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Effect of heat stress on histopathological alterations in kidneys of albino rats

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Abstract

The effect of heat stress was studied over two months (July and August) by using thirty adult male rats. The animals were divided into six groups (five animals per each group) and tested for 7, 14, 21, 28, 35 and 42 days, under controlled condition (45±5°C). The clinical observation indicated significant decrease in activity and body weight associated with oligourea and hypophagia. All these signs were prominent after five days of the experiment. The kidneys of rats under heat stress showed degenerated glomeruli began at 7th day of the study and widening of the capsular space. Atrophy of some glomeruli was also noticed. With prolong exposure to heat changes in the proximal and distal convoluted tubules were prominent when compared with normal rats' kidneys.

Key words: Heat stress, kidney changes, glomerular degeneration, Albino rats

Introduction

Stress as defined by Lee (1965) "denotes the magnitude of forces external to the body system which tend to displace that system from its resting g or ground state ". Scott (1981) added that this definition rather then putting the accent on unfavourable ambient conditions emphasize the forces that bring about changes in the body system. Morrow (2009) defined stress as the body's reaction to change that requires a physical, mental or emotional adjustment or response. Sel (2010) noted in numerous experiments that laboratory animal showed sub to acute changes including physical, emotional and pathological changes such as stomach ulceration, shrinkage of lymphoid tissue and adrenal gland enlargement. He later demonstrated that persistent stress could cause these animals develop various diseases similar to those seen in humans, such as heart attack, stroke and kidney diseases. Studies over years have proposed variety effects of stress on body biological systems (Maroni et al., 2003). In human being, Chia and Teo (2003) suggested that heat stress and associated hyperthermia can be presented as a spectrum of symptoms ranging from the headache and malaise to the harshest form of heat related injuries and many other abnormalities. Wang et al. (2009) found pathological changes in heart and liver of rats under hyperthermic conditions. Kumar et al. (2010) concluded that acute stress affects atrial and ventricular depolarization and repolarization in albino rats. Kidneys according to Foulkes and Hammond (1975) and Hook (1980) is a major site affected by toxic environmental pollutants, and since ambient temperature may be involve in environmental pollutant if it exceed the tolerated value.

In the present study, an animal model was created to find heat exposure (45°C) on albino rats for 42 days continuously on histological changes in the tissue of kidney.

Materials and Methods

Thirty adult male albino rats weighing 200±10gm were obtained from Sammara drug factory, Iraq. These rats were maintained and acclimatized in the College of Veterinary Medicine, Tikrit University under laboratory condition in July and August 2009 where the ambient temperature was 45±5°C. The rats were fed standard diet pellets and water *ad labium*. The rats were divided into 6 groups, 5 animals in each group. Group A survived for 7 days, group B survived for 14 days, group C survived for 21 days, group D survived for 28 days, group E survived for 35 days and group F survived for 42 days. During this study, two rats were died, one each from group A and B, while 2 rats diet each from C and D. In group F, 3 rats were died.

At the end of survived period of each group, the rats were killed under intensive dose of chloroform. Both kidneys from each rat were rapidly removed and micro dissected to obtain tissue samples of the cortex and medulla. The specimens were rinsed with normal saline and fixed in 10% formalin, dehydrated through graded alcohol serried (50–100%) then cleared in xylene and embedded in paraffin wax. Six micrometers

thick section were made by microtome and stained with routine stain (H &E) according to the method described by Luna (1968).

Photomicrographs of stained slide were taken using light microscope attached to digital camera. The whole photomicrographs were compared with kidney tissues of normal healthy rats prepared before the experiment.

Results

During the periods of the study clinical observations showed that the activities of the animals were significantly decreased by day 2 of exposure indicating the onset of heat response. After 5 days urine began to decrease indicating features of oligouria and hypophgia with decrease in body weight. Nervous and muscular systems dysfunction such as convulsion and coma were noticed. Death of some animals was recorded as mentioned before in the materials and methods.

