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Physiological and histopathological changes associated with aluminum induced toxicity on liver in experimental male rats

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Abstract

Background. Access to various chemicals has caused many cases of poisoning in the last decades, people are deliberately, accidentally poisoned or overdose of drugs and chemicals.

Purpose. Designed to clarify the pathophysiological changes caused by using varying doses of Aluminum chloride on liver and the ameliorative effect of zinc on these changes.

Methodology. Fourteen adult male rats were allocated randomly and in equal number (n=6) into seven groups: the rats were euthanized through administration of lethal dose of chloroform, the blood were taken via cardiac puncture method, for biochemical analysis (ALT, AST, ALP, etc.). further aliquot of blood placed in anticoagulant tubes for measurement of hematological parameters (RBC, WBC, PLT, etc.). histological study done by taking liver, kidney, tissue from all experimental groups.

Results. Aluminum chloride exposure significantly increased liver enzymes and caused tissue damage in liver. Zinc chloride treatment effectively restored these parameters to near-normal levels and improved tissue histology, demonstrating its protective role against aluminum-induced toxicity. Microscopic observations of liver sections from control rats revealed that the liver parenchyma was composed of lobules and hepatocytes arranged around the central vein. After dosing, these cells showed liver toxicity, damage, and hemorrhage. However, the zinc chloride-treated groups had healthier liver tissue, with most cells arranged as hepatic plaques separated by vascular sinusoids.

Conclusion. The exposure to $AlCl_3$, the greater its effects on the rats' organ, liver, more harmful, a dose of 100 mg of $AlCl_3$ is than a dose of 20 or 40 mg.

Keywords: aluminum; biochemical analysis; liver enzyme; zinc

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Introduction

Access to various chemicals has caused many cases of poisoning in the last decades people are deliberately or accidentally poisoned by misuse or overdose of drugs and chemicals. Heavy metals are important examples among these agents. Materials are released from natural sources or industrial wastes and can threaten human health [1]. One of these agents is aluminum (Al), which is a silvery white, soft, malleable, nonmagnetic, and ductile metal, Aluminum forms about 8% of the Earth's crust and is the third most abundant element after oxygen and silicon [2].

Aluminum reaches human bodies through the skin, the gastrointestinal system, and lungs [3]. Its long record of use in medicine includes its use as an adjuvant in vaccines and an agent against pathological hyperhidrosis, both of which have been proven to have minimal adverse effects in investigations [4]. The World Health Organization (WHO) suggests that 1 mg of aluminum per kilogram of body weight is the maximum amount that can be consumed each day [5].

Bread, rice, soft drinks, milk powder, and table salts are a few products which have a lot of aluminum additions [6]. Not only provides food include metal, but cooking using aluminum cookware, covering food in aluminum foil, and storing food in aluminum cans can also cause humans to consume large amounts of aluminum [7]. When using aluminum kitchen equipment, the heat from the tools causes metal to pass into the meal [8]. Metals are considered as important components of a physiologically active cell, and imbalance in their levels can lead to various diseased conditions.

Aluminum (Al) is an environmental neurotoxic ant, which is etiologically related to several neurodegenerative disorders like Alzheimer's, whereas zinc (Zn) is an essential trace element that regulates a large number of metabolic processes in the brain [9]. Heavy metals like aluminum can interfere with the absorption or function of essential minerals such as zinc if present in high amounts, potentially disrupting zinc-dependent biological functions. This is a general inference based on known mineral interactions [10]. Because zinc and aluminum compete for binding sites on metallothioneins and transport proteins

(such as divalent metal transporter 1, DMT1), zinc decreases the absorption of aluminum in the intestines and its buildup in tissues [11].

Zinc acts as a neuroprotective and hepatoprotective agent against aluminum toxicity by reducing oxidative stress, restoring antioxidant defenses, preventing tissue damage, and modulating metal accumulation [12]. Additionally, by maintaining the integrity of the blood-brain barrier (BBB), it guards against Al-induced neuronal damage [3]. Zinc up regulates metallothioneins (MTs), which bind both Zn and toxic metals like Al, facilitating their detoxification [13]. Zn-induced MTs can sequester Al, reducing its free ionic form and subsequent toxicity [14]. Zinc is utilized as an enzyme in the rubber industry, a white pigment in paints, and in the manufacturing of plastics, cosmetics, wallpaper, and printing inks. It is utilized in pharmaceutical formulations for anti-aging skin care products, among other things, as an antioxidant.

