Raj et al. (2013) documented medullary congestion in the kidneys of mice following oral administration of a cypermethrin–endosulfan combination. Faten and Abdulhadi (2016) also described extensive renal damage in albino rats, including Bowman's space dilation, tubular cell degeneration, glomerular atrophy, hemorrhage, epithelial sloughing, glomerulonephritis, tubular lining necrosis, eosinophilic material deposition, tubular destruction, inflammatory cell aggregation, disorganization of renal

architecture, glomerular destruction, and severe tubular necrosis.

The accumulation of cypermethrin in renal tissues likely contributes to these toxic manifestations. Prashanth (2011) suggested that the upregulation of metallothionein, a metal-binding protein, in response to cypermethrin exposure may impair organelle function within renal cells, thereby exacerbating tissue injury. Mamun et al. (2014) similarly reported that cypermethrin intoxication adversely affects kidney structure, causing glomerular shrinkage, tubular necrosis, hemorrhage, and epithelial sloughing. Once absorbed into the bloodstream, cypermethrin undergoes hepatic metabolism, producing reactive intermediates that bind to cellular macromolecules, inducing oxidative stress, lipid peroxidation, and subsequent cellular damage. Latif et al. (2011) observed renal lesions in cypermethrin-treated rabbits, including pyknotic nuclei, necrosis, epithelial sloughing, cast deposition, and increased urinary space.

Pyrethroids such as cypermethrin are known to induce oxidative stress by generating free radicals, which compromise the glomerular filtration barrier. This damage alters the negative charge of structural components—including endothelium, podocytes, and basement membrane—either neutralizing or reversing it, thereby increasing protein permeability. Degenerative changes in the kidneys result in detachment of epithelial cells from tubular basement membranes. These detached cells, together with leaked proteins, form epithelial casts within renal tubules (Khan et al., 2009).

Finally, the findings are corroborated by Kundu et al. (2026), who reported widespread inflammatory cell clusters, severe hypertensive glomerulosclerosis, and extensive tubular necrosis in the kidneys of cypermethrin-exposed mice. Additional pathological features included blackish glomerular discoloration, marked hemorrhage, and tubular enlargement. High-resolution histopathological imaging further revealed abnormal glomerular architecture and a significant reduction in viable cell counts in both hepatic and renal tissues of the treated group.

CONCLUSION

It could be concluded from this study that administration of cypermethrin caused significant histopathological changes in renal tissues of rabbits. Additionally, our results demonstrate that the nephrotoxicity intensity correlates with the increase in dose of cypermethrin administration.

RECOMMENDATIONS

Further research is required to clarify the molecular and cellular mechanisms underlying cypermethrin-induced nephrotoxicity. Regulatory measures limiting the use of cypermethrin in agricultural and veterinary practices should be considered to reduce human and animal exposure. In addition, systematic monitoring of cypermethrin residues in the environment and food supply is essential for evaluating potential long-term health risks.

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CONTRIBUTORS OF UTHORS

AMJ. Al-Arami and A AY Al-Rezaki have equally contributed in the designing, carried out, writing the first draft and final of the manuscript. Both authors have reviewed and approved the last submitted version.

COMPETING INTERESTS

Authors declare that there is no conflict of interest.

ETHICS APPROVAL

This study was performed in line with National Institutes of Health(NIH) guidelines for the care and use of laboratory animals, and approved by the Institutional Ethics Committee at the Faculty of education, Thamar University.

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None.

DATA AVAILABILITY STATEMENT

The data are available within the article.

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السمية الكلوية الناجمة عن السايبرميثرين في الأرانب: دراسة نسيجية مرضية

عتيق محمد جبران العرامي، عبدالكريم على يحي الرزاقي
قسم علوم الحياة ، كلية التربية ، جامعة ذمار
للمراسلة : histo.a2015@gmail.com

الملخص

تم تصميم هذه الدراسة للتحقق من التغيرات النسيجية في كلى الأرانب بعد التعرض لمبيد السيبرمثرين، وهو أحد مبيدات البيرثرويد من النوع الثاني، ويُستخدم على نطاق واسع لحماية المحاصيل من الآفات والحشرات، لكنه يرتبط أيضًا بالتلوث البيئي والمخاطر الصحية. استخدم في الدراسة 18 أرنبًا ذكرًا بالغًا، وتم تقسيمها عشوائيًا إلى ثلاث مجموعات، تضم كل مجموعة ستة حيوانات. المجموعة الأولى كانت مجموعة ضابطة وتلقت محلول ملحي طبيعي يوميًا. المجموعة الثانية أعطيت السيبرمثرين عن طريق الفم بجرعة تعادل 10/1 من الجرعة المميتة النصفية (66.5 ملغ/كغ من وزن الجسم) مرة يوميًا لمدة 21 يومًا متتاليًا. أما المجموعة الثالثة فقد تلقت جرعة أعلى تعادل 5/1 من الجرعة المميتة النصفية (133 ملغ/كغ من وزن الجسم) بنفس الجدول الزمني. أظهر الفحص النسيجي للمجموعات المعالجة تغيرات مرضية متعددة مقارنة بالمجموعة الضابطة؛ حيث تسبب السيبرمثرين في تنكس الكبيبات الكلوية، تغيرات مائية، نخر، التهابات، تنكس دهني، داء النشواني، توسع الأنابيب الكلوية، احتقان، توليف، دهن كبدي، وتنكس أنبوبي. **خلصت الدراسة الى ان** السيبرمثرين أحدث أضرارًا نسيجية كبيرة في كلى الأرانب التي خضعت للدراسة. ينبغي أجراء دراسات اضافية لتوضيح الآليات الجزيئية والخلوية للسمية الكلوية الناجمة عن السايبرمثرين.

الكلمات المفتاحية: السيبرميثرين، التغيرات النسيجية المرضية، الكلى، السمية، الأرانب.

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