Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2024.322700.3589 Volume 31, Issue 1, January. 2025
Manscript ID :ZUMJ-2409-3589
DOI : 10.21608/zumj.2024.322700.3589

Original article

Sub-Acute Toxic Effects of Acetamiprid on Epididymis and Seminal Vesicles of Adult Albino Rats

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Submit Date 21-09-2024
Accept Date 09-10-2024

ABSTRACT:

Background: Acetamiprid toxicity is now a public health problem, it's critical to investigate how hazardous it is to the epididymis and seminal vesicles. Therefore, using biochemical and histological analyses, we aimed to assess the harmful effects of multiple doses (1/5 and 1/20 of LD50) of sub-acute acetamiprid exposure on the epididymis and seminal vesicles in adult albino rats.

Methods: The study included 28 adult male albino rats, divided equally into: Group I (negative control group): received only regular diet and tab water orally. Group II (positive control group): gavaged orally with distilled water. Group III (Acetamiprid 1/5 of LD50): gavaged orally with acetamiprid at a dose of 40 mg/kg/day) dissolved in distilled water and group IV (Acetamiprid 1/20 of LD50) gavaged orally with acetamiprid at a dose of 10 mg/kg/day) dissolved in distilled water for 4 weeks. LD50 of acetamiprid 200 mg/kg in water.

Results: In acetamiprid-treated groups, there was a non-significant weight decrease of epididymis and seminal vesicles when compared to the negative control group. Also, acetamiprid administration significantly increased tissue levels of MDA and significantly decreased tissue levels of TAC. There was very highly significant difference in TAC and MDA levels in tissue of acetamiprid 1/5 of LD50 treated group when compared to acetamiprid 1/20 of LD50 treated group ($P < 0.001$). Histopathological studies revealed that acetamiprid exposure caused epididymal and seminal vesicular tissue damage with more damage in 1/5 of the LD50 group compared to 1/20 of LD50 group.

Conclusion: Sub-acute acetamiprid exposure causes significant biochemical and histopathological damage to epididymis and seminal vesicles of adult albino rats with more damage in 1/5 of the LD50 group compared to 1/20 of LD50 group.

Keywords: Sub-acute acetamiprid, Male reproductive system toxicity, Epididymis, Seminal vesicles, Adult male albino rats.

INTRODUCTION

Pesticides are chemical formulations that are being used more and more in public health, agriculture, and animal husbandry to eradicate diseases transmitted by insects and to destroy weeds, fungi, and insects [1]. These pesticides are hazardous to humans and animals at varying amounts, in addition to insects and other pests. The persistent application of pesticides poses significant risks to the physiological operations of multiple body systems [2].

When handling and combining pesticides on crops, farmers and pesticide applicators come into intimate contact with the chemicals. In addition, exposure can occur from eating polluted food, drinking tainted water and contaminated soil. Pesticide toxicity is mostly caused by low educational

attainment among agricultural laborers, inadequate knowledge about pesticide safety and inadequate personal protection when using pesticides [3]. I-The early 1990s saw the introduction of a brand-new class of active substances. Neonicotinoid chemicals belong to these classes [4]. These pesticides belong to a novel family that selectively agonist the nicotinic acetylcholine receptor (nAChR) in insects' central nervous systems [5]. Because they are so easy to use, neonicotinoids are favored over other insecticides. Additionally, they have high efficacy, broad-spectrum insect toxicity, and relatively low acute toxicity to non-target aquatic and terrestrial organisms [6].

Neonicotinoids have low sorption in soil, high water solubility, and comparatively extended half-lives in the soil. These elements play a part in the pesticides' environmental persistence and movement. There are several metabolic and systemic dysfunctions that can result from the residual quantities of these chemicals in exposed spray workers. Neonicotinoids normally biodegrade quickly, but they might linger and perhaps build in dry soils with a high organic matter concentration and low temperatures [7]. Some neonicotinoids have been found to translocate into pollen, vegetables, and fruits because washing does not completely remove neonicotinoid pesticides from fruits and vegetables that are consumed. Consequently, eating these items could expose people to these chemicals. Processing and washing can lessen neonicotinoid residues, but they cannot completely remove them [8].

One of the neonicotinoid pesticides, acetamiprid, is widely used in agriculture and household settings to control a wide range of insects. Farmers in Egypt use acetamiprid extensively because it has revealed effectiveness in controlling pests of rice, potato, and tomato plants, which are among the country's main crops. According to reports, acetamiprid can build up in plants and contaminate water, which could be harmful to people's health. In soil, acetamiprid has a half-life of 31–450 days [9]. There have been harmful consequences of acetamiprid on numerous organs and systems. On the other hand, the potential toxicity of acetamiprid on the male reproductive system remains unsatisfied [10].

