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Protective Role of Thyme Oil (Thymus vulgaris) Against Cadmium Chloride-Induced Renal Damage in Common Carp (Cyprinus carpio)

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ABSTRACT

The present study aimed to evaluate the role of thyme oil in reducing the histopathological effects in the kidneys of common carp (Cyprinus carpio) exposed to two different concentrations of cadmium chloride (16.86 and 56.2 mg/L). The experiment included 6 groups, each containing 16 fish: G1, G2, G3, G4, G5, G6. The results showed the presence of histopathological changes in the kidneys of fish. They were more severe in the two groups treated with cadmium chloride only. They decreased in the two groups treated with cadmium chloride and thyme oil together. The study also showed that the longer exposure period increased the severity of the changes in the two groups treated with cadmium chloride only, especially after 30 days from the beginning of the experiment. Histological changes included dilatation of renal tubules, expansion of Bowman's capsule space, necrosis and degeneration of endothelial cells and renal tubules, increased mast cells and melanomacrophage aggregates. In contrast, histological changes were less severe in the two groups treated with cadmium chloride and thyme oil. This indicates the protective role of thyme oil in reducing the severity of histopathological changes.

1. Introduction

Fish represent a vital renewable natural resource, contingent upon the availability of optimal environmental conditions. They are major food source for humans because they contain essential proteins and fats [1]. Common carp (C. carpio) is among the most important species from an economic and health perspective. They are abundant in Iraqi inland water bodies, especially in the southern and central governorates [2].Heavy metal pollution is one of the most serious environmental problems that negatively affects living organisms. Cadmium is one of these elements that have high toxicity even at low concentrations [3],[4]. The accumulation of cadmium leads to many health problems, including negative effects on the immune system and the induction of oxidative stress [5].

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Fish possess antioxidant defense mechanisms, such as glutathione, which enable them to withstand adverse environmental conditions. these mechanisms may need additional reinforcement, especially in fish farms with limited environments. Therefore, fish farmers seek to provide their feed with nutrients and elements that boost the immune system. Among these elements are natural antioxidants such as thyme, which belongs to the Lamiaceae family [6].

Thyme is characterized by its antiseptic and antioxidant properties. It is rich in vitamins and minerals that promote health and protect against diseases [7]. Thymol and carvacrol are the most important components of thyme oil. They are attributed to biological and pharmacological activity [8],[9] explained the use of many antioxidants, for their beneficial effect against oxidative stress, such as vitamin E, flavonoids and polyphenols. Many studies have shown the beneficial effects of medicinal plant extracts, including thyme. It has many compounds that have great potential for use in many cancer treatments due to their pharmacological properties and the elimination of cancer cells through different molecular mechanisms, including autophagy, cell necrosis and programmed cell death [10],[11],[12].
This study aims to evaluate the effectiveness of thyme oil in reducing the histopathological changes in the kidneys of carp fish resulting from exposure to cadmium chloride.

2. Materials and Methods

2.1 Experimental fish

The experiment was conducted over 30 days, during which the median lethal concentration (LC50) was determined [13]. The fish were divided into six groups, each consisting of 16 individuals, to study the effects of different concentrations, as follows:

1. **Control groupG1:** Fish raised in chlorine-free water and fed on commercial food with protein content.
2. **Thyme oil groupG2:** Fish fed on food containing thyme oil at a rate of (1 ml per 3 g food). Thyme oil is provided by the Indian company SHEER essence at a concentration of 100%.
3. **Low cadmium chloride groupG3:** Fish were treated with 16.68 mg/L of cadmium chloride added to the water, which was provided by the Indian company Thomas Baker.
4. **High cadmium chloride groupG4:** Fish treated with 56.2 mg/L of cadmium chloride added to the water.
5. **Low cadmium chloride and thyme oil groupG5:** Fish were exposed to cadmium chloride at a concentration of 16.86 mg/L in water. Thyme oil was incorporated into their diet at a ratio of 1 mL per 3 g of feed. Prior to cadmium chloride exposure, the fish were acclimated to the thyme oil-enriched diet for a period of 21 days.
6. **High cadmium chloride and thyme oil groupG6:** Fish were treated with 56.2 mg/L of cadmium chloride added to water. Thyme oil was added to the food at a rate of (1 ml of thyme oil per 3 g of feed). Fish were acclimated for 21 days to the food containing thyme oil before being exposed to cadmium chloride. They were fed daily on a diet containing 37% protein, 8.84% fat, and 42.64% carbohydrates at 5% of fish weight. They were dissected at (7, 15, and 30) days after treatment for the purpose of studying cadmium concentration and histopathological changes.

2.2 Study of histopathological changes

Kidney tissue sections were prepared using the method of [14]. The samples were fixed with Bouin's fixative for 24 hours. Then, they were washed with 50% ethyl alcohol several times. Then, the samples were passed through an ascending series of alcohols (70%, 80%, 90%, and 100%) for 2 h for each concentration. Then, they were decanted with xylene to penetrate the paraffin wax. After that, they were embedded in molten paraffin wax at 56 °C using metal moulds. The moulds were cut using a rotary microtome at a thickness of 5-7µm mounted on glass slides and stained with hematoxylin and eosin. The tissue sections were examined and photographed using a Zeiss microscope equipped with a Leica digital camera.

3. Results

3.1 Histopathological changes in fish kidneys

G1 consisted of renal tubules lined with simple epithelial tissue and a glomerulus composed of a network of blood vessels intermingled with altered epithelial cells and surrounded by a Bowman's capsule (Figures 1 and 2).

3.1.1. After 3 days

The results showed that the renal tubules and glomeruli in G2 were healthy. The presence of non-living materials in the renal tubule cavities accepts the acidic dye to varying degrees. Thus, the renal tissue appears normal, as shown in Figures (3, 4, 5 and 6).
The results showed the presence of various pathological tissue changes in G3. These changes were represented by the expansion of the Bowman's capsule space Figure (7) and a slight expansion in some renal tubules Figure (8 and 9). In contrast, the changes that appeared in G4 were necrosis of the cells lining the renal tubules (10) with degeneration of the renal tubules (11) and degeneration of the hematopoietic tissue (12 and 13) and an increase in mast cells and thickening of the Bowman's space (Figures 14 and 15). As for G5, it was found that there were no Histopathological changes in the renal tubule tissue. The glomeruli were normal and the hematopoietic tissue was intact in Figures 16 and 17. It was noted that in G6, there were fewer histological changes than in the treatment with cadmium alone. The hematopoietic tissue appeared normal between the tubules with clusters of melanocytes and expansion of some renal tubules, as in Figures 18 and 19.

3.1.2. After 7 days

The results of the microscopic examination of sections of kidney tissue in G2 showed the normal arrangement of tubules and glomeruli. The kidney tissue appeared normal, as shown in Figure (20 and 21). As for G3, various pathological changes were observed. These changes were represented by necrosis of some renal tubule cells. The blood-forming tissue was observed normally between the tubules Figure (22) with the accumulation of non-living materials Figure (23 and 24), and degeneration of the renal tubules Figure (25) with hypertrophy of the lining cells and expansion of the Bowman's capsule space as in Figure (26 and 27). In G4, the severity of the pathological changes continued to increase. These changes were represented by necrosis of large areas in the renal tissue. The disintegration of the epithelial tissue lining the renal tubules and the blood-forming tissue appeared as shown in Figures (28 and 29). A decrease in Histopathological changes was observed after adding thyme to G5. This was represented by expansion and necrosis in some cells lining the renal tubules, Figure (30 and 31) and expansion in the Bowman's capsule space. As for the blood-forming tissue, it was normal, Figure (32). However, edema appeared in the blood components, as shown in Figure (33).
In contrast, the integrity of the renal tubules was observed in other areas, Figure (34). The role of thyme oil in reducing the severity of pathological changes was observed after adding it to G6. The integrity of the renal tissue, blood-forming tissue, and glomerulus was represented in Figure (35), with the presence of melanomacrophage cells, as shown in Figure (36, 37, 38, and 39). In some areas, degeneration was observed in some cells lining the renal tubules, Figure (40).

3.1.3. After 15 days

7. The results of the microscopic examination of G2 showed the safety of the renal tissue (Figures 41 and 42). In contrast, the results showed the continuation of the occurrence of Histopathological changes and their increased severity in G3. The disintegration of blood-forming tissue cells represented this, an increase in the acidic pigment of some renal tubules and atrophy of their nuclei in the form of dark-pigmented balls as in Figure (43) as well as the occurrence of bleeding Figure (44). Oedema was observed between the renal tubules Figure (45) with the expansion of the Bowman's capsule space and hyperpigmentation with eosin of the cells of some renal tubules, as shown in Figure (46). As for G4, the effects were observed to be more

severe due to cadmium toxicity. These changes were represented by necrosis of the renal tubules and blood-forming tissue, with atrophy of the glomeruli, expansion of the Bowman's capsule space, and the occurrence of haemorrhage (Figure 47, 48, 49).

