Histological changes in the liver, kidney and spleen of White Albino Rat after Aluminum Chloride administration

Hadaf H. Mohammed[1]; Ibtisam K. Abd Ali[2] and Ali F. Reshag[1]

[1] Department of Anatomy and Histology, College of Veterinary Medicine, [2] Department of Basic Sciences, College of Nursing, Baghdad University, Iraq.

E-mail: hadafs@yahoo.com

Received: 8/6/2016

Accepted: 8/1/2017

Summary

The study was carried out to evaluate histological changes induced by Aluminum chloride in Wistar rats. Forty rats were uniformly divided into two different groups: The control group were given distilled water during period of experiment and the first group were given oral daily dose of Aluminium chloride (37 mg\kg B.W). After 60 days of treatment rats were sacrificed. Liver, kidney and spleen were processed and stained with Haematoxylin and Eosin the lesions of these organs were characterized by degeneration, necrosis, and congestion which meant that Aluminum chloride was nephrotoxic as well as hepatotoxic in nature. Microscopic examination results showed that Aluminum chloride exposure was harmful to the histological structure of liver, kidney and spleen.

Keywords: Aluminium Chloride, Liver, Kidney, Spleen, Wistar Rats.

Introduction

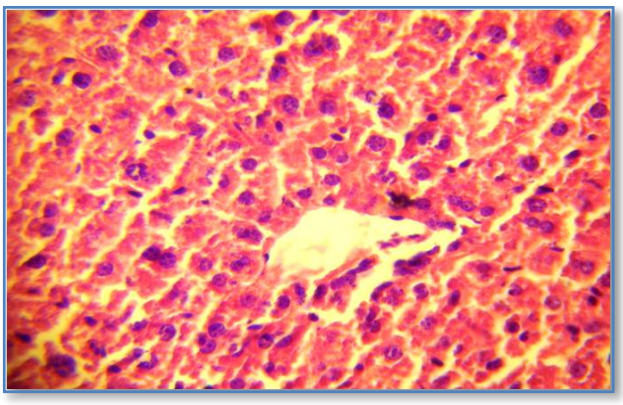
Aluminum chloride (ALCL3) is an element that occurs at the environment, in combination with other elements or salts. It combines with other elements to form compounds, some of which are soluble in water such as chloride, hydroxide, silicate, sulphate and phosphate. Aluminium is present in many manufactured foods and medicines and is also added to drinking water for purification purposes; it is commonly used in our daily life mainly in food products (1). The sources of Aluminum come from yellow cheese, salts, and spices also widely used in food additives, toothpaste and in medicine uses e.g. antacids buffered aspirin and vaccines (2 and 3). Human exposure to Aluminium has been increasing over the last years. Patients on dialysis or on long-term treatment with total parenteral nutrition have been shown to accumulate this metal in different organs such as brain, bone, liver and kidney accompanied by renal failure (4). Epidemiological studies have indicated a link between Aluminum in drinking water and Alzheimer's disease and memory deficits after Aluminum exposure. Repeated or prolonged contact may cause skin sensitization, central nervous system and resulting ataxia, sensory and memory disturbances, tremors, muscle weakness and kidney impairment (5). The objective of this study to investigate the histological changes in liver, kidney and spleen of albino rats due to Aluminum chloride administration.

Materials and Methods

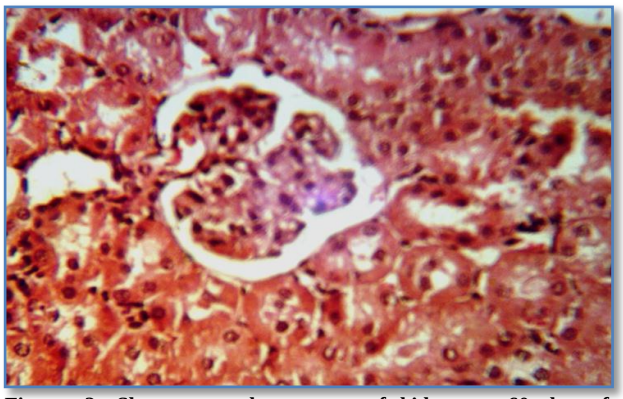
Fourty Albino Wistar rats of both sexes were used at weight range of (250-300) grams placed in the animal house of the College of Veterinary Medicine-Baghdad University. Housing conditions was maintained at 20-25ºC in air conditioned room, the room was ventilated by using ventilation vacuum, and the light-dark cycle was 14\10 in housing place. The litter of the cages was changed weekly (6). Special rat feed pellet and water provided. In this experiment, rats were divided equally into two groups; the control group was given distilled water throughout the experimental period. In the treated group orally dosed with Aluminum chloride daily at 37 mg/ kg B.W. according to (7) in the drinking water, all over the 60 days of the experiment. At the end of experiment the rats of both groups were sacrificed. Tissues obtained for histological evaluation included liver, kidney and spleen. All tissues were placed in 10% buffered formalin, then embedded in paraffin, and stained with hematoxylin and eosin (H and E) and examined by light microscope (8).