METHODS

1. Chemicals:

Acetamiprid: Acetamiprid, white crystalline solid, CAS Number: 160430-64-8, was purchased from Sigma Aldrich Co. branch in Cairo, Egypt. Distilled water: It was obtained from El- Nasr Co, Egypt and used as a solvent agent for acetamiprid.

Methods:

The Institutional Animal Care and Use Committee (IACUC), Zagazig University has approved the design of this vivo- experiment with approval number (ZU-IACUC/3/F/90/2023). Twenty-eight adult male albino rats weighing between 150 and 200 grams apiece were used. The rats were acquired from Zagazig University's Faculty of Medicine's Animal House. The study was carried out at Zagazig University's Faculty of Medicine's Histology, Forensic Medicine, and Clinical Toxicology departments, as well as in the animal house.

According to "The Guide for the Care and Use of Laboratory Animals," all animals were cared for in accordance with ethical regulations and animal care guidelines. [11]. Before the testing, every animal seemed to be in good health. For a duration of seven days, they underwent passive preliminary procedures to help them adjust to their new surroundings, ensure their physical health, and rule out any sick animals.

The animals were kept in separate plastic cages free of chemical pollutants, with controlled environments that included a 12-hour light cycle. An ambient temperature range of 22 ± 2 ºC, and a relative humidity of 50 ± 5% [12].

On other days, to prevent overcrowding and isolation, when the cages were cleaned, the bedding—soft wood shavings—was changed out. The rats were fed a well-balanced diet that included all the elements needed to keep them healthy both before and after the medicines were administered. It consisted of bread and barley. Water was provided in distinct, immaculate containers.

Experimental Procedure:

The 28 mature male albino rats weighed between 150 and 200 grams each were divided equally into four groups, with seven rats in each group: Group I (negative control group): To assess the fundamental parameters, each rat in this group was given an oral tab of water along with its regular feed. Group II (positive control group): Each rat in this group gavaged orally with distilled water (1mL for each rat) Group III (Acetamiprid 1/5 of LD50): Each rat received an oral dose of 40 mg/kg/day (1/5 of LD50) of acetamiprid, which was dissolved in distilled water. Group IV (Acetamiprid 1/20 of LD50): Every rat was given an oral dose of 10 mg/kg/day of acetamiprid that had been dissolved in

distilled water. LD50 of acetamiprid is 200 mg/kg in water [13]

Throughout the course of the study, all groups of treated and control rats had their animals closely monitored for any potential mortality. Every day, new dosages of every medication were prepared, and they were modified every week to account for variations in body weight.

At the end of the experiment, after 24 hours of the last dose of each medication, the rats in each group were anesthetized by inhaling diethyl ether. Following this, venous blood samples were collected from the retro-orbital plexus of the animals by capillary glass tubes using light ether anesthesia [14] and the animals were executed by beheading [15] and subjected to the following: weight measurement of the epididymis and seminal vesicles, assessment of oxidative stress biomarkers in tissue (epididymis and seminal vesicles): Malondialdehyde (MDA) and TAC (total antioxidant capacity) as antioxidant marker, and histological examinations by light microscope: for epididymis and seminal vesicles by using Hematoxylin and Eosin stain (H&E).

Samples collection:

At the end of the 4th week, 24 hours after the last dose rats were exposed to:

1-Tissue sample collection:

The epididymis and seminal vesicles were promptly removed from the euthanized rats and physically examined to determine their weight (weights were measured according to OCED Guidelines NO. 407, tissues were trimmed of any adherent tissue and their weights were taken as possible after dissection, the weight of the sides together was taken and epididymal fat pad was removed) portion of the tissue from these organs was promptly preserved in 10% neutral buffered formalin for histopathological examination of changes in these organs. The remaining portions of these tissues were sent on dry ice and kept at -80 °C to create tissue homogenates for the examination of oxidative stress biomarkers (Homogenization procedure of epididymal and seminal vesicular tissue was carried out for 2 min at 12 861×g in 5 mL of ice-cold Tris-HCl buffer (0.01 mol L^{-1}, pH 7.4) containing 0.01% EDTA- 2Na, 0.01 mol L^{-1} saccharose, and 0.8% NaCl. All procedures were performed at 4°C. Homogenate, supernatant and extracted samples were prepared to determine the activities of malondialdehyde (MDA) and TAC. [16].

Determination of the MDA level in tissue:

To ascertain the MDA (ng/mg protein) concentration in tissue samples, the procedure was outlined by Jain et al. [17].

Determination of Total antioxidant capacity (TAC):

Tissue TAC (nmol / g tissue) was colorimetrically analyzed using the technique of Koracevic and Koracevic [18].

Methods applied for histopathological studies:

1-Preparation and staining:

A. Paraffin technique according to Bancroft and Gamble [19].
B. Hematoxylin and Eosin stains according to Kiernan [20].

Statistical analysis:

On an IBM PC CPU, the data was analyzed using SPSS version 22 (Statistical Package of Social Science). The data were evaluated using suitable statistical methods, such as: LSD test: for comparison between two groups and one way analysis of variance (ANOVA or F-test): or comparison of means of more than two groups. The significance level (P-value) of “F” was obtained from “F” tables.