8. The results showed that G5 a decrease in the severity of the changes observed in the group treated with cadmium alone. The nature of the renal structure represented this with some slight changes in the expansion of Bowman's capsule (Figures 50 and 51) with the presence of melanomacrophag cells trapped inside the cells in the blood-forming tissue in a single form (Figure 52) or the form of clusters as in Figure (53). As for G6, a decrease in the severity of histopathological changes was observed. The changes were less than those observed in the treatment with cadmium alone. Necrosis was seen in the lining of some renal tubules (Figure 54). It was also noted that the capillary blood vessels in the glomeruli expanded in Figure (55) with the accumulation of non-living materials in the form of irregular masses in the blood-forming tissue and the tissue lining the tubules and metaplasia of the cells lining that cavity Figure (56 and 57).

3.1.4. After 30 days

The results of G2 showed that the renal tissue appeared normal with the presence of some melanomacrophage cells, as shown in Figure (58). The study indicated that the histopathological changes that occurred in G3 were more severe than in previous periods. It showed necrosis of most of the renal tubules and necrosis of the blood-forming tissue between the renal tubules. It appears as remnants of damaged tissue. Cell decomposition may be observed. They appear to have decomposed plasma membranes, and most of the cells lose their nuclei. The histopathological changes in the kidney also included glomerular atrophy and expansion of Bowman's capsule space, in addition to the expansion of the capillaries in the glomerulus, as shown in Figures (59 and 60). As for G4, the severity of the changes increased more than the previous treatment. In addition to the changes recorded in it, such as degeneration, necrosis and expansion of blood vessels, bleeding between the renal tubules and degeneration and necrosis of the tubule lining were observed. Glomerular degeneration was also observed with the expansion of the capillary blood vessels in the glomerulus, as in Figures (61, 62 and 63). The results of the microscopic examination in G5 showed a clear decrease in Histopathological changes. The structure of the renal tissue appeared normal, with some minor effects of cells in the lining of the renal tubules, which suffer from changes of varying degrees ranging from necrosis to degeneration, as shown in Figure (64). The severity of Histopathological changes decreased in G6 compared to treatment with cadmium alone. Some changes were observed in the renal tissue, such as expansion of the lumen of some renal tubules and necrosis of some of them. Expansion of Bowman's capsule space was noted with the presence of melanomacrophage clusters, as shown in Figures (65 and 66).

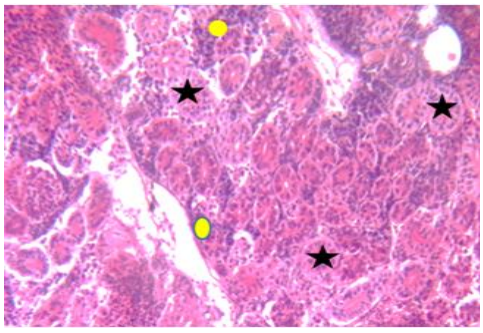

Fig. 1. Section cross of kidney tissue of G1. The components of the kidney tissue and renal tubules (★), and the blood-forming tissue (◎) were observed, H&E, 10 X.

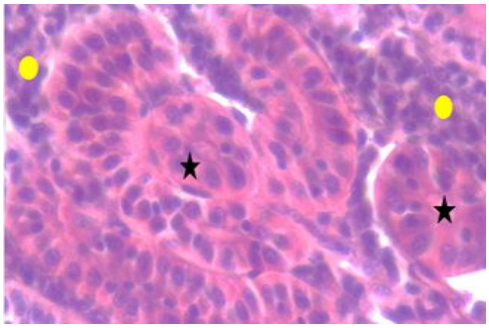

Fig. 2. Section cross of kidney tissue of G1. The figure shows the renal tubules (★) and the blood-forming tissue (◎), H&E, 40 X.

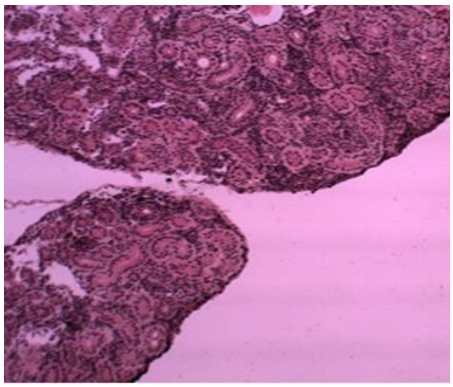

Fig. 3. Section cross of kidney tissue of G2 after 3 days of treatment. The figure shows the components of normal kidney tissue, H&E, X 10.

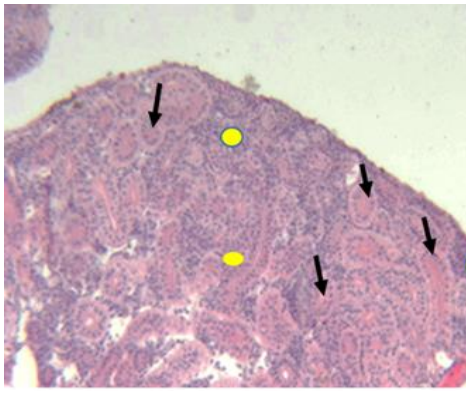

Fig. 4. Section cross of kidney tissue of G2 after 3 days of treatment. The figure shows the components of the kidney tissue: renal tubules (➔), and blood-forming tissue (◉), H&E, X 10.

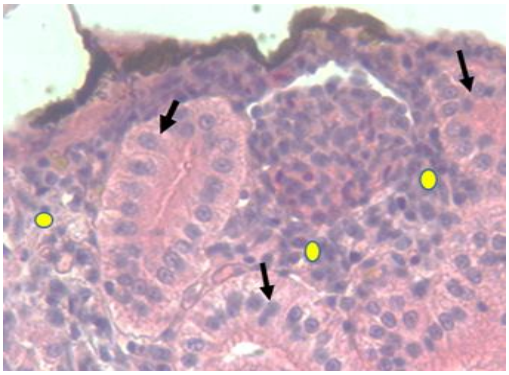

Fig. 5. Section cross of kidney tissue of G2 after 3 days of treatment. The figure shows the integrity of the renal tubules (➔) and the blood-forming tissue (◉), H&E, 40 X.

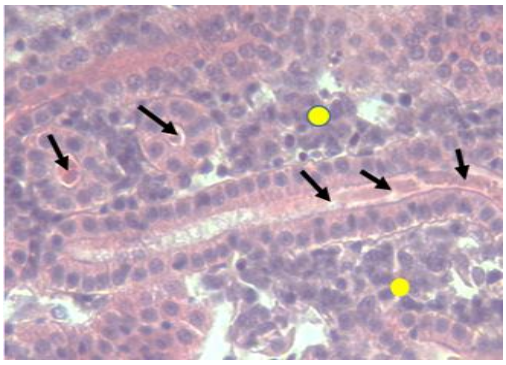

Fig. 6. Section cross of kidney tissue of G2 after 3 days of treatment. The figure shows the presence of non-living materials in the cavities of some renal tubules (➔). The figure shows the integrity of the blood-forming tissue (◉). H&E. 40 X.

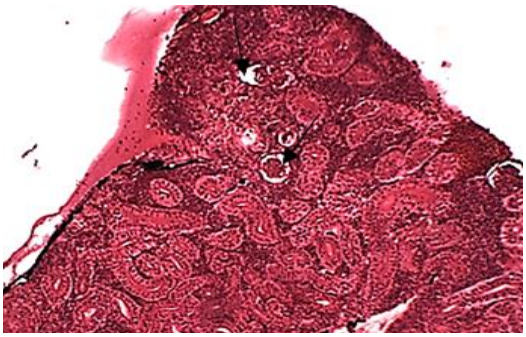

Fig. 7. Section cross of kidney tissue of G3 after 3 days of treatment. The figure shows the expansion of the Bowman's capsule space (➔), H&E, 10 X.

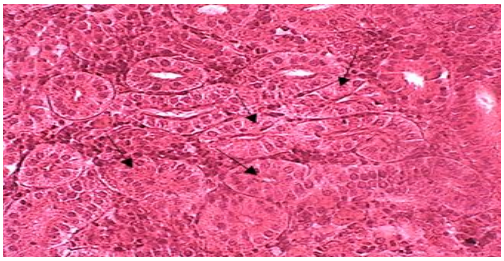

Fig. 8. Section cross of kidney tissue of G3 after 3 days of treatment. The figure shows the integrity of the renal tubules (➔), H&E, 40 X.

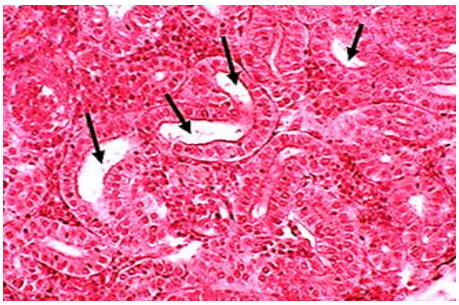

Fig. 9. Section cross of kidney tissue of G3 after 3 days of treatment. The figure shows the expansion of the lumen of the renal tubules (➔), H&E, 40 X.