Histological study of normal tissues of kidney for the non-stress rats revealed normal glomerulus is surrounded by the Bowman capsule and the proximal and distal convoluted tubules were demonstrated with out any inflammatory or pathological changes (Fig. 1). The histological kidneys picture of rats under heat stress showed degenerated glomeruli, appeared in the first group (A), killed at the day 7^{th} of the study is associated with widening of the capsular space and atrophy of the glomerulus (Fig. 2).

The group B showed hypertrophic changes in the epithelium of proximal and distal convoluted tubules (Fig 3). The group lasted up to 21 days of the experiment (C) showed edematous changes with deposition of necrotic debris or cells particles in the lumen of proximal and distal convoluted tubules (Fig 4). Hemorrhage and blood congestion plus necrotic cells were generally observed in the collecting tubules of the medulla of group (D) which lasted up to 28 days under the stress. The other two groups (E and F) showed desquamation of the tubular epithelium and generalized tissue degeneration.

Discussion

Kidneys are key organs affected by heat stress (Volokh, 2006). In the present study results has been speculated that heat stress is associated with deleterious changes on renal corpuscles, proximal and distal convoluted tubules of the renal cortex. Changes demonstrated in the medullary collecting tubules suggesting the distortion and disruption of the histological architecture of the kidney observed in this experiment. Other clinical symptoms demonstrated in this experiment such as oligouria, hypophgia, convulsion and weight loss are associated with direct and continuous contact with environmental temperature (Gaffen and Hubbard, 2001; Abderrezak et al., 2002; Rodrigus et al., 2003; Rajiv et al., 2010). The histological abnormalities observed in this study was

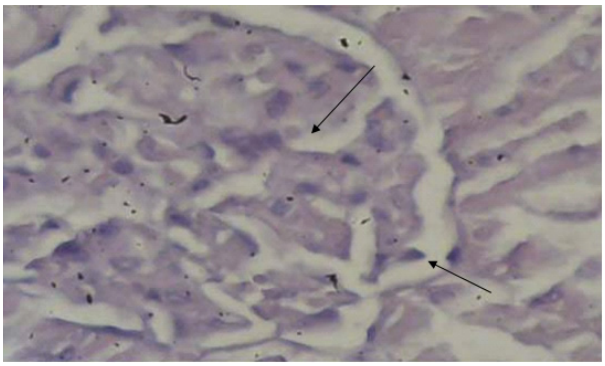

Fig. 1: The glomerulus of the renal corpuscle of non stressed rat (H & E X 400)

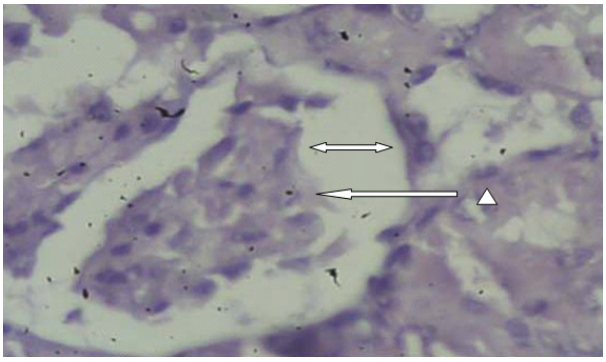

Fig. 2: The glomerulus of the stress rat (group B), Atrophy of glomerulus (arrow head) and widening of Bowman's space shown by arrows. (H & E X 400)

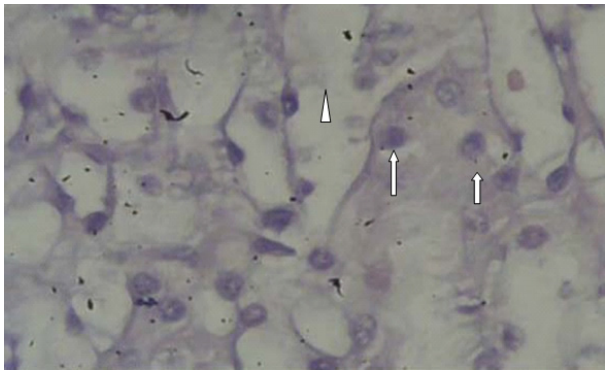

Fig. 3: Hypertrophy of the epithelial cells of the proximal (arrow) and distal convoluted tubule shown by arrow head (group C). (H & E X 400)