RESULTS

A) Weight of epididymis and seminal vesicles: -

When comparing the acetamiprid treated groups for 4 weeks to the negative control groups, the mean value of organs weight revealed non-significant decrease. (table 1).

B) Oxidative stress biomarkers:

1-MDA level in epididymal and seminal vesicular tissues:

The current study's findings indicate that the acetamiprid treated groups (1/5 of LD50 and 1/20 of LD50) had a statistically significant increase in mean values of MDA levels in tissues (epididymis and seminal vesicles) compared to the values of the negative control (tables2). There was very highly significant difference in MDA levels in tissue of acetamiprid 1/5 of LD50 treated group when compared to acetamiprid 1/20 of LD50 treated group ($P < 0.001$) by using LSD test (table 3).

2-TAC level in epididymal and seminal vesicular tissues:

The current study's findings indicate that the acetamiprid treated groups (1/5 of the LD50 and 1/20 of LD50) had a statistically significant decrease in mean values of TAC levels in tissues (epididymis and seminal vesicles) compared to the values of the negative control (tables 2). There was

very highly significant difference in TAC levels in tissue of acetamiprid 1/5 of LD50 (high dose) treated group when compared to acetamiprid 1/20 of LD50 (low dose) treated group (P < 0.001) by using LSD test (table 3).

D) Histopathological results:

1) Histopathological changes in epididymal tissue:

I. Macroscopic examination:

When the epididymis of each group was examined macroscopically, it showed no aberrant masses or cystic abnormalities, and appearance was normal.

II. Light microscopic examination:

Negative and positive control groups (group I & II): Examination of H&E-stained sections of epididymis in control group showed that it was formed of many tubules covered by connective tissue capsule and containing spermatozoa in their lumina. These tubules were lined by high epithelial lining with narrow lumen in the proximal parts and low epithelium and wide lumen in the distal parts of epididymis. Ducts were lined by pseudostratified columnar epithelium with stereocilia; Principal cells had apical stereocilia and smaller basal cells resting on the basement membrane. The ducts were surrounded by stroma formed mainly of smooth muscle fibers and connective tissue (Figs. 1 A, B). Group (III) acetamiprid 1/20 of LD50: Examination of H&E-stained sections of rat epididymis from the acetamiprid 1/20 of LD50 (low dose) treated group revealed thick capsule, congested blood vessels, wide interductal spaces, the ductal epithelium had dark nuclei and decrease in the number of spermatozoa in some ducts (Figs. 2 A, B). Group (IV) acetamiprid 1/5 of LD50: Examination of H&E-stained sections of rat epididymis from the acetamiprid 1/5 of LD50 treated group revealed wide interductal spaces, the ductal epithelium had small dark nuclei and decrease in the number of spermatozoa in some ducts and others had proliferation in their epithelial lining (Figs. 3A, B).

2) Histopathological changes in seminal vesicular tissue:

I. Macroscopic examination:

The seminal vesicles of all groups exhibited a normal appearance devoid of any notable alterations in size, cystic changes, or anomalous masses.

II. Light microscopic examination: -

Negative and positive control (Groups I & II): Examination of H&E-stained sections of seminal vesicles in the control group detected that they were formed of highly folded mucosa, underlying layer of loose connective tissue (lamina propria), musculosa and adventitia. Mucosa was lined by pseudostratified columnar epithelium having tall columnar nuclei and resting on a basement membrane. Musculosa was formed of inner circular and outer longitudinal smooth muscle fibers and adventitia was a layer of fibroelastic connective tissue (Figs. 1S A, B). Group (III) acetamiprid 1/20 of LD50: Examination of H&E-stained sections of rat seminal vesicles from the acetamiprid 1/20 of LD50 treated group revealed disorganized mucosa and musculosa; decrease in the height of the mucosa, shedding of the epithelium and acidophilic hyaline material in the lumen. (Figs. 4 A, B). Group (IV) Acetamiprid 1/5 of LD50: - Examination of H&E-stained sections of rat seminal vesicles from the acetamiprid 1/5 of LD50 (high dose) treated group revealed marked decrease in the mucosal height, wide lamina propria, and disorganized musculosa in some sections. Other sections showed shedding of epithelium lining mucosa and thickened adventitia (Figs. 5 A, B).

Table (1): Statistical comparison among different studied groups regarding weight mean values of epididymis and seminal vesicles using one-way ANOVA.

Weight (gm)	Negative control	Acetamiprid 1/20 of LD50 group	Acetamiprid 1/5 of LD50 group	F	p-value
	mean± SD				
Epididymis	1.57±0.03	1.55±0.52	1.48±0.70	0.060	0.942 NS
Seminal vesicles	0.38±0.04	0.35±0.02	0.36±0.02	2.0417	0.158 NS

N.B All values are expressed as mean± SD. (SD: standard deviation), Number of rats in each group=7 rats, NS: non-significant (P >0.05).