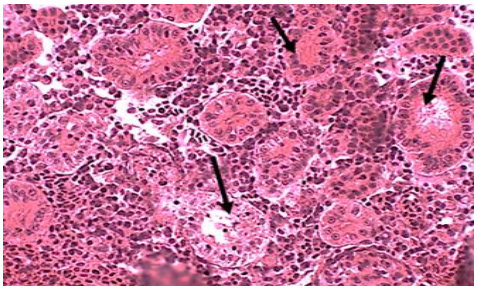

Fig. 10. Section cross of kidney tissue of G4 after 3 days of treatment. The figure shows necrosis of the cells lining the renal tubules (➔), H&E, 40 X.

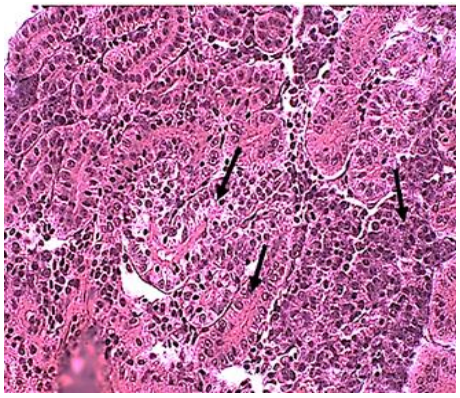

Fig. 11. Section cross of kidney tissue of G4 after 3 days of treatment. The figure shows degeneration of the renal tubules (➔), H&E. 40 X.

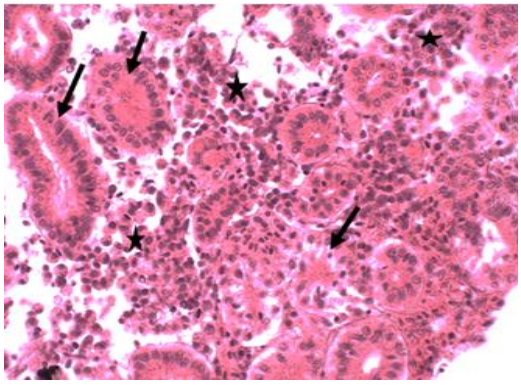

Fig. 12. Section cross of kidney tissue of G4 after 3 days of treatment. The figure shows degeneration of the cells lining the renal tubules (➔), disintegration of the blood-forming tissue, necrosis in most cells★ , H&E, 40 X.

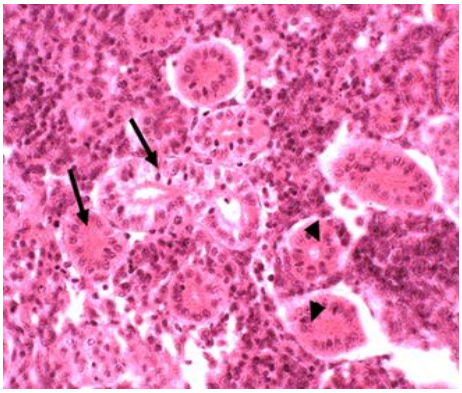

Fig. 13. Section cross of kidney tissue of G4 after 3 days of treatment. The figure shows degeneration of renal tubules (➔), necrosis of the tubule lining (➔), H&E, 40 X.

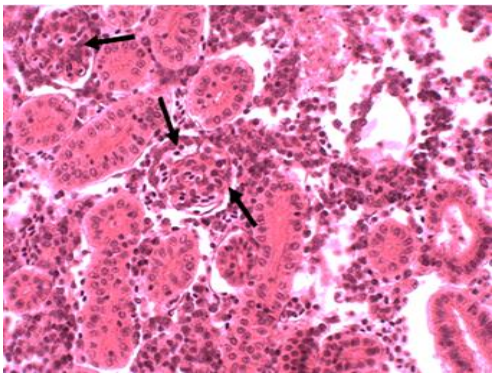

Fig. 14. Section cross of kidney tissue of G4 after 3 days of treatment. The figure shows thickening in Bowman's space (➔) H&E, 40 X.

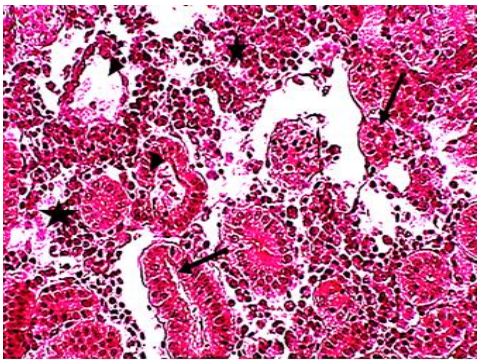

Fig. 15. Section cross of kidney tissue of G4 after 3 days of treatment. The figure shows dilatation of renal tubules (➔), necrosis of most tubules (arrowhead), disintegration of blood-forming tissue (star), E & H, 40 X.

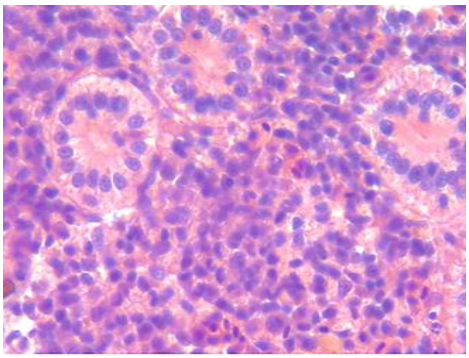

Fig.17. Section cross of kidney tissue of G5 after 3 days of treatment. The figure shows the integrity of the kidney tissue, H&E, 40 X.

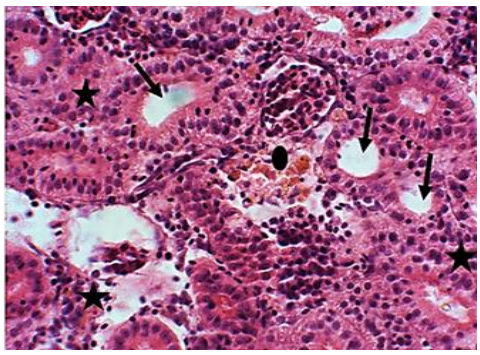

Fig. 19. Section cross of kidney tissue of G6 after 3 days of treatment, expansion of renal tubules (➔), normal blood-forming tissue (★, aggregation of melanomacrophage cells (◉),H&E, 40 X.

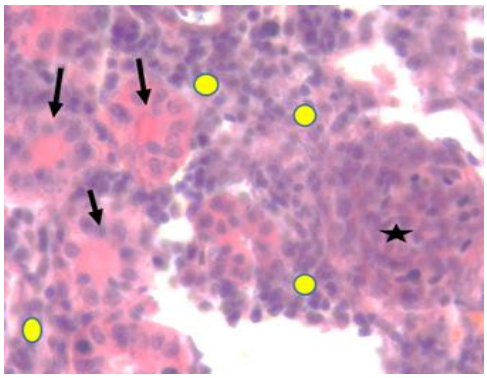

Fig. 16. Section cross of kidney tissue of G5 after 3 days of treatment. The figure shows the integrity of the renal tubules (➔), glomeruli (★) and blood-forming tissue (◉), E & H, 40 X.

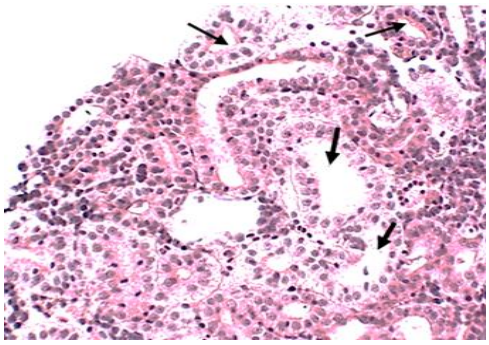

Fig. 18. Section cross of kidney tissue of G6 after 3 days of treatment. Expansion and degeneration of renal tubules (➔), aggregation of melanomacrophage cells (star), metaplasia in the tubule lining (➢), H&E, 40 X.

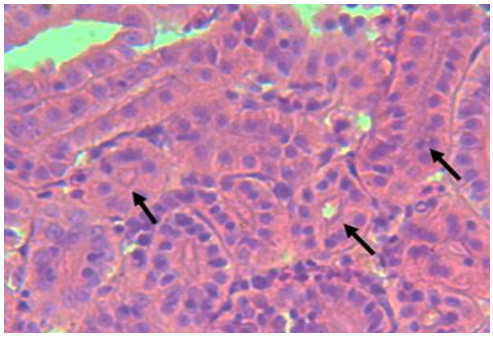

Fig. 20. Section cross of kidney tissue of G2 after 7 days of treatment. The figure shows the integrity of the renal tubules and (➔), H&E, 40 X.

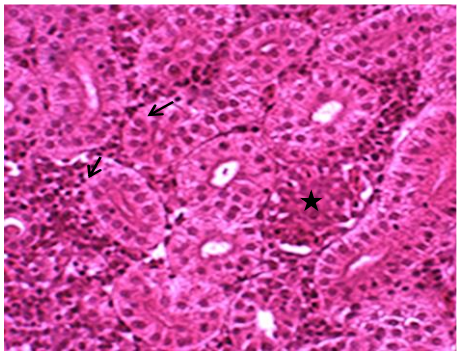

Fig. 21. Section cross of kidney tissue of G2 after 7 days of treatment. It shows the integrity of the renal tubules and (➔), the presence of the glomerulus in a normal form(★) H&E, 40 X.