Results and Discussion

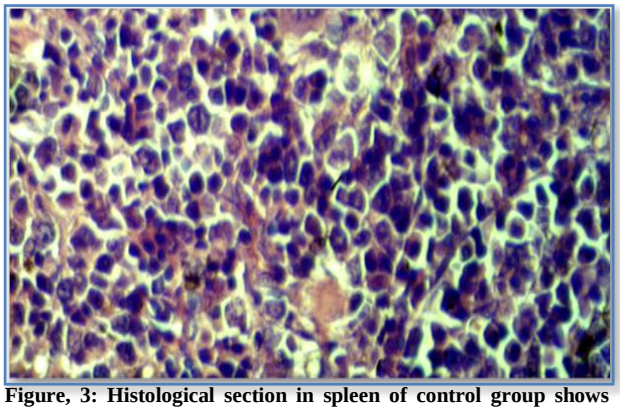
Histological examination of the control group shows normal appearance of hepatic lobules. Each lobule was formed of cords of hepatocyte cells radiating from the central vein with sinusoidal, dilation the liver cells were polyhedral cells with acidophilic cytoplasm and each cell had a rounded pale stained nucleus (Fig. 1). Section tissues for kidney showed normal glomeruli, and tubulointerstitial cells (Fig. 2) while structure of spleen was composed of white and red pulp, and blood vessels (Fig. 3). In the treated group, the liver tissue showed a lack in normal pattern of hepatic cords with dark nuclei of irregular shape of hepatocytes, dark division nuclei of hepatocytes (Fig. 4). There was coagulative necrosis of hepatocytes characterized by pyknotic or disappear and dilated of sinusoids with severe fatty changes represented by round clear sharp vacuoles in the cytoplasm of hepatocytes (Fig. 5). The kidney of treated group showed of inflammatory cell infiltration particularly neutrophils, macrophages and lymphocytes between renal tubules, with the congested blood vessels and vacuolar degeneration of epithelial cells and severe congested blood vesseles between renal tubules (Fig. 6), and also the kidney showed inflammatory cells infiltration in the wall of collected tubules with hyperplasia of epithial cells of collected ducts and these cells aggregated as hyperchromatic pleomorphic cells arranged as mass or sheath or glandular structure and atrophy of glomerular tufts with dilated Bowmanns' space ,congested blood vessls and acute cellular degeneration (Fig.7). Section of spleen tissue in treated group showed severe depletion of white pulp with congested red pulp and inflammatory cells particularly neutrophils in congested red pulp and blood vessels (Fig. 8).



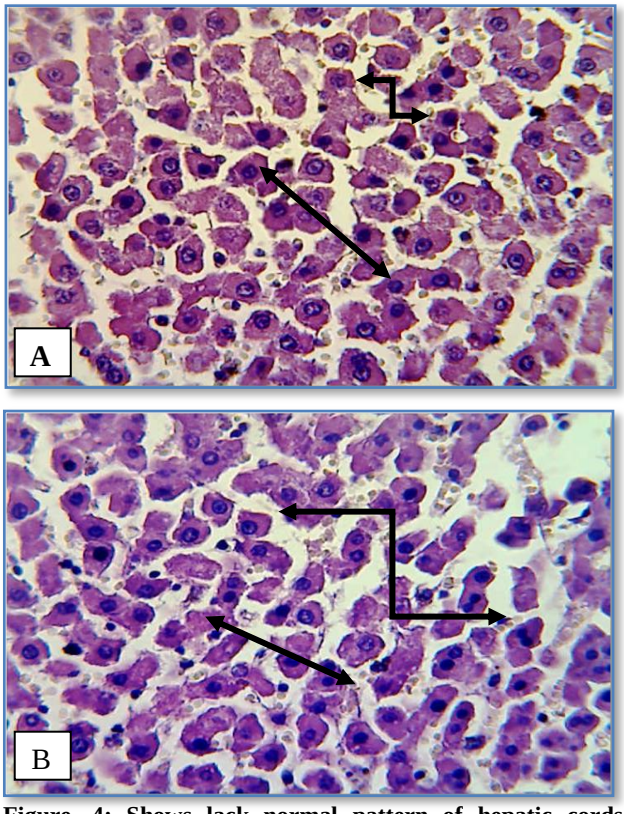
Figure, 1: Histological section in liver shows normal appearance of liver at 60 day of experiment in the control group, (40 x H and E).