Table (2): Statistical comparison among different studied groups regarding tissue oxidative stress markers mean values after 4 weeks using one-way ANOVA.

Oxidative stress markers	Negative control	Acetamiprid 1/20 of LD50 group	Acetamiprid 1/5 of LD50 group	F	p-value
	mean± SD				
Epididymis MDA (ng/mg protein)	6.80±0.42	7.38±0.48[a]	11.32±0.46[a]	200.991	<0.001**
Epididymis TAC (nmol / g tissue)	0.91±0.11	0.80±0.04[a]	0.36±0.04[a]	105.801	<0.001**
seminal vesicles MDA (ng/mg protein)	6.80±0.42	7.71±0.75[a]	11.32±0.47[a]	124.088	<0.001**
seminal vesicles TAC (nmol / g tissue)	0.98±0.15	0.82±0.05[a]	0.41±0.05[a]	58.156	<0.001**

N.B All values are expressed as mean± SD. (SD: standard deviation), Number of rats in each group=7 rats, **: very highly significant (P<0.001) [a] Significant versus control group

Table (3): Least significance difference (LSD) for comparison between mean values of oxidativ1e stress markers in epididymal and seminal vesicular tissues among different studied groups after 4 weeks.

Parameters	Group	Acetamiprid 1/20 of LD50 group	Acetamiprid 1/5 of LD50 group
Epididymis MDA (ng/mg protein)	-ve control	0.028*	<0.001**
	Acetamiprid 1/20 of LD50 group		<0.001**
Epididymis TAC (nmol / g tissue)	-ve control	0.016*	<0.001**
	Acetamiprid 1/20 of LD50 group		<0.001**
seminal vesicles MDA (ng/mg protein)	-ve control	0.018*	<0.001**
	Acetamiprid 1/20 of LD50 group		<0.001**
seminal vesicles TAC (nmol / g tissue)	-ve control	0.021*	<0.001**
	Acetamiprid 1/20 of LD50 group		<0.001**

*: statistically significant (p<0.05)

**: statistically very highly significant (P<0.001)

Number of rats in each group= 7rats.

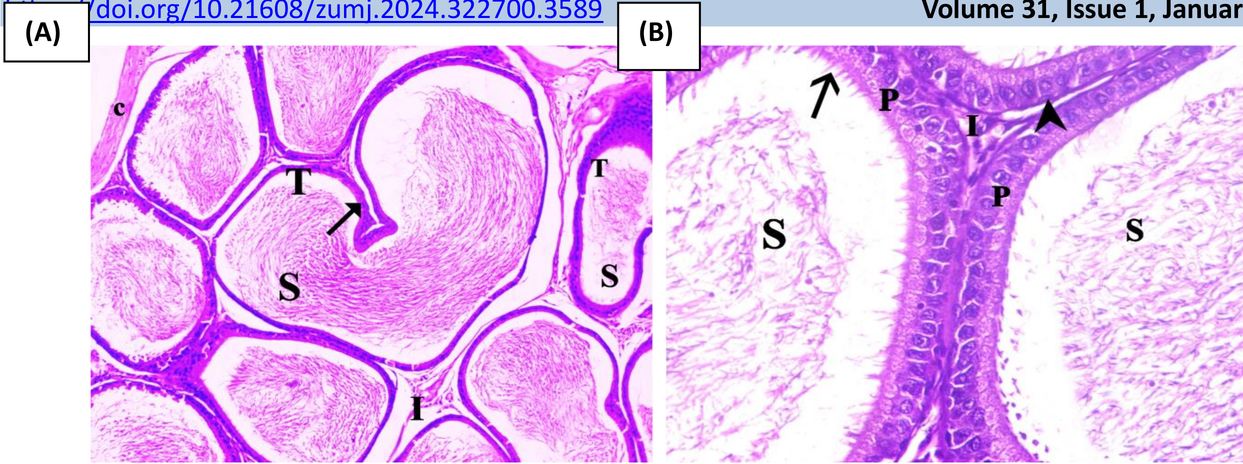

Figure (1 A, B): Photomicrographs of H&E-stained sections in control epididymis of adult male albino rat showing that it is formed of many tubules (T) covered by A tissue capsule and containing spermatozoa (S) in their lumina. These tubules are lined by pseudostratified columnar epithelium with stereocilia (arrow); Principal cells (P) have apical stereocilia and smaller basal cells (arrow head) resting on the basement membrane. There is connective tissue interstitium (I) inbetween the ducts. (A: H&E×100, B: H&E×400)