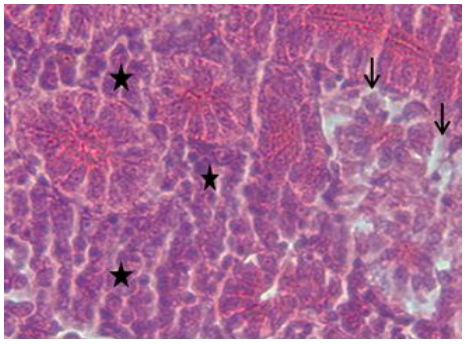

Fig. 22. Section cross of kidney tissue of G3 after 7 days of treatment. Necrosis of renal tubules (➔), integrity of blood-forming tissue (★),H&E, 40 X.

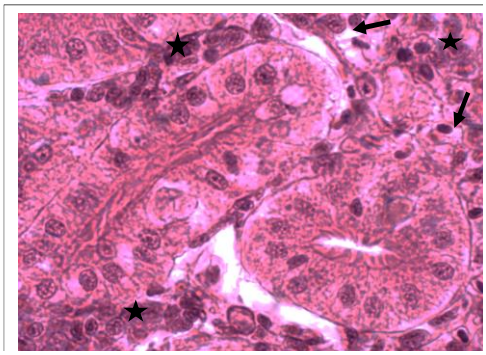

Fig. 23. Section cross of kidney tissue of G3 after 7 days of treatment. The figure shows necrosis of renal tubules (➔), accumulation of non-living materials (★), H&E, 100 X.

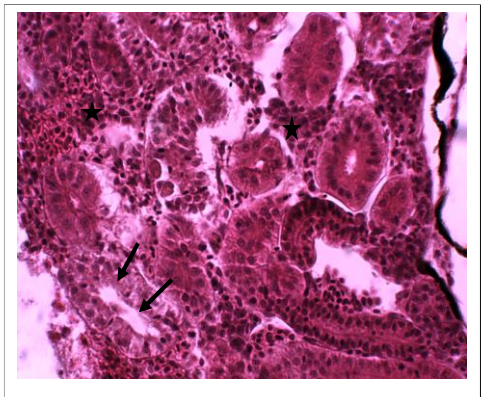

Fig. 24. Section cross of kidney tissue of G3 after 7 days of treatment. The figure shows necrosis of renal tubules (➔), accumulation of non-living materials (★), H&E, 100 X.

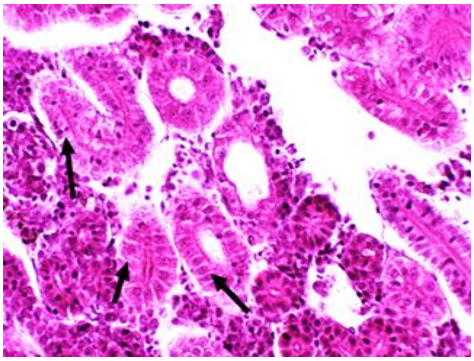

Fig. 25. Section cross of kidney tissue of G3 after 7 days of treatment. The figure shows the degeneration of renal tubules (➔), H&E, 100 X.

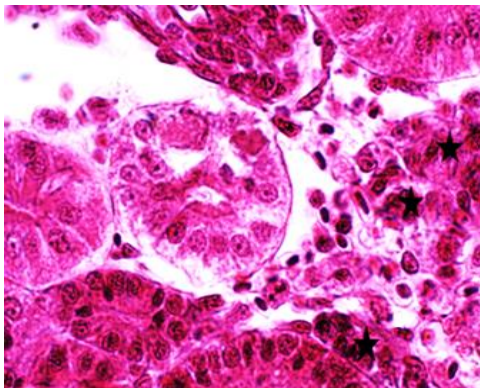

Fig. 26. Section cross of kidney tissue of G3 after 7 days of treatment. The figure shows the accumulation of non-living materials (★), H&E, 100 X.

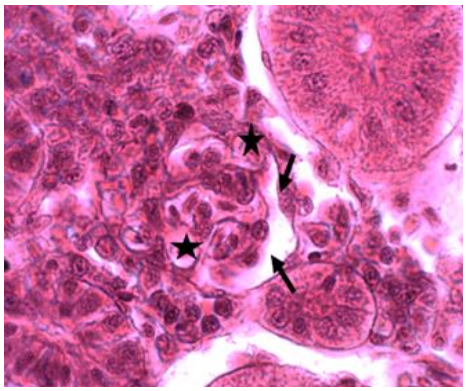

Fig. 27. Section cross of kidney tissue of G3 after 7 days of treatment. The figure shows the expansion of Bowman's capsule (➔) and the hypertrophy of the cells lining Bowman's capsule (★),H&E, 100 X.

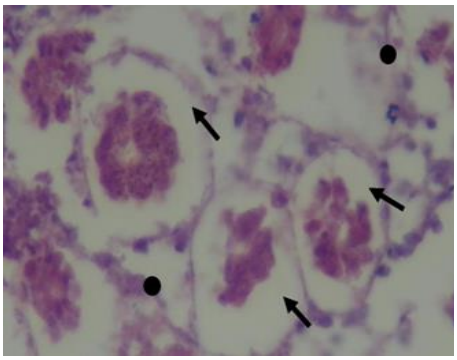

Fig. 29. Section of the kidney tissue of G4 after 7 days of treatment. The figure shows large areas of the renal tissue. It shows the disintegration of the epithelial tissue lining the renal tubules (➔), and the dissolution of the hematopoietic tissue (◉) H&E, 40 X.

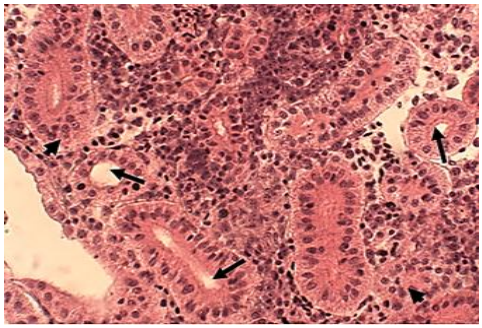

Fig. 31. Section of the kidney tissue of G5 after 7 days of treatment. Expansion of some cells lining the renal tubules (➔), necrosis of some tubules (➢), H&E, 40 X.

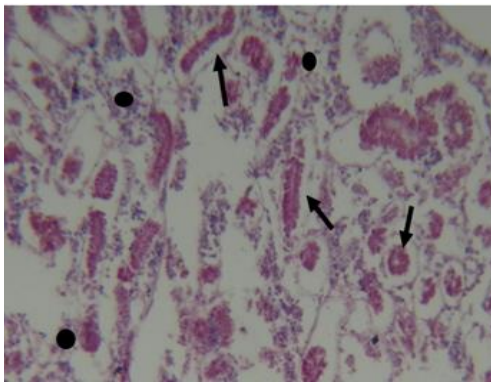

Fig. 28. Section cross of kidney tissue of G4 after 7 days of treatment. Necrosis of the lining tissue of the renal tubules (➔), and dissolution of the hematopoietic tissue (◉) H&E, 10 X.

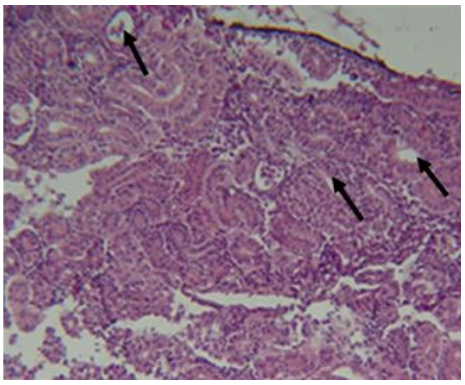

Fig. 30. Section cross of the kidney tissue of G5 after 7 days of treatment. The figure shows the expansion of the renal tubules (➔), H&E, 10 X.

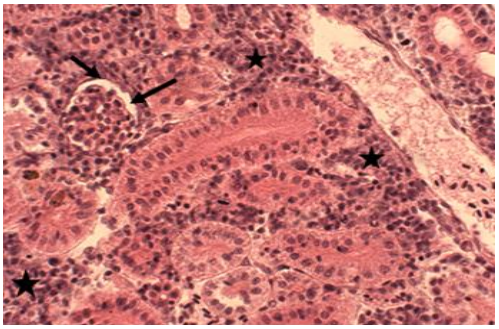

Fig. 32. Section of kidney tissue of G5 after 7 days of treatment, expansion of Bowman's capsule (➔), normal blood-forming tissue , 40 X.H&E), ★(

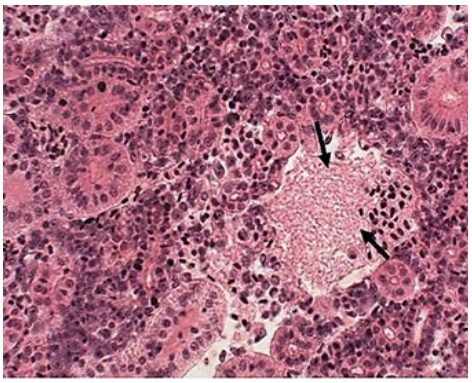

Fig. 33. Section cross of kidney tissue of G5 after 7 days of treatment, edema in the components of the renal tissue (➛), H&E, 40 X.