Methodology

Materials

The materials used in this study are aluminum chloride (AlCl_3) and zinc chloride (ZnCl_2). Both chemicals were procured from Alpha Chemical India. Aluminum chloride (AlCl_3) and zinc chloride (ZnCl_2). Their main chemical and physical properties are summarized below. Aluminum Chloride (AlCl_3): A white to yellowish crystalline solid with a purity of 98%, chemical formula: AlCl_3 , molar mass: 133.34 g/mol, density: 2.44 g/cm³, sublimates at approximately 190 °C, soluble in water, ethanol, and chloroform. Zinc Chloride (ZnCl_2): A white crystalline solid with a purity of 98%, chemical formula: ZnCl_2 , molar mass: 136.28 g/mol, density: 2.91 g/cm³, melting point: 290 °C, boiling point: 732 °C.

Study design and sampling

Forty-two male rats (*Rattus norvegicus*) were used in these experiments, the animals were obtained from the College of Veterinary Medicine, University of Basrah. They were maintained and cared for under appropriate environmental conditions, including room temperature, proper nutrition, and controlled lighting, in accordance with ethical guidelines for laboratory animal care. Rats were separated and divided randomly into (7) equal groups, (6) rats in each group as the following: Group one (control group): In this group rats were drenched with normal saline (1ml) every day for the experiment periods. Group two: Within this group rats were drenched orally toxic dosage with (20 mg/kg single dosage/B.W) of AlCl_3 [15]. Group three: For this group

rats were drenched orally toxic dosage with (40 mg/kg single dosage/B.W) of AlCl_3 [16]. Group Four: Into this group rats were drenched orally toxic dosage with (100 mg/kg single dosage/B.W) of Aluminum chloride [17]. Group Five: Received both (50 mg/kg B.W) of ZnCl_2 and (20 mg/kg B.W) of AlCl_3 [18]. Group six: Co-administered with (50 mg/kg B.W) of ZnCl_2 and (40 mg/kg B.W) of AlCl_3 . Group seven: Cotreated with both (50 mg/kg B.W) of ZnCl_2 and (100 mg/kg B.W) of AlCl_3 .

Biochemical analysis study

Following each treatment, the rats were euthanized through administration of lethal dose of chloroform, the blood were taken via cardiac puncture method, serum was separated by centrifugation for 10min at 300 orpm and stored at -20°C and utilized for biochemical analysis (ALT, AST, ALP, BUN, CREA, Testosterone, FSH, LH, Parathyroid hormone, Erythropoietin, MDA, and Glutathione peroxidase). Some biochemical tests (RBC, WBC, PLT, LYM, MON, GRA) were done on rats serum to determine liver enzymes activity by using special kits.

Histopathological Study

histological study done by taking liver, kidney, tissue from all experimental groups, washed with normal saline to remove blood and tissue debris then, suitable size of specimens were cut and transferred to be fixed with (10%) formalin fixative process [19; 20].

Statistical Analysis

The data of this study were analyzed by using SPSS version 26, based in using one was ANOVA for mean variation, and independent sample t-test.

Results

The results in (Table 1) and (Fig. 1-3), showed significant increase ($p<0.05$) in AST level in rats treated with 100 mg/kg B.W/ AlCl_3 compared with control group and with 20mg AlCl_3 treated group. However, there was significant decrease ($p<0.05$) in AST level in group co-treated with both 20 mg AlCl_3 and ZnCl_2 compared with 40 mg and 100 mg chloride aluminum treated groups.

Rats co-administered with 40 mg AlCl_3 and ZnCl_2 showed significant decrease ($p<0.05$) in AST concentration, in comparison with 100 mg AlCl_3 , treated group, while there was significant increase ($p<0.05$) in AST level in 40 mg AlCl_3 , treated group compared with control group, in addition, ALP showed significant increase ($p<0.05$) in rats treated with 100mg AlCl_3 in comparison with control group and other groups expect group co-administered with both 100 mg AlCl_3 and ZnCl_2 , our present study revealed to significant increase ($p<0.05$) in

ALP concentration in 100 mg AlCl_3 + ZnCl_2 co-treated group compared with group co-treated with both 20 mg AlCl_3 combined with ZnCl_2 and with 40mg AlCl_3 combined with ZnCl_2 .