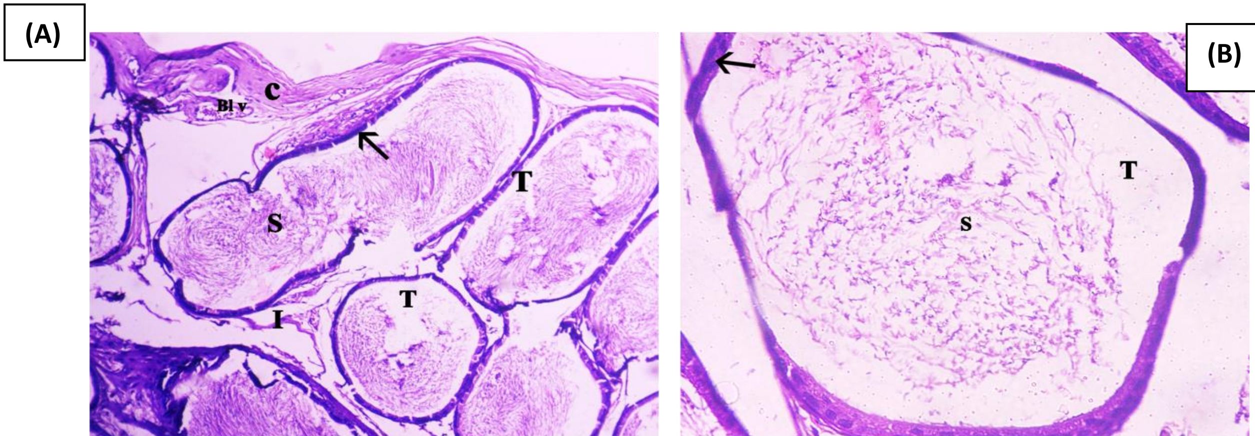

Figure (2 A, B): Photomicrographs of H&E stained sections in epididymis of acetamiprid 1/20 of LD50 treated group showing thick capsule (c), congested blood vessels (Bl v), wide interductal spaces (I), the ductal epithelium have dark nuclei (arrow) and decrease in the number of spermatozoa (S) in the lumen of some tubules (T). (A: H&E×100, B: H&E×400)

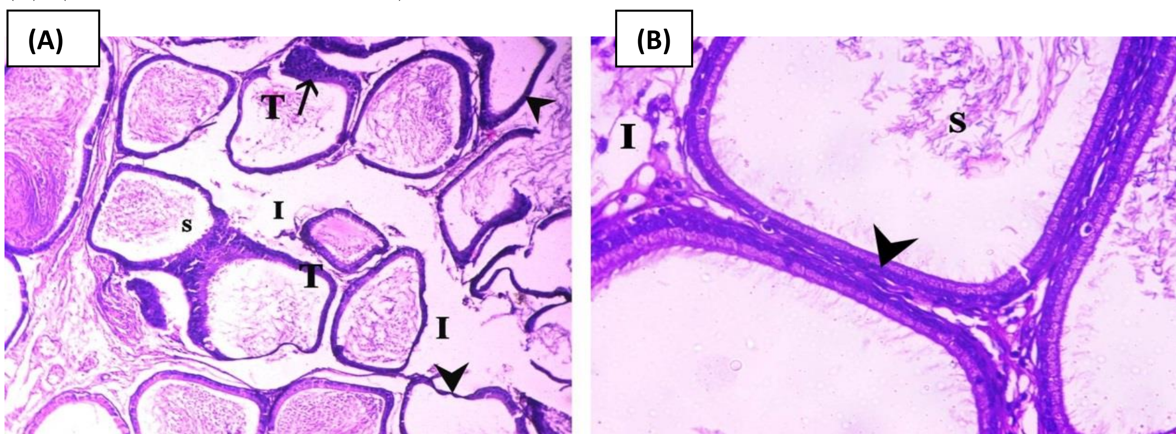

Figure (3 A, B): Photomicrographs of H&E-stained sections in the epididymis of acetamiprid 1/5 of LD50 treated group showing wide interductal spaces (I), the ductal epithelium has small dark nuclei (arrow head) and decrease in the number of spermatozoa (S) in some ducts (T) and others have proliferation in their epithelial lining (arrow). (A: H&E×100, B: H&E×400)