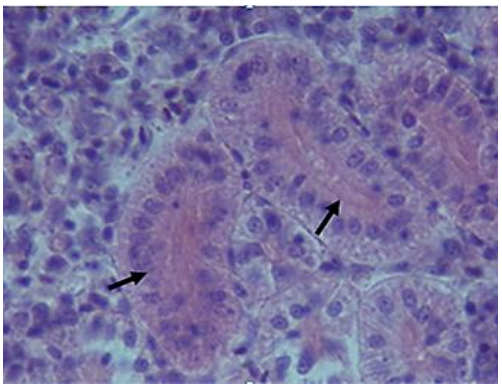

Fig. 34. Section of kidney tissue of G5 after 7 days of treatment, integrity of renal tubules (➛), H&E, 40 X.

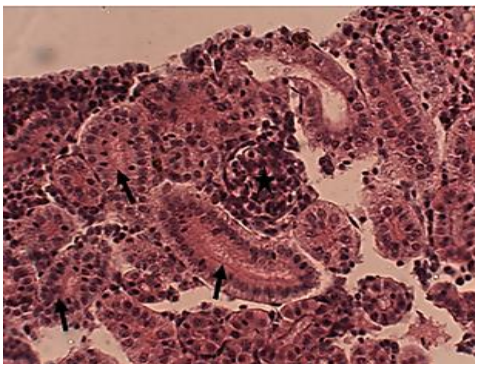

Fig. 35. Section cross of kidney tissue of
G6 after 7 days of treatment. Integrity of renal tubules (➛), normal glomerulus (★), H&E, 40 X.

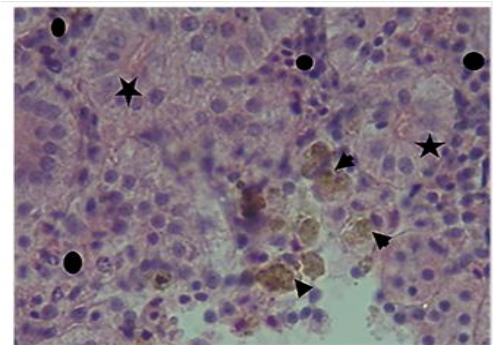

Fig. 36. Section cross of kidney tissue of G6 after 7 days of treatment. Integrity of tissues in terms of structure (★). Hematopoietic tissue integrity (◉), melanomacrophage cluster (arrowhead), H&E, 40 X.

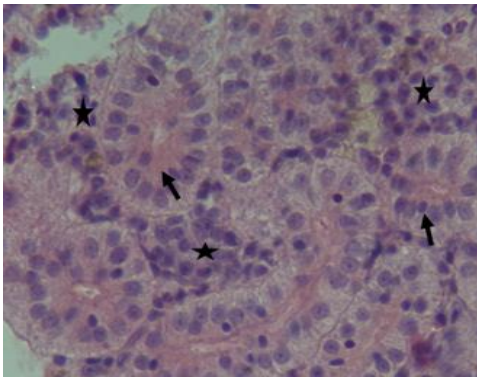

Fig. 37. Section cross of kidney tissue of G6 after 7 days of treatment, normal structure of renal tubules (➛). Normal hematopoietic tissue (★), normal renal tissue, H&E, 40 X.

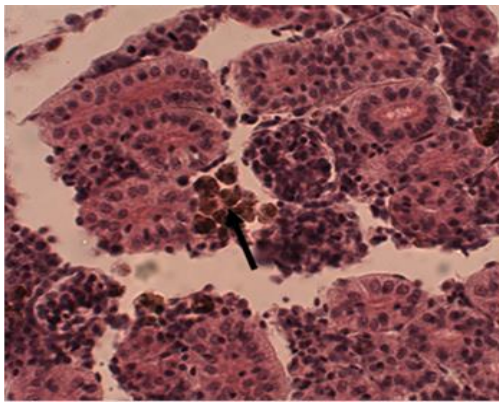

Fig. 38. Section cross of kidney tissue of G6 after 7 days of treatment. Normal renal tissue, presence of non-living materials (➛), H&E, 40 X.

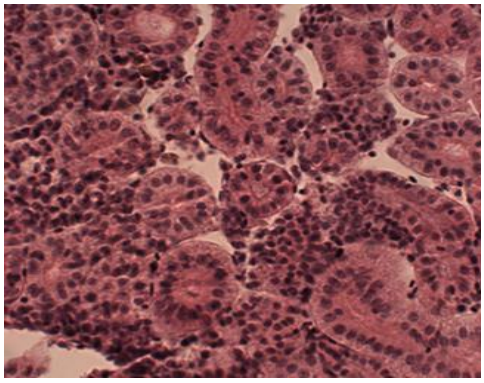

Fig. 39. Section cross of kidney tissue of G6 after 7 days of treatment. Normal renal tissue, H&E, 40 X.

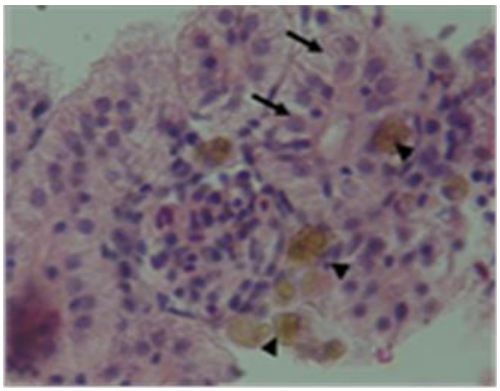

Fig. 40. Section cross in kidney tissue of G6 after 7 days of treatment, degeneration in the cells lining the renal tubules (➔), normal glomerulus (star), melanomacrophage cells (➢), H&E, 40 X.

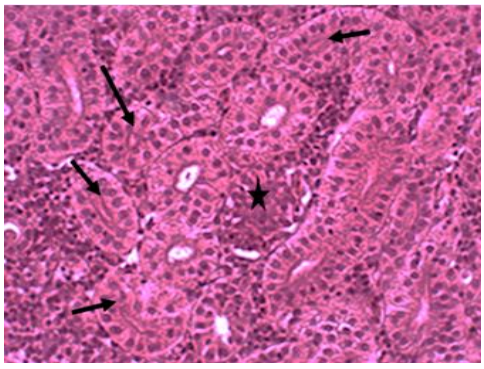

Fig. 41. Section cross in kidney tissue of G2 after 15 days of treatment, intact renal tubules (➔), intact glomerulus (★), H&E, 40 X.

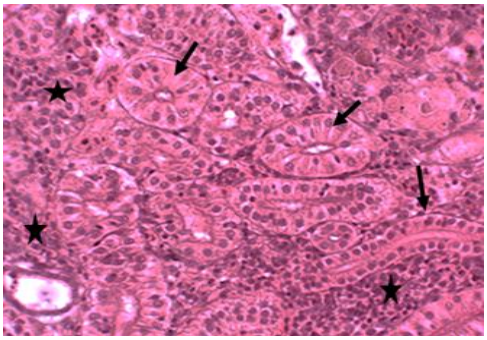

Fig. 42. Section cross in kidney tissue of G2 after 15 days of treatment, intact renal tubules (➔), intact blood-forming tissue (★), H&E, 40 X.

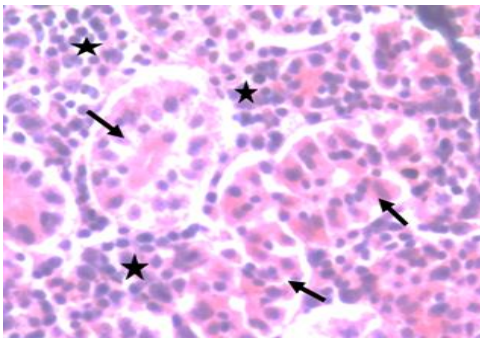

Fig. 43. Section cross in kidney tissue of G3 after 15 days of treatment. Increased acidic pigmentation of some renal tubules and atrophy of their nuclei in the form of vaguely pigmented balls (➔) were observed, disintegration of blood-forming tissue cells (★), H&E, 40 X.

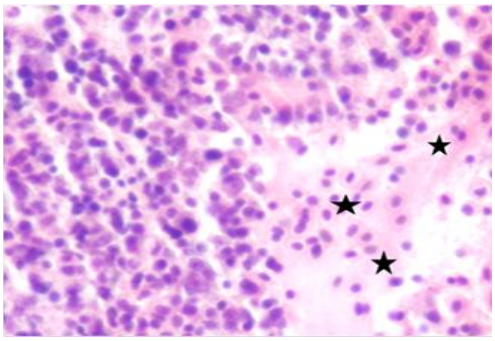

Fig. 44. Section cross of kidney tissue of G3 after 15 days of treatment. Bleeding was observed , 40 H&E), ★(X.

