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EFFECT OF HYDROCORTISONE IN SOME FEMALE WHITE LABORATORY MICE ORGANS

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ABSTRACT

The current study evaluated the resulting pathological tissue in some mice When injected with a dose of the hormone hydrocortisone Mus musculus L. Laboratory. All groups included 8 mice, the first group gave the saline solution, As a control group, the second group was injected with hydrocortisone with a dose of 0.21mg / kg. The histological examination of liver and kidney sections showed dosedependent pathological changes when compared with a group Of some liver cells and blood necrosis. Necrosis and degeneration, and the liver was the occurrence of degeneration. In the portal and central veins a as well as the presence of hemorrhage Expansions and Congestion expansion. Inflammatory cells in different parts of the body.

Infiltration changes in the papillary area and hemorrhage. Sloughing In the kidney, some of the tubing of the lining of the protrusions have been degraded while other cells have been observed Some cells lining the protrusions as well as congestion, bleeding and inflammation of inflammatory cells in the capsules.

KEYWORD: Hydrocortisone, Histopathology, Liver, Kidney, Glucocorticoids.

INTRODUCTION

Glucocorticoids (Gcs) are produced by the adrenal cortex and affect the organs of the body, which play an important role in regulating stress. Hormones levels are regulated by the hypothalamus, pituitary gland and adrenal gland, which are affected by several factors including stress, feedback, delayed supply and contains receptors under hypothalamus and pituitary gland.^[1] These hormones are associated with cytothelial receptors in the target cells and are affected by increased mRNA. These receptors are located on cell surfaces or within the cytoplasm.^[2] Steroid hormones pass through the plasma membrane by spreading through the lipid layer and linked to the cytoplasm receptors to form the hormone complex receptor, this complex enters the nucleus of the cell and stimulates mRNA production of protein-building. These receptors called nuclear receptors and associated with special transcription factors.^[3] Previous study reported that, Daxamethasone had an effect on bone marrow rabbits after injection of 1.56 mg / kg and another group were injected with a dose of 3.12 mg / kg for two weeks, histological examination of the planktonic teeth and a statistical analysis of the number of osteoporotic cells showed a steady increase in the number of cells of osteoporosis and its increased numbers that indicate osteoarthritis.^[4] Other study showed effect of cortical hormones on the growth and mineralization of bone and these hormones lead to modify the growth line and pointed out that the cortical glycoproteins have an effect on the structural system and produced effects on bone metabolism, direct action on bone cells and the number of bone cells.^[5]

MATERIALS AND METHODS

Hormone

Cortisol hormone is in the form of a small glass container of 2 ml capacity, containing 100 mg of hydrocortisone and added sodium succnate, The Supplied Supplied by A.D. Hemopharm, FARC, Sardia. The dose preparation was 0.21mg/kg dissolved in a normal saline solution of 0.9%.

Experimental Design

Female Mus Musculus mice (n=12), 10-12- week-old, weighting 22-25g were utilized in this study and divided into two groups control and treat group (per group= 6). Animal housed in a communal cage under controlled conditions interims of temperature of 20- 25 C and provided with water and food ad libitum, Cycle of Lighting 12/12 hours light and dark throughout one year. Efforts were made to avoid any unnecessary distress to the animals. further, Animal raised in the animal house at the Department of Life Sciences /Faculty of Education /university of Basra- Iraq. Control group injected with 0.1 ml of the phosphate solution, whereas treat group were injected with 0.21mg/ kg of corticosteroids (hydrocortisone). In this study, Humasan^[6] method utilized in the preparation of histological sections of liver, kidney.

RESULTS

Liver

The liver is made up of six-shaped lobes and central vein concentrated in each lobe. The vein is composed of cells called hepatic cells, which contain one nucleus, arranged in a bar shape in radial system (1A,B). The study of the histological sections of the female mice showed satisfactory changes such as Kargonacresis, and the necrosis of some cells (figure 1,C), the effect of slight infiltration in some liver regions, density of other areas (Figure 2B), degeneration of hepatic cells (figure1,E), The hypertrophy of the cells and change the forms of the nucleus and take irregular forms (figure 1F), hemorrhage (Figure1 D), Congestion of spaces between cells (figure 2A), inflammation of inflammatory cells and expansion of portal vein (figure2 B), congestion of the sinuses (figure 2 C), pubic veins(figure 2D), hypertrophy of hepatic cell nuclei(figure2E), clarification of Eto cells (figure3A) and.

Kidney

The results of the tissue examination showed that the kidney was composed of a network of blood vessels and associated with epithelial cells formed a synthesis called Glomerulus. Glomerulone is surrounded by the Bowman capsule, inside lined via capillaries and the outer layer forming the outer surface (figure 3B). Our study showed an effect of corticosteroids on the renal tissue compared to the control group. The effects were swelling of some of the cells lining the distant tubes (Figure 3C), inflammation of the inflammatory cells around the blood vessels (Figure 3D), hyperplasia of the lining Proximal twisted tubules and hypertrophy of their cells and renal glomerular congestion (Figure 3E).