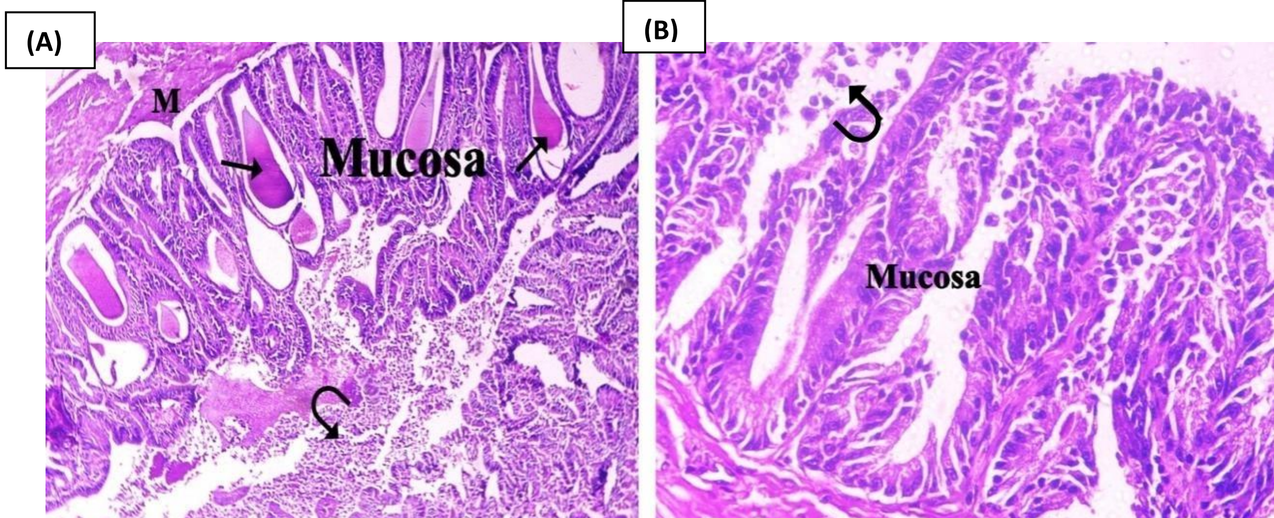

Figure (4 A, B): Photomicrographs of H&E stained sections in seminal vesicles of acetamiprid 1/20 of LD50 treated group showing disorganized mucosa and musculosa (M); decrease in the height of the mucosa, shedding of the epithelium (curved arrow) and acidophilic hyaline material (arrow) in the lumen of the gland. (H&E×100, B: H&E×400)

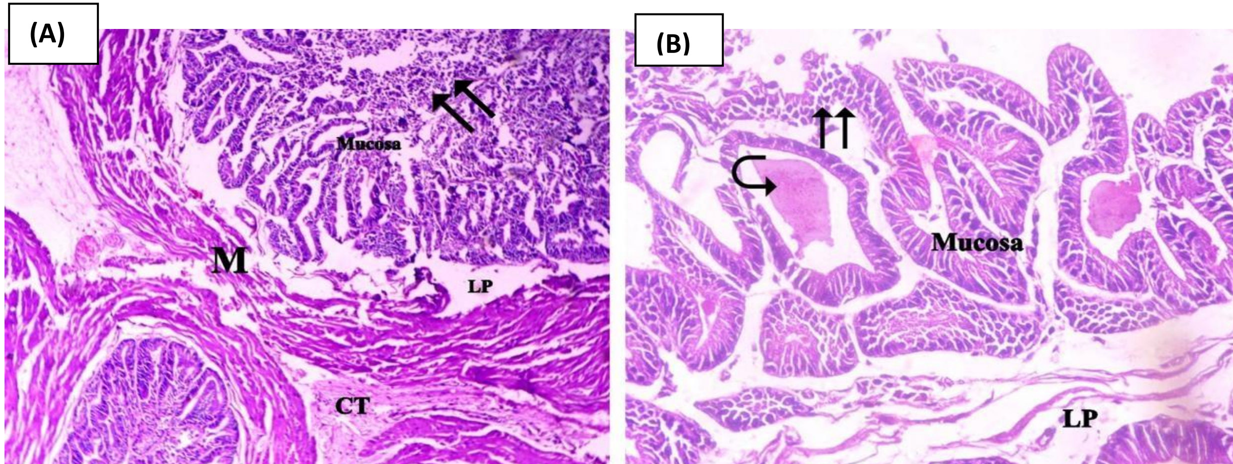

Figure (5 A, B): Photomicrographs of H&E-stained sections in the seminal vesicles of acetamiprid 1/5 of LD50 (high dose) treated group showing marked decrease in the mucosal height, shedding of the epithelial lining of the mucosa (double arrow), acidophilic material in the lumen (curved arrow), wide lamina propria (LP), and disorganized musculosa (M). Notice, thickened adventitia (CT). (A:H&E×100, B: H&E×400).

DISCUSSION

Neonicotinoid insecticides, such as acetamiprid, are used to manage Hemiptera and Aphids that infest fruits, vegetables, and tea plantations. However, due to its long half-lives and high-water solubility, it tends to accumulate in the environment even when used in very little doses. [21-23]. Despite reports that it causes lower levels of toxicity in mammals than other pesticides, several researches showed that neonicotinoids could pose health hazards to mammals, including humans [6].

Studies on the effects of acetamiprid exposure on several organ systems in mammalian models have suggested that the general mechanism underlying the drug's toxicity (hepatotoxicity, neurotoxicity, nephrotoxicity, cytotoxicity, and genotoxicity) may be the creation of oxidative stress [24-26].