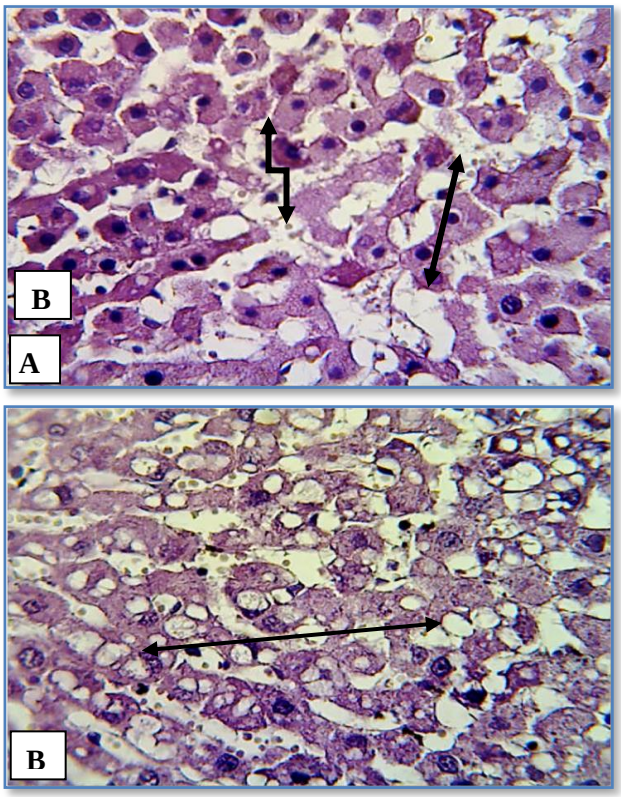
Figure, 2: Shows normal structure of kidney at 60 day of experiment of the control group, (40x H and E).



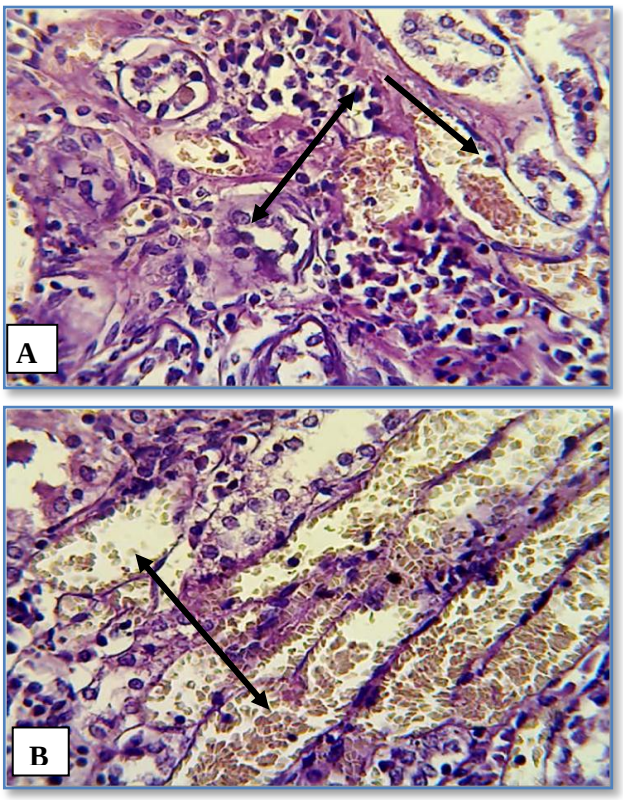
Figure, 3: Histological section in spleen of control group shows normal histological structure (40 x H and E).