Table 1.

Effect different doses of AlCl_3 and combination with ZnCl_2 on liver enzymes mean $\pm$ SD in experiment al rats

Groups	Parameter		
	ALT	AST	ALP
Control (Normal saline 0.9%. NaCl)	55.66 $\pm$ 7.23 a	97.67 $\pm$ 7.63 C	152 $\pm$ 17.08 C
G2 AlCl_3 (20mg)	56.33 $\pm$ 5.50 a	111.67 $\pm$ 20.74 bc	240 $\pm$ 17.52 Bc
G3 AlCl_3 (40mg)	58.33 $\pm$ 6.11 a	134.67 $\pm$ 23.69 ab	271 $\pm$ 215.64 Bc
G4 AlCl_3 (100 mg)	59.66 $\pm$ 3.78 a	163 $\pm$ 31.57 a	472 $\pm$ 81.279 A
G5 AlCl_3 20mg + 50mg ZnCl_2	55 $\pm$ 0.00 a	105 $\pm$ 21.37 C	205 $\pm$ 62.37 Bc
G6 AlCl_3 40mg+ 50mg ZnCl_2	55.77 $\pm$ 2.54 a	116.67 $\pm$ 19.65 bc	213 I 112,263 Bc
G7 AlCl_3 100mg+50 mg ZnCl_2	61 $\pm$ 5.29 a	148.67 $\pm$ 18.58 ab	370.67 $\pm$ 14.048 Ab

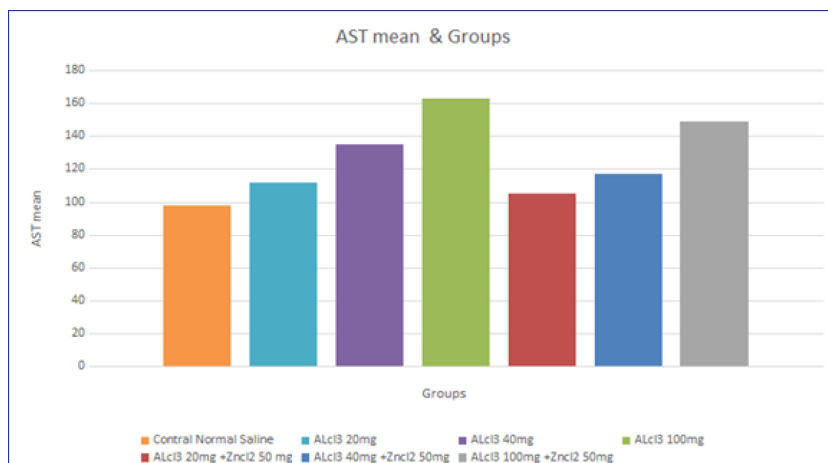


Fig. 1. Effect different doses of AlCl_3 and combination with ZnCl_2 on liver enzyme AST mean $\pm$ SD U/L in experiment al rats

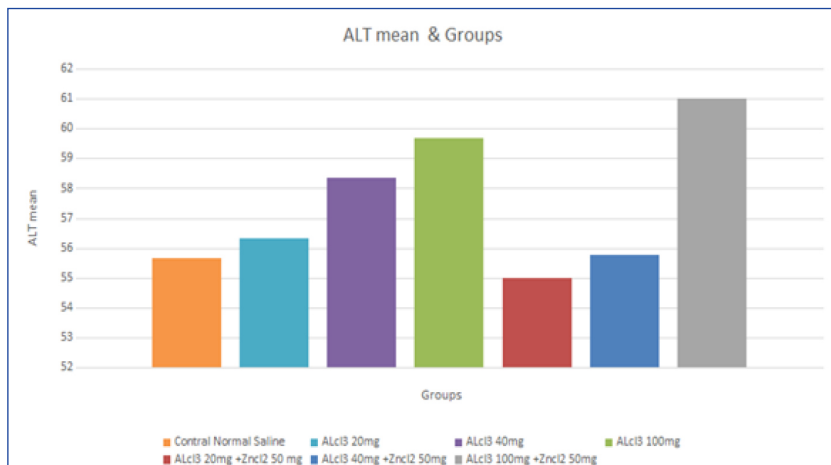


Fig. 2. Effect different doses of AlCl_3 and combination with ZnCl_2 on liver enzyme ALT mean $\pm$ SD U/L in experiment al rats