One of an organism's most vulnerable functions to toxins from the environment and the workplace, especially pesticides, is reproduction [24]. Thus, the purpose of this study was to examine the toxicity of subacute acetamiprid exposure on epididymis and seminal vesicles in adult male albino rats.

The current study's findings indicated that, although not statistically significant, the treatment groups (acetamiprid 1/5 (40 mg/kg) and 1/20 (10 mg/kg) of LD50) had lower weight mean values for

epididymis and seminal vesicles when compared to the values of the negative control group.

Inconsistent with the results of the present study, Zhang et al. [27] discovered that when Kunming male mice were given 30 mg/kg of acetamiprid orally over the course of 35 days, the drug drastically decreased both the mice's body weight and the weight of their seminal vesicles, or epididymis. Since acetamiprid appeared to enhance oxidative stress markers such p38 MAPK activation and decrease the activity of antioxidant enzymes, this suggests that oxidative stress may be the origin of acetamiprid's harmful effects.

Also, Awasthy [28] claimed that, in comparison to the control group, there was a significant drop in the absolute weights of the seminal vesicles and vas deferens of Wistar rats given acetamiprid for 60 days at doses of 26.25 mg/kg and 52.5 mg/kg.

Similarly, Mosbah et al. [22] observed a reduction in the weight increase of the body and the absolute weights of the reproductive organs (seminal vesicles) in Wistar rats given a 45-day treatment of acetamiprid (27 mg/kg). This suggests that the alterations could be caused by a lack of androgens and a decrease in the production of pituitary FSH and LH. Additionally, they proposed that this decline might be the result of lower testosterone levels, which are important in regulating the size and shape of the genital tract.

The results of the present investigation showed no appreciable difference in mean tissue MDA and TAC levels between the positive and negative control groups.

The acetamiprid-treated groups showed a very significant drop in tissue TAC and a very large increase in tissue MDA when compared to the negative control group. Additionally, there is a highly significant difference between the mean tissue MDA and TAC values in the acetamiprid 1/5 (40mg/kg) and acetamiprid 1/20 (10mg/kg) LD50 groups.

The results of the present study were in agreement with Zhang et al. [27] They found that acetamiprid elevated MDA levels in testicular tissue, suggesting the development of lipid peroxidation and potentially leading to the loss of membrane structure and function. They also found that the activity of the enzymes T-SOD, GSH-Px, and CAT was decreased by acetamiprid. Additionally, Kong et al. [29] found that in Leydig cells, acetamiprid at a dose of 30 mg/kg caused aberrant increases in ROS and mitochondrial damage.

Moreover, Mosbah et al. [22] showed that 45 days at a dose of 27 mg/kg of acetamiprid resulted in an increased formation of free radicals (ROS) and a decrease in the activity of antioxidant enzymes such as SOD, CAT, and GSH-PX, indicating that the reproductive toxicity of acetamiprid insecticide may be mediated by free radical-induced oxidative cell damage.

Besides, Anand et al. [30] revealed that the oxidative stress and overexposure of cellular antioxidant capacity were caused by ROS produced by the metabolism of acetamiprid 52.5 mg/kg for 28 days in rats. This inhibition of antioxidant enzyme expression occurred as a result.

Similarly, Arican et al. [25] demonstrated that, in testis homogenates, acetamiprid caused dose-dependent lipid peroxidation, GSH (glutathione) depletion, an increase in TOS (total oxidant status), and a decrease in TAS (total antioxidant status). According to their findings, acetamiprid causes oxidative stress in the liver, kidney, and brain, among other organs. This oxidative stress is linked to testicular malfunction, which ultimately results in infertility. Reactive oxygen species (ROS) are produced in the testis and cause oxidative damage in addition to destroying spermatogenesis and sterodogenesis.

Elevated levels of malondialdehyde (MDA), one of the primary oxidation products of peroxidized polyunsaturated fatty acids, are a prominent indicator of lipid peroxidation [31]. Pan et al. [32] claimed that MDA is a commonly used indicator of the presence of oxidative stress and is a sensitive biomarker of lipid peroxidation. Total antioxidant content (TAC) is the total impact of all antioxidants, enzymatic and non-enzymatic, that are present in plasma, body fluids, and tissues. Therefore, compared to biological information obtained by the measurement of individual components, it provides more pertinent biological information. [15].

A slice of the epididymis from the 1/20 (10 mg/kg) LD50 acetamiprid group stained with hematoxylin and eosin showed a thick capsule, clogged blood vessels, broad interductal gaps, dark nuclei in the ductal epithelium, and a decrease in spermatozoa in some ducts.

Rat epididymis from the 1/5 LD50 (40 mg/kg) acetamiprid treated group showed broad interductal gaps, ductal epithelium with tiny dark nuclei, and a

decrease in spermatozoa in some ducts with growth in the epithelial lining in other ducts.