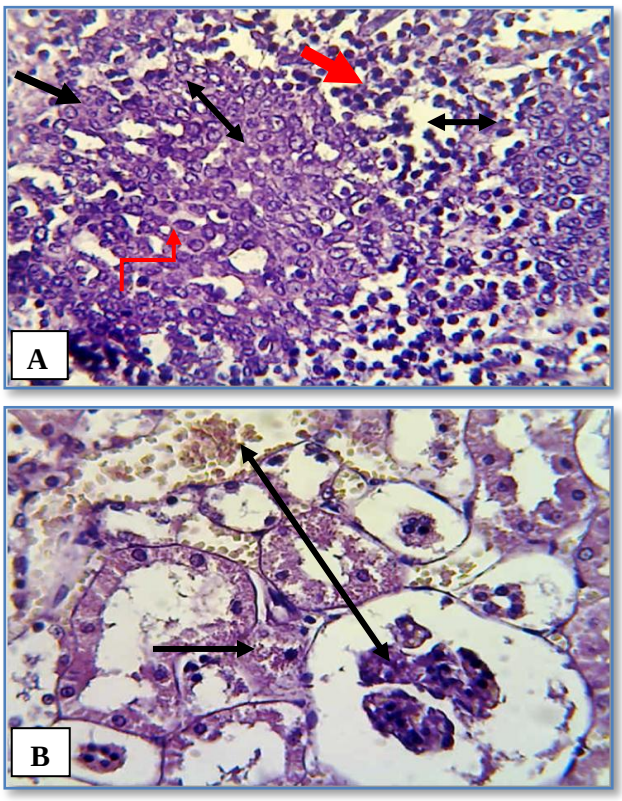
Figure, 4: Shows lack normal pattern of hepatic cords dark division nuclei of irregular shape of hepatocytes (B) at 60 day of experiment in treated group (H and E stain 40X)



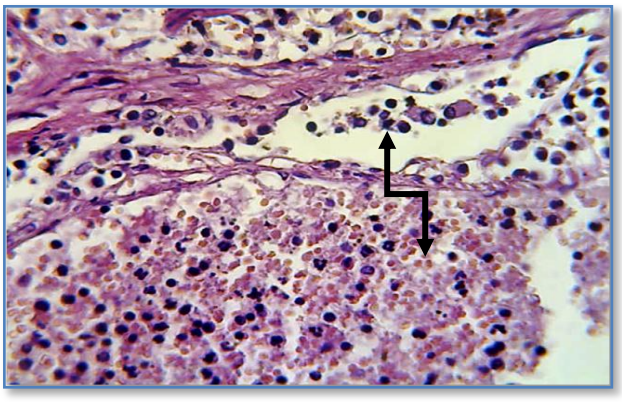
Figure, 5: Shows coagulative necrosis of hepatocytes characterized by pyknotic or disappear and dilated of sinusoids (A), severe fatty changes characterized by round clear sharp vacuoles in the cytoplasm of hepatocytes (B) at 60 day of experiment in treated group (H and E stain 40X)