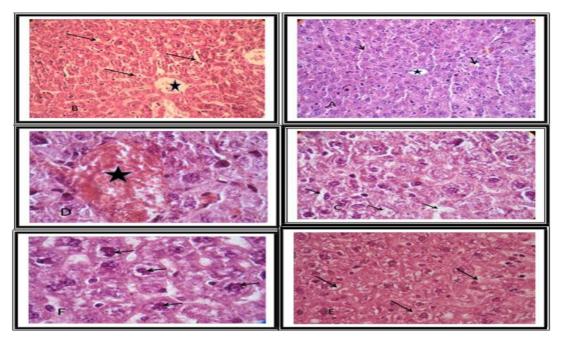
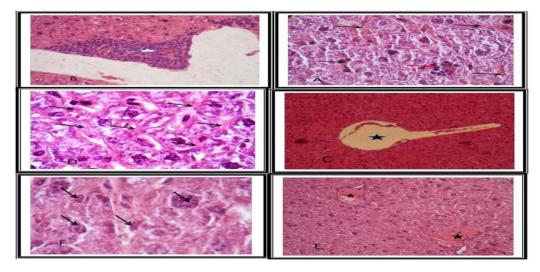
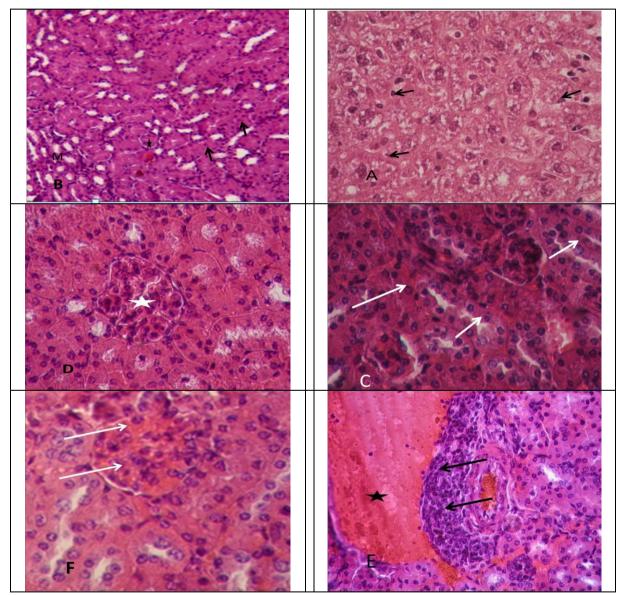


Figure (1) A- A section of the liver from the control group showing the structure of the liver shows the central vein (aster) and the sinuses (arrows), H & E, 10X. B- A section in the liver of mice from the control group shows the sinuses (arrows), central vein (star), H & E, 400X. C- A section in the liver of the mice from the treatment group showing the necrosis of some cells (arrows). H & E, 400X. D- A section in the liver of mice from the treatment group shows hemorrhage between cells (star), H & E, 1000X, E- A section in the liver of Mice from the treatment group showing hepatocycetes degeneration, H & E, 400 X, F. A section in the liver from the treatment group indicates the hypertrophy of the cells, H & E, 1000X.



Figure(2)A-) A section in the liver of mice from the treatment group shows inflammation and congestion of sinusoidal, H & E, 400X, B- A section in the liver of mice from the treatment group shows inflammation of the liver in the hepatic areas

(star), H & E, 400X, C- A section in the liver of mice from the treatment group showing the expansion of the portal vein (star) H & E, 100X, D- A section in the liver of the Mice of the treatment group showing hemorrhage of the hepatic sinuses (arrows), H&E, 400X. E- A section in the liver of Mice from the treatment group shows the congestion of the venous veins (stars), H&E, 100X, F- A section in the liver of Mice from the treatment group showing variability of nucleus forms and taking unstructured forms (arrows), H & E, 1000X.



Figure(3) A- A section in the liver of mice from the treatment group shows Eto'o cells (arrow) B- The kidneys of the control group show the renal tubules (arrow), the renal glomerulus (the star) and the pulp region = M, H&E, 100X, C- A kidney from the treatment group shows the bulging of some of the cells of the distant tubules, H&E, 1000X, D- A kidney of the treatment group indicates hyperplasia of renal glomerular

cells (star), H&E, 400X E- A kidney from the treatment group demonstrates inflammatory cell infiltration around blood vessels (blood vessels) and blood vessel congestion (star), H&E, 400X, F- The kidney of the treatment group demonstrates hyperplasia of the cells and congestion of renal Glomerulus (arrows), H&E, 1000X.