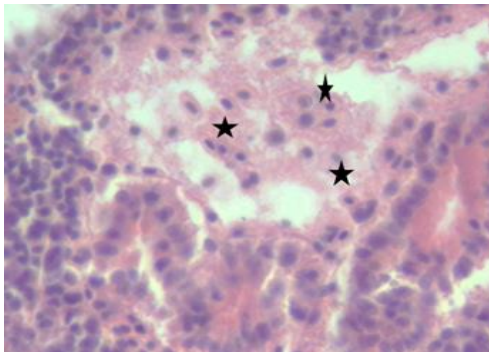

Fig. 45. Section of kidney tissue of G3 after 15 days of treatment. Presence of edema between renal tubules (★), H&E, 40 X.

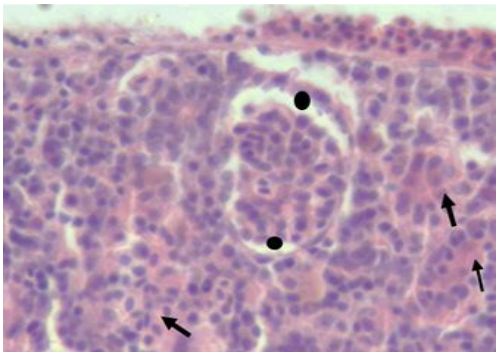

Fig. 46. Section of kidney tissue of G3 after 15 days of treatment. Expansion of Bowman's space (●) and hyperpigmentation with eosin (➔) were observed, H&E, 40 X.

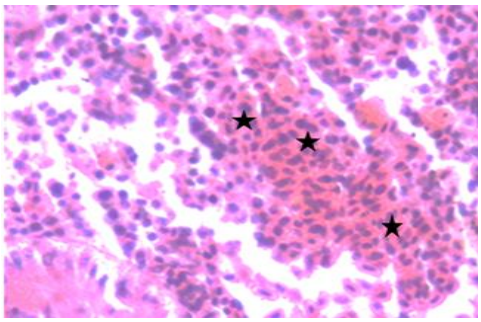

Fig. 47. Section of kidney tissue of G4 after 15 days of treatment. Hemorrhage (★) was observed, H&E, 40 X.

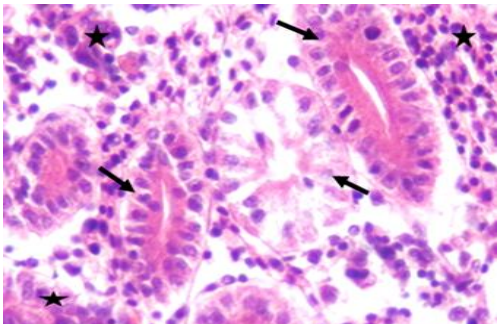

Fig. 48. Section of kidney tissue of G4 after 15 days of treatment. Necrosis of renal tubules (➔), necrosis of blood-forming tissue (★), H&E, 40 X.

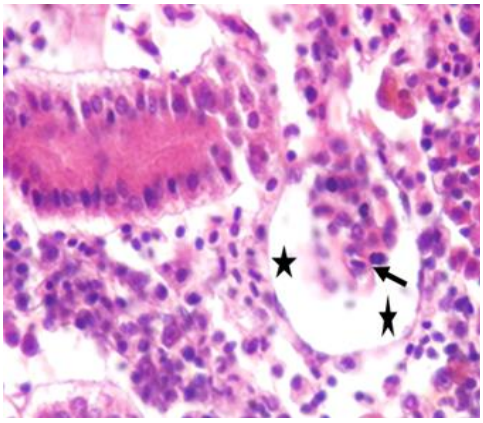

Fig. 49. Section of kidney tissue of G4 after 15 days of treatment. Glomerular atrophy (➔), expansion of Bowman's capsule space (★), H&E, 40 X.

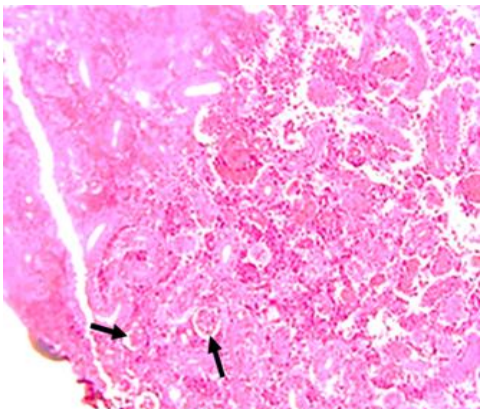

Fig. 50. Section of kidney tissue of G5 after 15 days of treatment. The renal tissue is normal with some minor changes such as dilatation of the Bowman's capsule (➔), H&E, 10 X

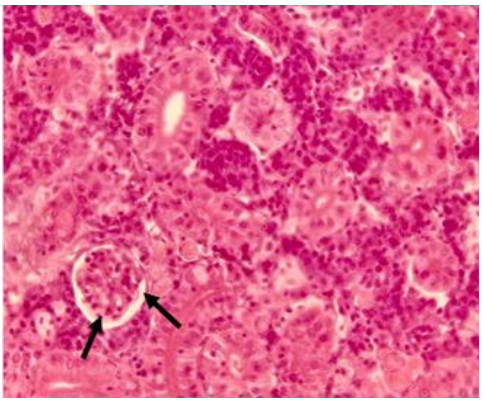

Fig. 51. Section cross of the kidney tissue of G5 after 15 days of treatment. The renal tissue is normal with some minor changes such as dilatation of the Bowman's capsule (➔), H&E, 40 X.

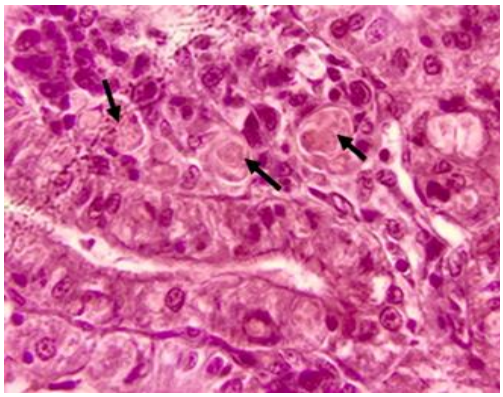

Fig. 52. Section cross of the kidney tissue of G5 after 15 days of treatment. Presence of non-living materials inside the tissue individually (➔), H&E, 100 X.

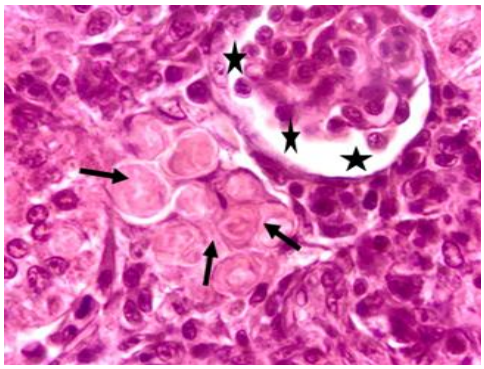

Fig. 53. Section cross in kidney tissue of G5 after 15 days of treatment, aggregation of melanomacrophage cells (➔), expansion of Bowman's capsule space (★), H&E, 100 X.

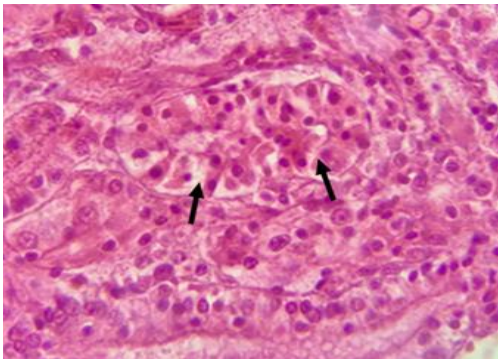

Fig. 54. Section cross in kidney tissue of fish in G6 after 15 days of treatment, necrosis in the lining of a few renal tubules (➔), H&E, 100 X.

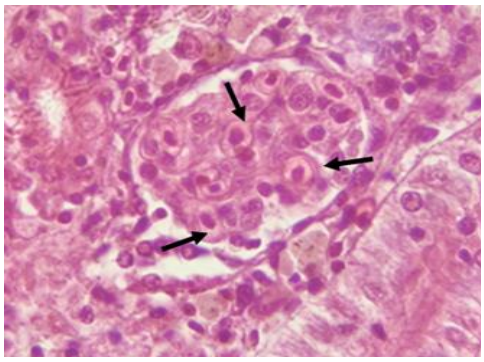

Fig. 55. Section cross in kidney tissue of G6 after 15 days of treatment, expansion of capillary blood vessels (➔), H&E, 100 X.

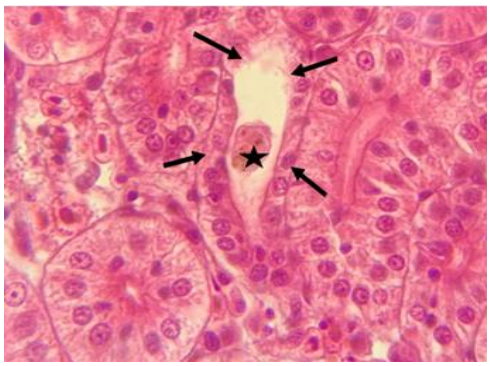

Fig. 56. Section cross in kidney tissue of G6 after 15 days of treatment, endothelial metaplasia of renal tubules (➔), accumulation of non-living materials (★), H&E, 100 X.