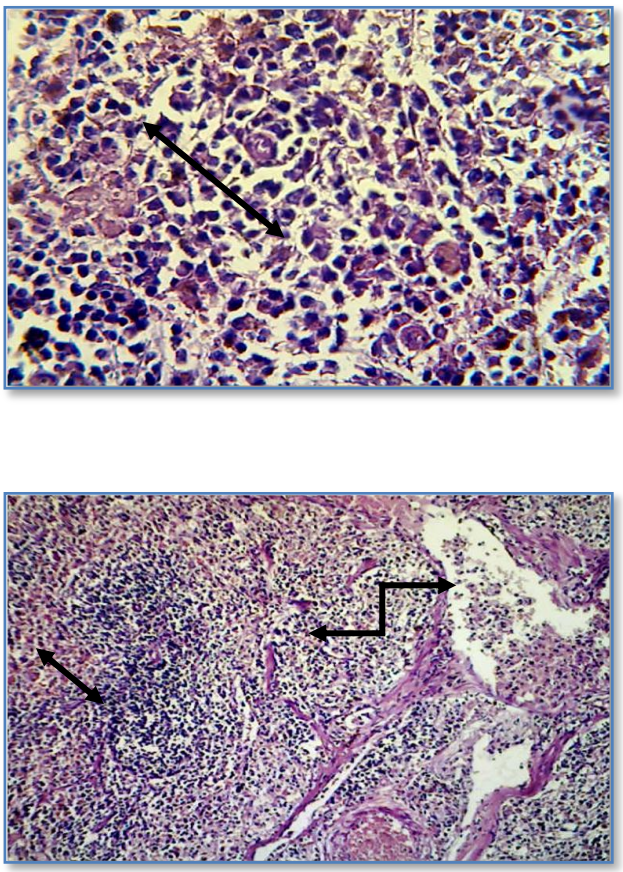


Figure, 6: Shows inflammatory cells infiltration particularly neutrophils, macrophages and lymphocytes between renal tubules, around congested blood vessels and vacuolar degenerationnof epithelial cells (A) and showing severe congested dilated blood vesseles between renal tubules (B) at 60 day of experiment in treated group (H and E stain 40X)



Figure, 7: Shows inflammatory cells infiltration in the wall of collected tubules with hyperplasia of epithial cells of collected ducts and these cells aggregated as hyper chromatic pleomorphic cells arranged as mass or sheath or glandular structure atrophy of glomerular tufts (A) and dilated Bowmanns, space, congested blood vessls with acute cellular degeneration (B) at 60 day of experiment in treated group (H and E stain 40X).





Figure, 8: Shows depletion white pulp ↔ with inflammatory cells particularly neutrophils in congested red pulp and blood vesseles at 60 day of experiment in treated group ↵ (H and E stain 40X).

Aluminum is a heavy metal that occurs in nature as oxide or salts; it is one of the most hazardous and cumulative environmental pollutants (9). It is absorbed from gastrointestinal tract, bounds to erythrocytes and is widely distributed initially to soft tissues such as liver, kidney, and spleen. Liver is the major site of Aluminum accumulation in the experimental animals (10). The lesions in liver may be due to the action of Aluminum chloride on hepatic glycogen and imbalance in the antioxidant protective mechanisms leading to oxidative stress in the cells also effect on DNA content and the ability to incorporate amino acid into protein (11). Treatment with Aluminum chloride induced the increase in reactive oxygen species accumulating in rat brain leading to lipid peroxidation, protein degradation, and finally to cell death (12). The changes of hepatocyte in the present study are showing the hepatocytic degenerations, necrosis and severe congestion of the blood vessels. These are similar to the findings of (13) and in agreement with (14) who found that oxygen consumption in liver cells decrease by treatment with Aluminum chloride which in turn interference with histological changes that leads to necrosis and fatty changes in liver cells. Also the fatty change probably occurs due to disturbance in liver metabolism of some material and lipid which leads to fat deposition. The vaculation of hepatocytes was related to fatty changes, which occur due to effect of endotoxin on hepatocytes (15). Necrosis of hepatic cells which showed in many tissue sections may be due to partial occlusion of blood vessels by several inflammatory reactions and may account for an ischemic effect on hepatic cells (16). The kidneys are responsible for elimination of drugs and metabolites, these organs are also capable to realize diffused biotransformation reactions. Many studies demonstrate that nephrotoxicity induced by chemical agents are one of the consequences of the accumulation of certain metabolites in kidney damage which is one of the most prominent reasons of death due to intoxication (17). The present lesions of nephrotoxicosis in the kidneys were commonly consisted of tubular changes of epithelial vacuolar degeneration in addition to intertubular mononuclear cell infiltrations. These findings are in agreement with (18) and this probably due to renal insufficiency which occurred due to the toxic effect of Aluminum on the kidney (19) the insufficiency resulting in decrease excretion process and that lead to accumulation of toxin within the cortical tubules. That was similar to the result of researches (20) which found in high concentrations is readily released into the urine in the renal tubular injury oxidative stress can promote the formation of a variety of vasoactive that can affect renal function directly by initiating renal vasoconstriction or decreasing the glomerular capillary ultrafiltration coefficient and thus reducing glomerular filtration rate. Ninety percent of the particles taken orally transferred to circulation it accumulates in the body. Aluminum accumulates in the soft tissues it is distributed to liver, kidneys, spleen, and muscle. Kidneys excrete 75% of the daily intake without altering it some accumulates in nonviable tissues such as nail and hair (21). Spleen in the present work appeared with changes of