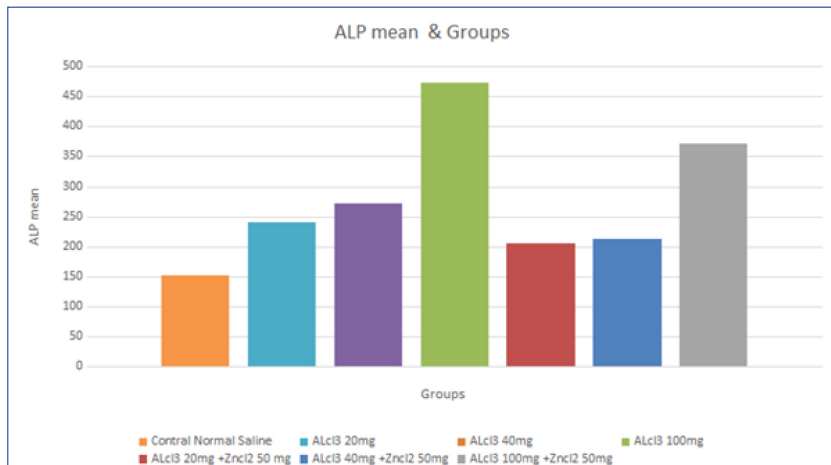


Fig. 3. Effect different doses of AlCl_3 and combination with ZnCl_2 on liver enzyme ALP mean $\pm$ SD U/L in experiment al rats

Recent study showed that liver sections related rats exposed to at 20,40 mg/ AlCl_3 /B.W dose and treated with 50 mg ZnCl_2 B.W dose have less and mild histological changes, liver tissue more healthy, most liver cells (hepatocytes) arranged as hepatic plates separated by vascular Sinusoids and the hepatocytes

with ovoid nucleus, still mild to moderate cytoplasmic vacillation and eosinophilia especially with liver sections of rats treated with AlCl_3 (40) mg then with ZnCl_2 , moreover normal Central vein lining with endothelial cells, mild focal degeneration and Karyopyknosis (Fig. 4-9).

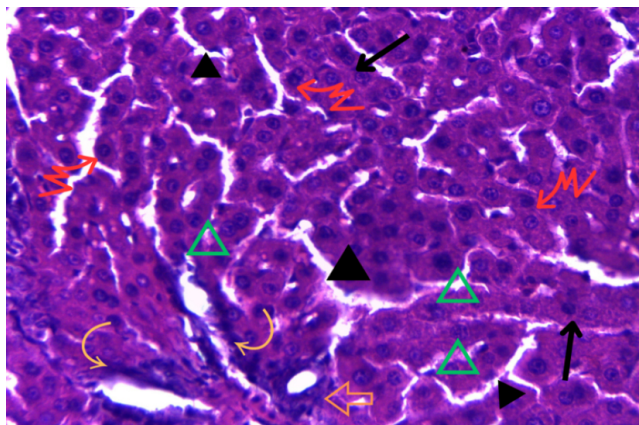


Fig. 4. Photomicrograph in liver section from control rat showing each liver lobule consist of hepatic cords (plate) (→) arranged hepatocytes (△) with dense, ovoid nuclei (↪), branch of bile duct (⇨) and dilated normal portal ducts (↘), the hepatic cords separated by normal sinusoids (▲). (H&E) stain (40x).