The results of the present study were in agreement with Awasthy [28] revealed that male rats given acetamiprid (52.5 mg/kg) because the ductules of their epididymis were bordered by cuboidal epithelium, which lacked microvilli and had less spermatozoa in the lumen. Spermatozoa cells settled in the middle of the lumen and congregated there, but in certain areas the ductular lumen was almost empty or completely devoid of them. Male rats given acetamiprid (26.25 mg/kg) exhibited rather minor alterations in their lining epithelium. Spermatozoa were present in the lumen of the cuboidal epithelium-lined epididymal duct, albeit their concentration was lower than in the control.

On the other hand, Zhang et al. [27] revealed that there were essentially no sperm in the epididymis lumens in the group receiving acetamiprid treatment. The number of spermatids within the epididymis lumen dropped, but the structure of the epididymis remained unaffected by acetamiprid, they discovered. They proposed that acetamiprid, rather than the epididymis, affected the testis, which in turn decreased the reproductive capability of male mice.

Rat seminal vesicles from the 1/20 (10 mg/kg) of the LD50 acetamiprid treated group had disordered mucosa and musculosa, as well as a decrease in mucosal height, epithelium shedding, and acidophilic hyaline material in the lumen, according to sections stained with hematoxylin and eosin.

Rat seminal vesicles from the 1/5 (40 mg/kg) of the LD50 acetamiprid treated group sections stained with hematoxylin and eosin in the acetamiprid-treated group demonstrated a significant reduction in mucosal height. a broad lamina propria, and in some cases, disordered musculosa. Thicker adventitia and mucosal epithelium shedding were seen in other sections.

The results of the present study were in agreement with Gaber et al. [33] They found that rats given dosages (94 and 281 mg/kg) of dinotefuran (a third-generation neonicotinoid) had seminal vesicles with congested blood arteries between the tubules and loss of epithelium.

Conclusion:

Sub-acute acetamiprid exposure causes significant biochemical and histopathological damage to epididymis and seminal vesicles of adult albino rats.

Financial Disclosures

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

Conflict of interest

The authors declare that they have no conflicts of interest rshipwith respect to autho or publicationof this article.

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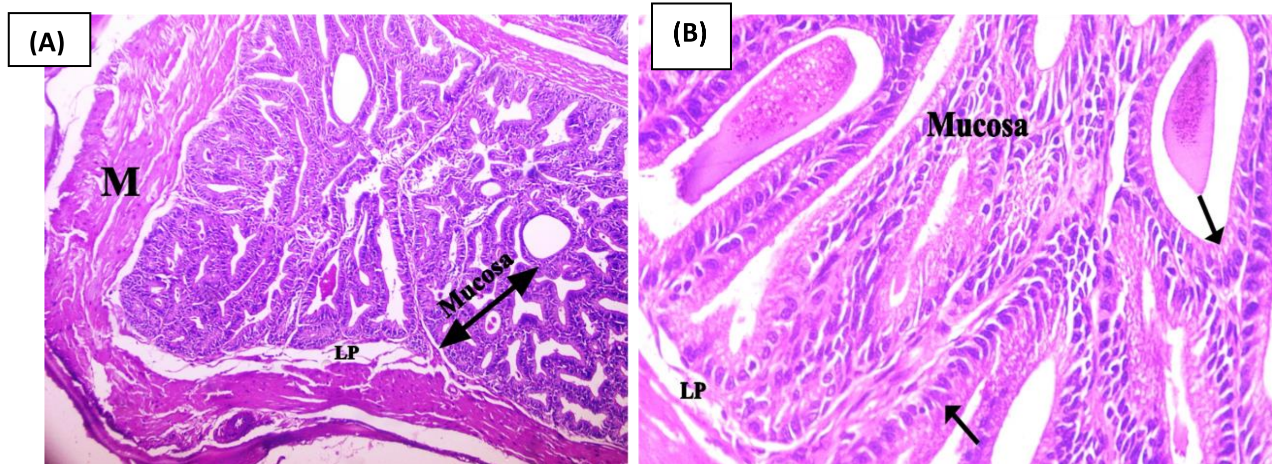

Figsure (1S A, B): Photomicrographs of H&E-stained sections of adult male albino rat control seminal vesicles demonstrating the musculosa (M), underlying layer of loose connective tissue (LP), and heavily folded mucosa (double headed arrow). The pseudostratified columnar epithelium with tall columnar nuclei (arrow) lines the mucosa. (A:H&E×100, B: H&E×400)

Citation

Mesallam, D., Abass, M., Ahmed, S., Ghoneim, A., Morsi, A. Sub-Acute Toxic Effects of Acetamiprid on Epididymis and Seminal Vesicles of Adult Albino Rats. *Zagazig University Medical Journal*, 2024; (197-207): -. doi: 10.21608/zumj.2024.322700.3589