depletion of the lymphocytes with dispersion of the lymph follicles as well as congestion of the capillary sinuses in the red pulp, less is known about the influence of Aluminum chloride toxicity on Lymphatic and hematopoietic organs such as spleen, thymus for identifying Immunotoxicity, the results of other authors who observed hyperplasia of Lymphoid tissue following exposure to certain toxic substances (22 and 23) by indirect effect on the structure of the spleen via disturbing the function of nervous system, the toxin is highly lipophilic which primary effects central nervous system (CNS) or that effect may produced as antioxidant effect reflect on the histopathology of the spleen (24) deposition of haemosiderin in the spleen cells increases, this is probably due to increased levels of destruction of red blood cells in the pulp (25) spleen tissue showed lymphocytic depletion and multiple focal necrosis (26) suggested that Aluminum chloride reduced population of lymphocyte and macrophage in the spleen and its accumulation in the infected area, these changes in the spleen are similar to the results reported by other authors (27) who have noted increasing proliferation of lymphoid tissue in the organs of the immune system under conditions of stress factors and that related with hematological and immunosuppressive effects. In conclusion, the results of the present study indicate that chronic used of Aluminum chloride results changes in the histological structure of liver, kidney and spleen.

References

1. Becaria, A.; Campbell, A. and Bondy, S.C. (2002). Aluminium as a toxicant. Toxicol. Ind. Health, 18:309-320.
2. Yokel, R.A. and McNamara, P.J. (2001). Aluminum toxicokinetics: An updated minireview. Pharmacol.Toxicol., 88:159-167.
3. Buraimoh, A.A.; Ojo, S.A.; Hambolu, J.O.; and Adebisi, S.S. (2011). Behavioural endpoints of adult wistar rats, following aluminium chloride exposure. Brit. J. Pharmacol. Toxicol., 2(5):273-276.
4. Yokel, R.A. (2002). Brain uptake, retention, and efflux of aluminium and manganese. Environ. Health Perspect., 110:699-704.
5. Yousef, M. I. (2004). Aluminum-induced changes in hemato-biochemical parameters, lipid peroxidation and enzyme activities of male rabbits: protective role of ascorbic acid. Toxicol., 199:47-57.
6. Hafez, E.S.E. (1970). Reproduction and breeding techniques for laboratory animals. Lea and Febiges Philadelphia. 55:156-178.
7. Scott, M. (2000). Aluminum compounds. Review of toxicological literatures abridged final report. National Institute Integrated Laboratory System, Research Triangle Park, North Carolina, USA. 121:55-59.
8. Suvarna, S.K.L.; Christopher, L.D. and Bancroft, J.D. (2013). Bancroft theory and practice of histological techniques. 7 Ed, Churchill livingstone, Newyork, USA, Pp:109-121.
9. WHO. (2007). Aluminium from all sources, including food additives. In: Safety evaluation of certain food additives and contaminants. Geneva, World Health Organization, Pp:110-208. (WHO Food Additives Series, No. 58.
10. Buraimoh, A.A.; Ojo, S.A.; Hambolu, J.O. and Adebisi, S.S. (2012). Effects of aluminium chloride exposure on the histology of the liver of adult wistar rats. IOSR J. Pharmacy. 2(3):525-533.
11. Bogdanovi, M.; Begi, A.J. and Bulat, P. (2008). Aluminum hepatotoxicity in rats. Arh. Hig. Rada. Toksikol., 59:97-101.
12. Abubakar, M.G.; Taylor, A. and Ferns, G.A. (2003). Aluminum administration is associated with enhanced hepatic oxidant stress that may be offset by dietary vitamin E in the rat. Int. J. Exp. Pathol., 84(1):49-54.
13. Alessandra, S.; Antonio, L.; Matteo, F.; Giorgio, S.; Rossella, B.; Rita, R. and Luigi, F.R. (2008). Effects of aluminium sulphate in the mouse liver: Similarities to the aging process. Experimental Gerontology, 43(4): 330-338.
14. Gonzales, M.; Alvarez, M. L.; Pisani, G.; Bernal, C.; Roma, M. and Carrillo, M. (2007). Involvement of oxidative stress in the impairment in biliary secretory function induced by intraperitoneal administration of aluminium to rats. Biol. Trace Elem. Res., 116:329–342.
15. Gagliano, N.; Grizzi, F. and Annoni, G. (2007). Mechanism of aging and liver functions. Digest. Dis. Sci., 25:118-123.