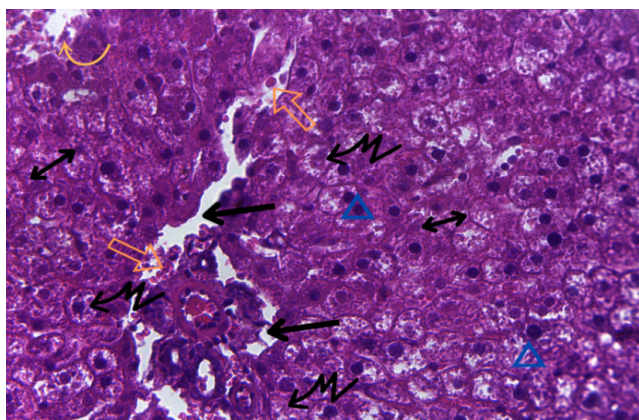


Fig. 5. Photomicrograph in liver section from rat group (2) treated with AlCl_3 (20 mg) showing dilated sinusoids (→) pyknotic nuclei (△), eosinophilia (↪) extravasation of RBCs (⇨), hemorrhage (⇨) the hepatocytes lost their boundaries (↔) (H&E) stain (40x).

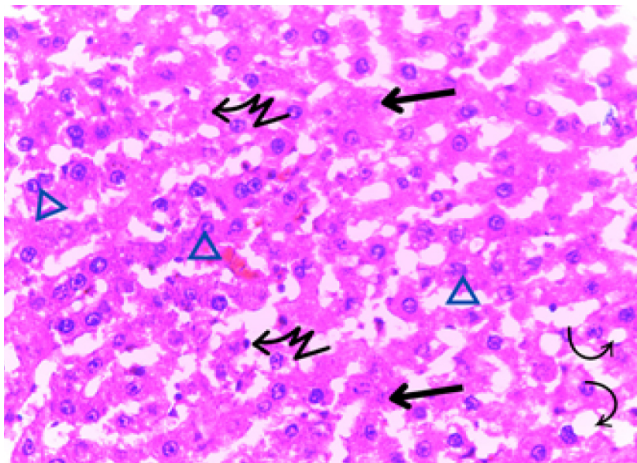


Fig. 6. Photomicrograph in liver section from rat group (3) dosage AlCl_3 (40mg) showing hepatocyte degeneration ($\rightarrow$), hepatocytes with karyorrhexis nucleus ($\triangle$), moderate valuations ($\Rightarrow$), and most cells lost their normal boundaries, ($\curvearrowright$). (H&E) stain (40x).

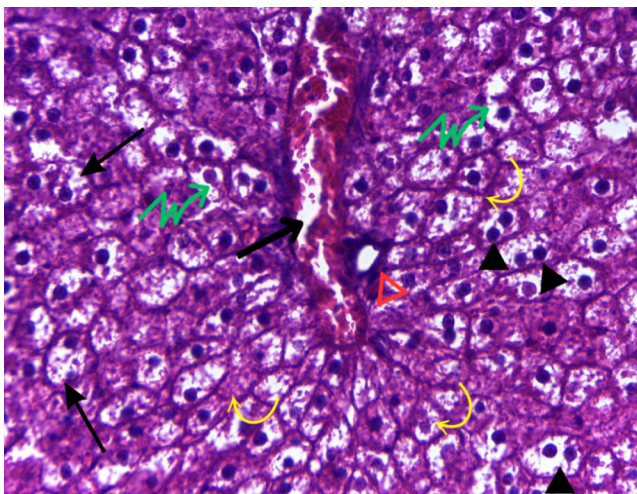


Fig. 7. Photomicrograph in liver section from rat group (4) dosage AlCl_3 (100 mg) showing Severe inflammation, severe congestion ($\rightarrow$), bile duct hyperplasia ($\triangle$), hepatocytes vacuolation ($\Rightarrow$), hepatocytes necrosis ($\rightarrow$), most hepatocytes with two nuclei ($\blacktriangle$) showed eosinophilia and increase thickness of liver cells cell membrane ($\Rightarrow$). (H&E) stain (40x)