DISCUSSION

Our investigated of histological sections of liver, kidney organs in female mice showed that tissue changes were significantly different from those of control group. liver plays a key role in removing the toxins of the foreign substances entering the body, further, the liver injury is very dangerous.^[7], The results of the present study showed that a emergence of changes in the tissue, including the expansion of sinuses and congestion, the expansion of hepatic sinuses, inflammatory cell infiltration, hypertrophy of hepatic cell nuclei, changed forms, taking irregular forms, moreover, has been observed relapse in some sections. The findings of the present study were consistent with^[8,9], which demonstrated the pathogenic of liver effects on rats treated of Hydrocortisone.

A study of Eken et al.^[8] reported that the rats injected with dexamethasone and a dose of 0.12 mg / kg daily lead pathological changes in the liver, including inflammation of the inflammatory cells, expansion of sinuses, hypertrophy of hepatic cells and relapse of cells and referred that the changes are caused by the over-fat oxidation.

Zhang et al.^[10] showed an injection of 0.25 mL / 100g of dexamethasone caused damage to the liver tissue and observed hepatic hyperplasia and spherical shape, the emergence of infections, widening of sinuses, necrosis of the liver cells and inflammation of the inflammatory cells.

Chronic exposure to hydrocortisone leads to liver damage due to bleomycin inhibition, which leads to fibrobrosis.^[11] Dexamethasone is a cytokine inhibitor that affects hepatic cell reproduction.^[12] Changes in cells at the level of cytoplasm and nucleus,represented by chromatin decomposition or disintegration of the nucleus, change its forms and take irregular forms and disintegration seems to be an initial step of apoptosis.^[13,14]

Additionally, the histological study showed relapse in hepatic cells and the emergence of gaps in the cytoplasm due to the accumulation of water and ions in the cytoplasm which passes easily through the plasma membrane, may be associated with an imbalance in

metabolism that leads to accumulation of fat or glycogen appears in gaps in tissue sections.^[15] The effects of relapse may be because of the effect of oxidative stress which resultant from free radicals due the hormone hydrocortisone, which interact with unsaturated fatty acids, which affects membrane permeability and increase its permeability, which facilitates the entry of substances into cells and deformation of cellular components.^[7]

In line with this, blood vessel congestion is caused by excessive blood in the veins, thus increasing blood pressure in the veins and capillaries^[16] and accompanied by decreased blood supply and waste accumulation.^[17] Thus, inflammatory response and protein-rich secretions, which increase blood viscosity, decrease blood flow, stagnation and congestion, occur due to lack of normal oxygen and nutrient processing.^[18] The appearance of inflammatory cell infiltration in the liver is considered as defensive line of the body to controlling The causative agent of disease, moreover, using of glucocorticoids leads to the migration of white blood cells to the inflammation area through the walls of blood vessels due to the contraction of endothelial cells or due to the loss of the Desmosomes.^[19]

Previous study showed that hemorrhage in some studied sections due to inflammation of the tissues as a result of exposure to the hormone or metabolism or due to damage that occurs in the walls of blood vessels.^[20]

The kidney is the most sensitive organ after the liver against the toxic damage caused by hyperhidrosis or drugs and plays a major role in the metabolism and removal of foreign substances from the body.^[21] In addition, our study demonstrated histopathological changes seen in kidney tissues is a bulge of some lining cells of distant tubules, leak of the inflammatory cells, hyperplasia in the lining of the nearby twisted tubules and the congestion of glomerulosclerosis.

The results of the current study differed with.^[20] and confirmed that there was no effect of glycoproteins on kidney. our study showed a puffiness in the lining cells of the kidneys that were due the toxic substances inhibit sugary decomposition and oxidative phosphorylation processes which lead to a decrease in ATP production and consequently lead to the failure of the work of sodium and potassium pumps and the water and sodium flow easily into the cells and the potassium Causes mitochondria to bloom.^[19]

Increased cell size is associated with an increase in mitochondrial volume or resulting in the clustering of these cells.^[18] Injections of the hormone caused a disturbance of Bowman's portfolio such as bloody congestion and Inflammation of inflammatory cells^[19] while the histological changes caused by corticosteroids in the lung tissue were represented in Inflammation of inflammatory cells, hyperplasia of the occipital epithelium of the aortic vesicles and congestion of blood vessels. Hyperplasia, which is observed in some organs, is consider as a form of inflammation caused by the effect of overgrowth of the injected material on cell reproduction, which increases the size of the organ, and may be a hormonal cause or the presence of endocrine glands.^[19] Hydrocortisone stimulates cell differentiation and differentiation processes.