16. Walton, J.R. and Wang, M.X. (2009). APP expression, distribution and accumulation are altered by aluminum in a rodent model for Alzheimer's disease. J. Inorg. Biochem., 103(11):1548-1554.
17. Buraimoh, A. and Ojo, S.A. (2012). Effects of aluminium chloride exposure on the histology of the kidney of wistar rats. Int. J. Bio. Pharm. Allied Sci., 1(11):1556-1568.
18. Stella, M.M.; Ne'stor, G.; Marcela, C.; Maria, D. and Maria, E. (2005). Alterations of the renal function and oxidative stress in renal tissue from rats chronically treated with aluminium during the initial phase of hepatic regeneration. J. inorganic. Biochem., 99: 1858-1864.
19. Belaïd-Nouira, Y.; Bakhta, H.; Haouas, Z.; Flehi-Slim, I. and Ben Cheikh, H. (2013). Fenugreek seeds reduce aluminum toxicity associated with renal failure in rats. Nutr. Res. Pract., 7:466-474.
20. Roy, A.K.; Taloukder, G. and Sharma, A. (1991). Similar effects in vivo of two aluminium salts on the liver, kidney, bone and brain of Ratus norvegicus. Bull. Environ. Contam. Toxicol., 47:288-295.
21. Becaria, A.; Campbell, A. and Bondy, S.C. (2002). Aluminium as a toxicant. Toxicol. Ind. Health, 18:309-320.
22. Blaylock, R.L. and Maroon, J. (2011). Immunoxcitotoxicity as a central mechanism in chronic traumatic encephalopathy-a unifying hypothesis. Surg. Neurol. Int., 2(107):104-180.
23. Platt, B.; Fiddler, G.; Riedel, G. and Henderson, Z. (2001). Aluminium toxicity in the rat brain: histochemical and immunocytochemical evidence. Brain. Res. Bull, 55(2):257-267.
24. El-Demerdash, F. M. (2004). Antioxidant effect of vitamin E and selenium on lipid peroxidation, enzyme activities and biochemical parameters in rats exposed to aluminium. J. Trace Elem. Med. Biol., 18(1):113-121.
25. Geyikoglu, F.; Turkez, H.; Bakir, T.O. and Cicek, M. (2013). The genotoxic, hepatotoxic, nephrotoxic, haematotoxic and histopathological effects in rats after aluminium chronic intoxication. Toxicol. Ind. Health, 29:780-791.
26. Synzynys, B. I.; Sharetskii, A. N. and Kharlamova, O.V. (2004). Immunotoxicity of aluminum chloride. Gig Sanit 4:70-72.
27. Zhu,Y.Z.; Zhao, Li.; XW, H.S.; Zhang, L.C.; Hu, C.W.; Shao, B.; Sun, H.; Bah, A.A. and Zhang, Z.G. (2011). Effects of subchronic aluminum exposure on the immune function of erythrocytes in rats. Biol. Trace Elem. Res., 143:1576-1580.

دراسة التغيرات النسجية للكبد والكلى والطحال في الجرذان المختبرية البيضاء بعد تجريعها بمادة كلوريد الألمنيوم

هدف هاشم محمد[١] و ابتسام خلف عبد علي[٢] و علي فارس رشك[١]

[١] فرع التشريح والأنسجة، كلية الطب البيطري، [٢] قسم العلوم الأساسية، كلية التمريض، جامعة بغداد، العراق.

E-mail: hadafs@yahoo.com

الخلاصة

أجريت الدراسة الحالية لغرض تقييم التغيرات النسجية الناجمة عن تأثير تجريع كلوريد الالمنيوم حيث استخدمت في هذه التجربة اربعون من الجرذان المختبرية البيضاء قسمت على مجموعتين، المجموعة الأولى جرعت عن طريق الفم بجرعة ٣٧ ملغم\كغم من وزن الجسم أما المجموعة الثانية فكانت مجموعة السيطرة وأعطيت الماء المقطر فقط خلال مدة التجربة والتي استمرت ٦٠ يوما وبعد انتهاء فترة التجربة قُتِلت الحيوانات وأخذ كبده والكلى والطحال وبعد أن جُمعت العينات وأجريت الفحوصات النسجية عليها تبين وجود التنخر والتنكس وتحلل الخلايا واحتقانها مما يدل على وجود التأثير السمي التراكمي لكلوريد الألمنيوم على الكبد والكلى والطحال.

الكلمات المفتاحية: كلوريد الألمنيوم، الكبد، الكلى، الطحال، الجرذان المختبرية البيضاء.