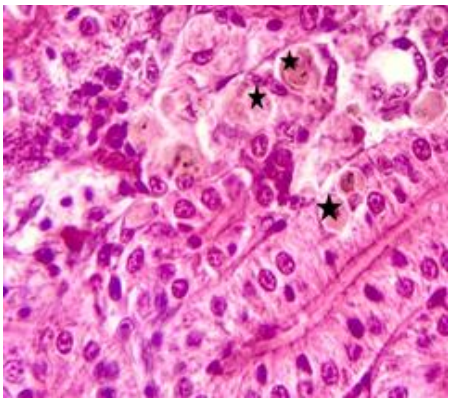

Fig. 57. Section cross in kidney tissue of G6 after 15 days of treatment, accumulation of non-living materials in the tubule lining cells (★), H&E, 100 X.

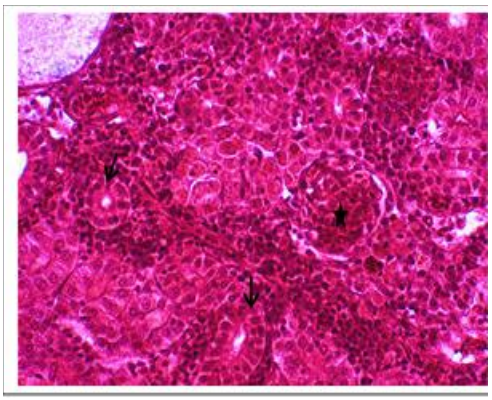

Fig. 58. Section cross in kidney tissue of G2, after 30 days of treatment, renal tissue appears normal, glomerulus (★), integrity of renal tubule (➔), H&E, 40 X.

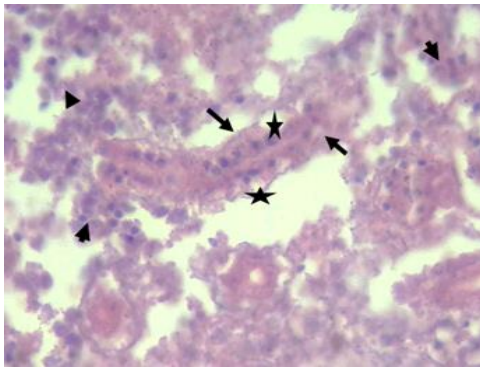

Fig. 59. Section cross in kidney tissue of G3 30 days of treatment, necrosis of most of the renal tubules (➔), cell lysis (★), necrosis of the blood-forming tissue (➢), H&E, 40 X.

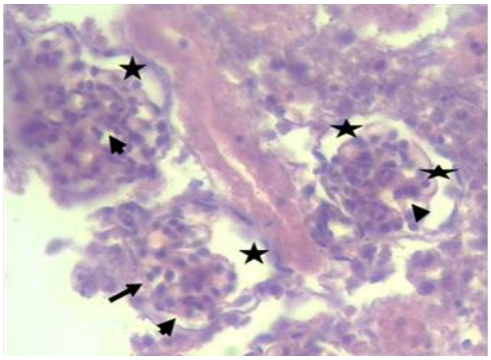

Fig. 60. Section cross of kidney tissue of G3 after 30 days of treatment, glomerular atrophy (➔), expansion of Bowman's capsule (★), expansion of blood vessels in the glomerulus (➢), H&E, 40 X.

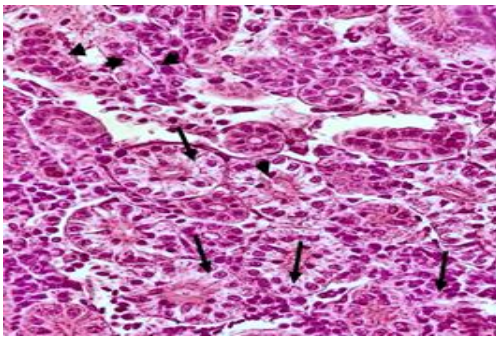

Fig. 61. Section cross of kidney tissue of G4 after 30 days of treatment, renal tubule degeneration (➔), necrosis of the renal tubule lining (➢), H&E, 100 X.

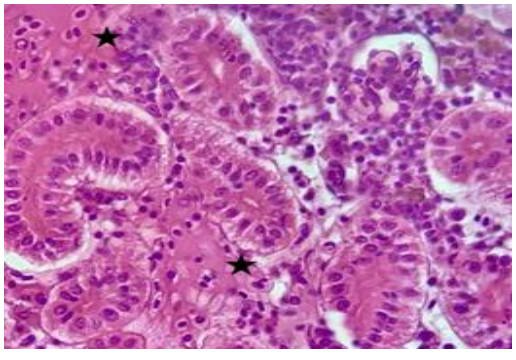

Fig. 62. Section cross of kidney tissue of G3 after 30 days of treatment, hemorrhage (★), H&E, 100 X.

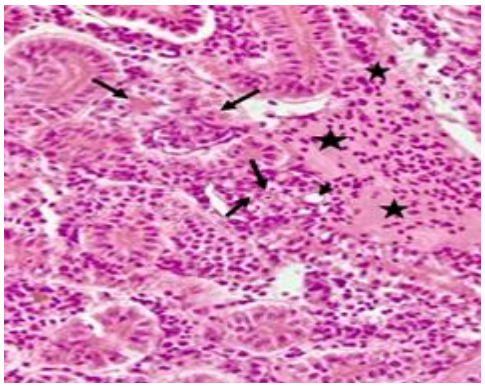

Fig. 63. Section cross in kidney tissue of G4 after 30 days of treatment, degeneration of glomeruli and renal tubules (➔), expansion of blood vessels in the glomerulus (➢), hemorrhage (★), H&E, 100 X.

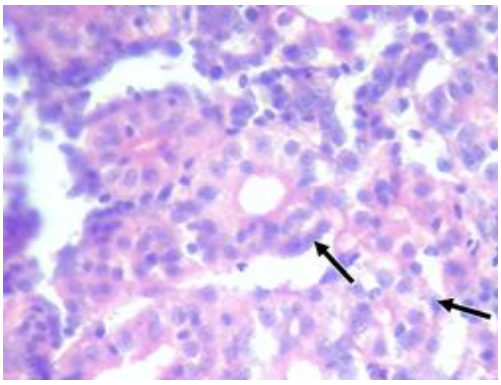

Fig. 64. Section cross in kidney tissue of G5 after 30 days of treatment, necrosis of renal tubules(➔), H&E, 40 X.

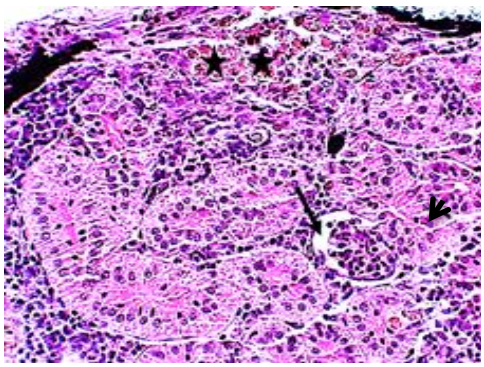

Fig. 65. Section cross in kidney tissue of G6 after 30 days of treatment, expansion of the renal space Bowman ➔ , melanomacrophage clusters (★), intact tubules (➢), H&E, 40 X.

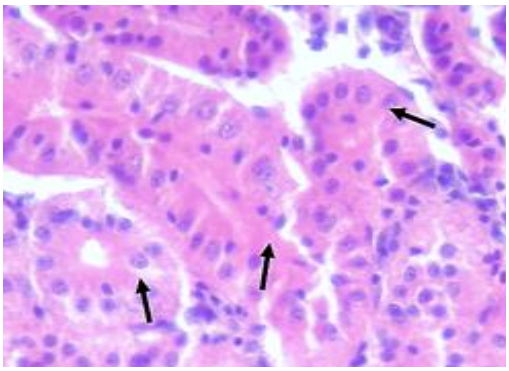

Fig. 66. Section cross in kidney tissue of G6 after 30 days of treatment, necrosis in the renal tubules(➔), H&E, 40 X

4.Discussion

The study of histopathological changes is of great importance in evaluating the health status of fish [15], [16]. These physiological and structural changes and disorders are a response to environmental pollutants [17]. The results of the histopathological study showed the presence of some histopathological changes in the kidneys of fish exposed to two different concentrations of cadmium chloride (16.86 and 56.2) mg/L during successive periods (3, 7, 15 and 30) days of the experiment. These are natural reactions and defence mechanisms that work to prevent the entry of pollutants from the aquatic environment [18]. The kidney works as an essential organ in the elimination of liquid waste and the purification and filtration of blood laden with toxins [19]. The dilatation of renal tubules may be due to the disturbance of osmotic pressure due to cadmium toxicity, which affects

the adhesion particles and cell-cell connections that connect the epithelial cells of the renal tubules to the basement membrane. This causes the breakdown or denaturation of cell-cell communication proteins, which in turn leads to the separation of these cells from the epithelial tissue lining the tubules[20]. This dilatation is reflected in the absorption and reabsorption processes along the renal tubule [21]. Increased acidic staining of the cytoplasm was observed in the epithelial cells of the renal tubules. This may occur as a result of a change in the pH of the cytoplasm, which may cause inhibition of cytoplasmic proteins and lead to degeneration [22]. The atrophy of the nuclei, their appearance as dark spheres and their hyper staining with eosin indicate the early stages of necrosis.