REFERENCES

- 1. Vegloulos, A. and Herzig, S. (2007). Glucocorticoids Metabolism and Metabolic diseases, Molecular and cellular, Endocrinology, 275(1-2): 1-52.
- 2. Pocock, G and Richards C.D. (2006). Human Physiology the basis of the Medicine Br. J. Sports Med., 40(10): 880-883.
- 3. Kulle, A.; Krone, N.; Holterhus, D.M.; schuler, G.; Greaves, R.F.; Junl, A; Rijke, Y; Hartmann, M. F.; et al. (2017). Steroid hormone analysis diagnosis and treatment of DSD: Position Paper of EV cost Action BM 1303 DsDnet, Eur. J. Endocrinol., 176(5): 1-9.
- 4. Yasear, A. Y. and Hamouda, S. A. (2009). Effect of dexamethasone on Osteoclast Formation in the alveolar bone of Rabbits, Iraqi, Journal of Veterinary Sciences, 23/(1): 13-16.
- 5. Donatti, T. L.; Koch, V. H. K.; Takayama, L. and Pereira, R.M.R. (2011). Effects mineralization, Journal de Pediatria, 87(1): 4-12.
- 6. Humason, G.L. (1972). Animal tissue techniques, 3rd edition, with Freeman and company Heikal, T. M.; El-sherbing, M.; Hassan, S.A.; Arafa, A. and Ghanem, H. (2012). Antioxidant effect of Selenium on hepatotoxicity induced by chlorpyrifos in male rat, Int. J. Pharm. Sci., 4(4): 603-609.
- 7. Eken, H.; Ozturk, H. Ozturk, H. and Buyukbayram, H. (2006). Dose related effects of due tobile duct ligation in rat, World, J. of Gastroenterology, 7(12): 5379-5383.
- 8. Shama, S. and Lichtentein, A. (2008). Dexamethasone induced apopotosis mechanisms is myeloma cells investigated by analysis of mutant glucocorticoid receptors., Blood, 112(4): 1338-1345.
- 9. Zhang, X.P.; Zhang, L.; Chen, L.; Wang, J.; Cai, W.; Shen, H. and Cai, J. (2007).

- Influence of dexamethasone mediators and NF-kB expression in multiple Organs of rat with Severe acute pancreatilis, Word J. Gastroenterol, 28(4): 548-556.
- 10. Dik W.A., McAnulty R.J., Versnel M.A., Naber B.A., Zimmermann L.J., Laurent G.J., Mutsaers S.E. (2003). Short course dexamethasone treatment following injury inhibits bleomycin induced fibrosis in rats. Thorax, 58: 765-771.
- 11. Naoy, P.; Kiss, A.; Schhnur, J.; Thorgersson, S.S. (1998). Dexamethason inhibits the Proliferation of Hepatocytes and Oval cells But Not Bile Duct cells in Rat Liver, Hepathology, 28(2): 423-430.
- 12. Zhang, X.P.; Zhang, L.; Wang, L.; Cheng, Q.H.; Wang J.M.; Caj, W.; Shen, H.P. and Cai, J. (2007). Study of the protective effect of Dexamethasone on Mutliple Organ Injury in Rats with Severe Acute Pancreatilis, Jop. J. Pancreas, 8(4): 400-412.
- 13. Hartmann, K.; Koenen, M.; Schauer, S.; Witting Blaich, S.; Ahmed,; M.; Bashant, V. and Tuchermann, J.P. (2016). Molecular actions of Glucocorticoids in Cartilage and bone during Health disease and steroid Therapy, Phsiol. Rer., 96(1): 409-447.
- 14. Gores, G.J. Herman, B. and Lemasters, J.J. (1990). Plasma membrane bleb formation and rupture: a common feature of hepatocellular injury Hepatology, 11(4): 690-698.
- 15. Rady, M., I. (1990). Effects of exposure to diazinon on the Lung and small intestine of guinea Pig, Histological and some histochemical changes, Braz., Arch. Biol. Thechnol., 52(2): 317-326.
- 16. Haschek, W.M. and Rousseaux, C.G.(1991). Hand book of Lexicologocal Pathology, 13th ed, Academic Press. London and Newyork.
- 17. Herrington, C.S. (2014). Muir's textbook of Pathology 15th ed CRC Press. Taylor and Francis :PP589.
- 18. Mas sween, R. and Whaley, K. (1992). Maurixtex book of Pathology, 13th rd, Edward Arnold London PP1245.
- 19. Yavuz, T.; Delihas, N.; Yildirim, B.; Altuntas, I.; Candir, O.; Cora, A.; Karahan, N.; Ibrisim, E. and Kutsal, A. (2004). Vascular Wall damage in rat induced drug effects of vitamins E and C. Arch. Toxical., 78(11): 655-659.
- 20. Sharma, D. and Sangha, G. K. (2014). Triazophos induced Oxidactive stress and histomorphological Changes in Liver and Kidney of female albino rat Pest. Biochem. Physiol., 110: 71-80.