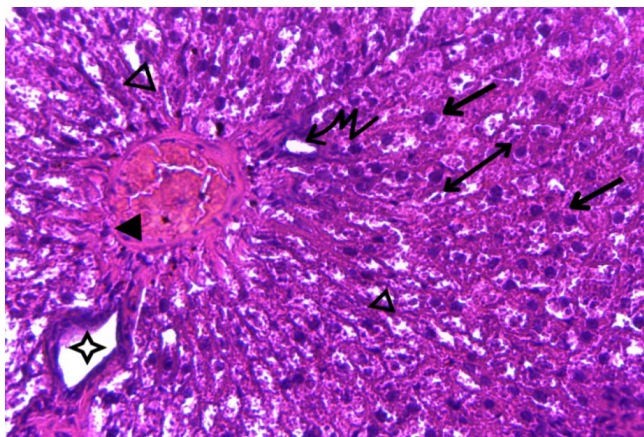


Fig. 8. photomicrograph in liver section from rat group (5) dosage AlCl_3 (20 mg) + ZnCl_2 (50 mg), showed normal liver tissue ($\longleftrightarrow$), most hepatocytes ($\rightarrow$) with avoid nucleus, vascular sinusoids (Δ), normal central vein ($\star$), normal branch of bile duct ($\hookrightarrow$) and mild hemorrhage ($\blacktriangle$) (H&E) stain (40x)

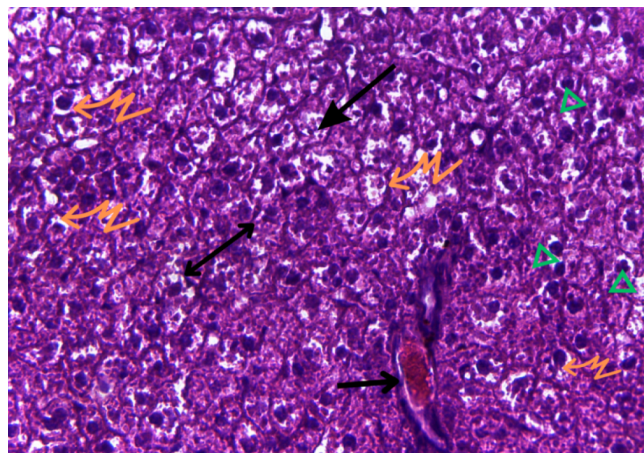


Fig. 9. Photomicrograph in liver section from rat group (6) dosage AlCl_3 (40 mg) + ZnCl_2 (50 mg) showed normal central vein ($\rightarrow$), mild cytoplasmic vacuolation ($\hookrightarrow$), karyopyknosis (Δ), still hepatocytes with granular cytoplasm ($\rightarrow$)the liver parenchyma more regular ($\longleftrightarrow$). (H&E) stain (40x).

The present study revealed ameliorated histological changes when using ZnCl_2 compared to (AlCl_3) treatment alone, mild focal necrosis areas with sever infiltra-

tion of inflammatory cells within hepatic lobule and around the central vein, the cells also showed manifestation of fatty changes, however there was less protective effect of (ZnCl_2) in the group that was treated with high a dose of (AlCl_3), degenerative and necrotic changes in the hepatocytes at different stages within the same region and diffuse of Kupffer cells between degenerated hepatocytes (Fig. 10).

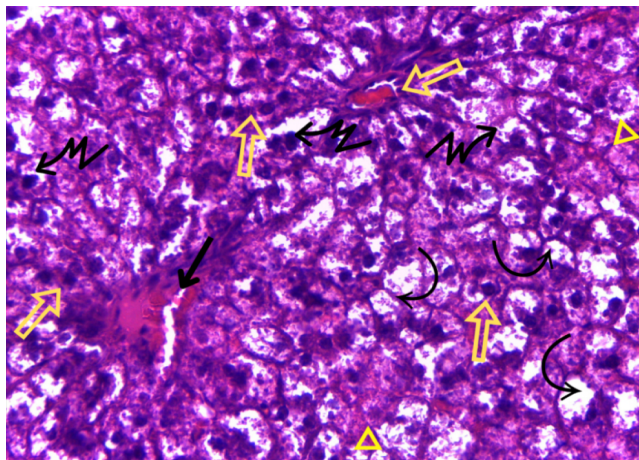


Fig. 10. Photomicrograph in liver section from rat group (7) dosage AlCl_3 (100 mg) + ZnCl_2 (50 mg) showing congestion of central vein ($\rightarrow$) cytoplasmic vacuolation ($\curvearrowright$) hepatocyte degeneration ($\triangle$), infiltration of Kupffer cells ($\Rightarrow$), between hepatocytes and fatty changes ($\curvearrowright$). (H&E) stain (40x).