Thus, the severity of the changes increases, and their development reaches the state of severe necrosis [23]. Under conditions of oxygen deficiency, the toxic effect of cadmium on the cell membrane increases. It affects the lateral bonds between the cells lining the blood vessels, causing these bonds to break. Consequently, their permeability increases, causing red blood cells to leak out of the blood vessel. This leads to bleeding [24]. The cause of the vacuolar degeneration observed in some tissue sections may be due to the imbalance in osmotic pressure. This imbalance leads to the accumulation of water inside the cells. Necrosis may occur due to poor blood delivery to these cells as a result of continued exposure to toxic substances [25]. The cause of degeneration may be due to inhibition of cellular protein synthesis, which in turn leads to a decrease in adenosine triphosphate (ATP) due to an increase in the process of glycolysis [26]. Alternatively, it may occur as a result of the accumulation of multiple sugars, water and fatty substances in the cell. Thus, it appears in The formation of vacuoles that fill the cell cytoplasm due to weakness or dysfunction in some of the cell's vital functions, such as cellular metabolism. This may lead to cell atrophy and then death [27].

The cause of edema in the kidneys is attributed to the expansion and congestion of blood vessels. This leads to slowing down blood circulation, increasing the exudation of these vessels, and the accumulation of fluids outside the cell. Consequently, the viscosity of the blood increases, and it becomes stagnant, which is known as striasis [28]. Sometimes, it occurs as a result of changing the permeability of the plasma membranes of cells due to the presence of pathogens, including heavy elements, in the aquatic environment [29],[30]. Increased pressure on the walls of blood vessels may lead to the dissolution of the epithelial cells lining the blood vessels. This causes intracellular bleeding [31].

The entry of heavy metals and toxic substances into the body produces and activates free radicals, in addition to the presence of these radicals formed naturally during energy production and cellular metabolism [32]. Free radicals may affect the antioxidant system. This leads to oxidative stress in cells [33]. This explains the reason for the cellular damage observed in different areas of the renal tissue, such as atrophy of some cells, degeneration of various types, necrosis, etc. The severity of the cells' effects increases with increasing oxidative stress and exposure period [33].

In some areas of necrotic cells, aggregates of melanomacrophage cells were observed. These cells contain a variety of pigments, including melanin. These pigments increase in extent and size as a result of exposure to pathogens and environmental stress. They appear to act as a central reservoir for resistant intracellular bacteria that may develop in chronic infections. As a result of hemolysis, the cells capture and store iron. These appear to be important functions of these cells [34].

Metaplasia observed in some renal tubule cells is one of the forms of the inflammatory response of epithelial tissue that suffers from stress due to pollutants. It is a type of cellular adaptation [20]. Cells change from their normal shape to another shape under the influence of the pollutant so that the new shape is more resistant to the toxicity of cadmium and the substances resulting from its metabolism and an attempt to reduce the toxic effect on the cell and an attempt to keep the cell alive. Where it can perform its functions as much as possible. However, when exposure to the pollutant that causes harm continues and exceeds the cell's limits to tolerate its damage, the cell may undergo pathological changes that end in its death and decomposition [35]. This explains the reason for the cases of necrosis, especially in the last periods of the experiment.

The appearance of pink, non-living aggregates and their accumulation in irregular masses in the blood-forming tissue and within the renal tubule cavities may be a defence mechanism of the cells against the stress they may be exposed to due to cadmium toxicity. Therefore, it secretes protein materials as a result of its damage [27].The formation of brown non-living materials occurs in the tissues of cells of some organs due to exposure to pathogens. They may represent protein materials such as Ubiquitin, which are produced by cells as a defensive response to neutralize the harmful toxic effect or remove waste by activating the Ubiquitin-proteasome system [27]. Cadmium affects the permeability of the membranes of lysosomes. This is considered an indicator of the cellular response when cases of poisoning with heavy metals occur [36] due to its interference with the glycolipid in the plasma membrane. Thus, it makes it more permeable to acid hydrolase and more fragile. This leads to cellular degeneration [37]. The severity of histopathological changes in the kidneys increased with the length of the experiment. Increasing the concentration of cadmium chloride works to prevent the Bioaccumulation of pollutants within the body's tissues and the inability of the special systems to remove and get rid of these toxins despite the presence of many responses to the toxic action. This is confirmed by the emergence of inflammation and histopathological changes, the severity of which increases with the increase in the period of exposure and results from the accumulation of free radicals or as a result of lipid oxidation processes [38].

The results of the study showed the role of thyme oil in reducing the severity of histopathological changes in the groups treated with thyme oil alone and the combined treatment with cadmium chloride with thyme oil. It had a significant effect, especially in the last periods of the experiment, as it contains antioxidants, which are essential for regulating free radical levels, maintaining the integrity of renal tissue, and preventing cell damage [39]. The presence of biologically active compounds in medicinal plant oils, such as flavonoids, phenols, sugars, alkaloids, organic acids, volatile oils, and others, enhances the activity of antioxidant enzymes [40],[41]. Phenolic compounds are strong antioxidants and can interact with the phospholipids of the phospholipid bilayer of the membrane to form peroxide chain reactions [40]. These oils enhance growth performance, blood and physiological condition, strengthen the immune system, and antioxidant activity [42],[43]. Thymol is the main component of thyme oil and has liver-protective properties [44]. Its antioxidant activity is attributed to the presence of phenolic hydroxyl groups that act as hydrogen donors to radicals resulting from lipid oxidation. Thus, it delays the formation of lipid peroxide and hydrogen peroxide [45].

Many studies agree with our current study, which demonstrates the effective role of thyme oil as a result of it containing many active compounds, the most important of which are thymol and oleic acid -Octadicinnoic acid, which is attributed to the biological effectiveness in resisting diseases and histological pathological changes[46]-[51].

5. Conclusion:

The results of the current study showed the role of thyme oil in reducing the accumulation of cadmium in the kidneys of carp fish over the experimental period. It became clear through studying the pathological tissue changes in the kidneys, which can be relied upon as a bioindicator of pollution in the aquatic environment, which differs according to the tissues of the organs and the type of fish.

Declaration of competing interest

The authors declare that they have no competing interests.

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نموذج الملخص باللغة العربية لمجلة ابحاث البصرة (العلميات)

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معلومات البحث

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الكلمات المفتاحية

زيت الزعتر ، كلوريد الكادميوم ، كلى الاسماك ، التغيرات النسجية ، Cyprinus carpio

الملخص

هدفت الدراسة الحالية الى تقييم دور زيت الزعتر في تقليل التأثيرات المرضية النسجية في كلى اسماك الكارب الاعتيادي(Cyprinus carpio) المعرضة الى تركيزين مختلفين من كلوريد الكادميوم (16.86 و 56.2 ملغم / لتر)، شملت التجربة ست مجموعات تحتوي كل منهاعلى 16 سمكة ، مجموعة السيطرة ،مجموعة المعالجة بتركيز (16.86) ملغم / لتر من كلوريد الكادميوم ، مجموعة المعالجة بتركيز (56.2) ملغم / لتر من كلوريد الكادميوم ، مجموعة المعالجة بزيت الزعتر فقط ،مجموعة المعالجة بتركيز (16.86) ملغم / لتر من كلوريد الكادميوم مع زيت الزعتر ،ومجموعة المعالجة بتركيز 56.2 ملغم / لتر من كلوريد الكادميوم مع زيت الزعتر.

اظهرت النتائج وجود تغيرات مرضية نسجية في كلى الاسماك ، اذ كانت اشد في المجموعتين المعالجة بكلوريد الكادميوم فقط ، بينما انخفضت في المجموعتين المعالجة بكلوريد الكادميوم وزيت الزعتر معاً . كما اظهرت الدراسة ان طول فترة التعرض زاد من شدة التغيرات في المجموعتين المعالجة بكلوريد الكادميوم فقط ، وخصوصا بعد 30 يوم من بداية التجربة .

شملت التغيرات النسجية توسع النبيبات الكلوية ،توسع حيز محفظة بومان ، تنخر وتنكس الخلايا المبطنة وللنبيبات الكلوية ،وزيادة الخلايا البدينة وتجمعات melanomacrophage. في المقابل كانت التغيرات النسجية اقل حدة في المجموعتين المعالجة بكلوريد الكادميوم مع زيت الزعتر ، مما يدل على الدور الوقائي لزيت الزعتر في تقليل حدة التغيرات المرضية النسجية .

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