Discussion

The results of the present study showed a significant increase in serum AST and ALT in rats treated with aluminum chloride compared with the healthy group, which is consistent with previous studies [21-23].

The reason for that rise of the AST enzyme is also attributed to damage to hepatic cells, and the increase in the level of this enzyme is a sign of liver damage and changes in its function [24]. The accumulation of aluminum (Al), a neurotoxic metal, in the kidneys, bones, and brain causes oxidative stress, inflammation, and cellular damage. Chronic exposure to aluminum in rats is linked to: neurotoxicity (deficits in memory, cognitive decline, and disease similar to Alzheimer's), and oxidative stress (decreased antioxidant enzymes such as SOD, CAT, and GSH, and increased lipid peroxidation).

A crucial topic of study in materials science and biomedical engineering is the toxicity of aluminum and the therapeutic potential of zinc as a miti-

gating agent. Although aluminum is utilized extensively in consumer and industrial settings, when it builds up in biological systems, it can have neurotoxic consequences associated with oxidative stress, cellular dysfunction, and neurodegenerative diseases [25]. The results of the present study showed a significant increase in serum AST and ALT in rats treated with aluminum chloride compared with the healthy group, which is consistent with previous studies [21-23]. Who explained that the exposure to aluminum chloride causes liver necrosis [26].

The reason for that rise of the AST enzyme is also attributed to damage to hepatic cells, and the increase in the level of this enzyme is a sign of liver damage and changes in its function [24]. There was significant decrease ($p < 0.05$) in AST level in group co-treated with both 20mg $AlCl_3$ and Zinc chloride compared with 40mg and 100mg chloride aluminum treated groups, which agree with Eze *et al.*, who stated that Zinc may play an important role in the prevention of hepatocellular damage [27]. Zinc is effective in attenuating the liver damage inflicted by Al toxicity [28].

An increase in serum AST and ALT in rats treated with $AlCl_3$ was in agreement with previous findings demonstrating that $AlCl_3$ exposure promoted liver necrosis [29]. ALP showed significant increase ($p < 0.05$) in rats treated with 100 mg $AlCl_3$ in comparison with control group and other groups which agree with Al-Hazmi *et al.*, who showed that increased Al concentration in the body is shown to induce detrimental biochemical and histological changes [23]. Zinc (Zn) is an essential trace element with antioxidant, anti-inflammatory, and neuroprotective properties. However, there was same result about histological changes in liver of rat when administered with another toxic material such as lead acetate which include fusion and clumping of hepatocyte, degeneration, pyknosis and formation of bubbles [30].

Mechanisms by which $ZnCl_2$ may counteract aluminum toxicity competitive interaction aluminum uptake may be decreased by zinc and aluminum competing for binding and absorption sites in the brain and gut. Antioxidant effects, zinc decreases aluminum-induced oxidative damage and increases the activity of antioxidant enzymes (SOD, CAT, and GSH) [31].

Zinc has an anti-inflammatory effect by inhibiting pro-inflammatory cytokines (TNF- α , IL-6) that are increased as a result of exposure to aluminum. Zinc inhibits aluminum-induced neurodegeneration, stabilizes neuronal membranes, and modifies synaptic function. Although zinc is not a chelator in and of itself, it may aid in reestablishing metal homeostasis, which would lessen the toxicity of aluminum in an indirect manner [32].

Conclusion

The present study concluded that the damage was more severe with an increase in the dose of AlCl_3 100 mg and less severe with lower doses of 20, 40 mg. Clear improvements in all of the above were observed when ZnCl_2 was dosed at a dose of 50 mg, which had a clear role in returning things to almost normal, which proved the therapeutic and preventive role of zinc.

Ethics committee conclusion. This study follows the ethics guidelines of the Faculty of Biology Department, College of Science, University of Basrah, Iraq.

Conflict of interest information. The authors declare that there is no conflict of interest.

Sponsorship information. This study didn't receive any funding support.

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