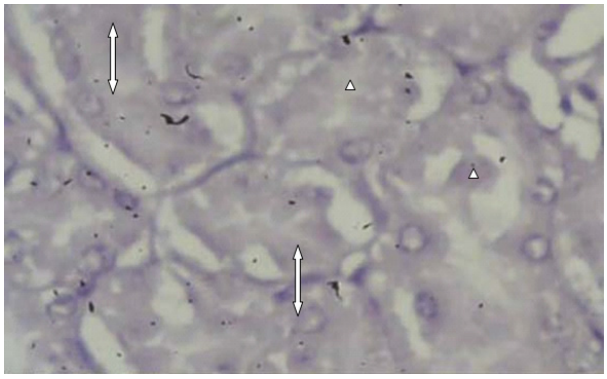

Fig. 4: Edematous change with deposition of necrotic debris or cells in the lumen of proximal (arrow head) and distal convoluted tubules shown by double arrows (group D). (H & E X 400).

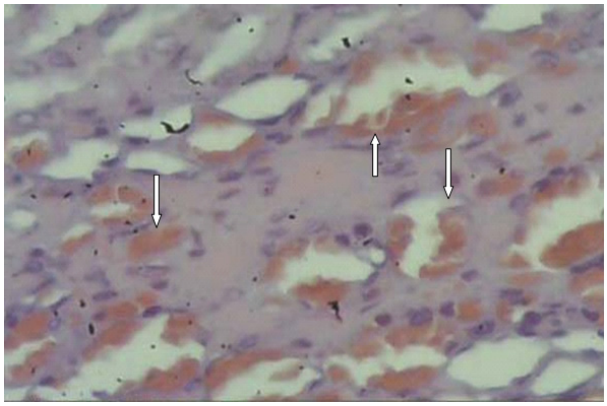

Fig. 5: The hemorrhage of the collecting tubules in the medulla of kidney shown by arrows (group E). (H & E X 400).

similar to previous studies reported by Saunders et al. (1983), who stated that the stress interferes with Na/H and Cl/HCO3 ions exchange thus inhibiting Na and Cl ions uptake and consequently affecting the contractile elements of the cardiac muscle and the rats die of heart failure, where Gaffin and Hubbard (2001) mentioned that in hyperthermia K ion released from cells leading to reduce renal blood flow and reduce renal filtration and urine flow. Kumar et al. (2010) stated that acute stress affect arterial and ventricular depolarization–repolarization.

Yang and Lin (2002) stated that heat stress induces free radicals, leading to oxidative damage in the organs such as liver, heart, brain and the kidney. The implication is that the arbitrary chronic or excessive heat stress affect the health status of animals, the importance of this concept lies in the potential adverse effect on histological pattern of the tissues. Hook (1980) suggested that kidney is the major site for toxic effects of environmental factors since it is involved in the reabsorption of water in the distal tubules relatively with high concentration of toxic metabolites or pollutants which affect the renal cells.

In the present study pathological changes demonstrated in the kidney of animals under heat stress may be attributed to changes occur in the homeostasis and increase production and elimination of toxic metabolites.

References

Abderrezak, B., James, P. and Knochel, M. 2002. Heat stroke. *New England Journal of Medicine*, 46: 1978–1988.

Chia, S.E. and Teo, K..J. 2003. Prognosis of adult men with heat exhaustion with regard to postural stability and neuro behavioural effect “A 6 months follow up study “ *Neurotoxicology and teratology*, 25: 503-508.

Foulkes, E.C. and Hammond, P.B. 1975. Toxicology of kidney .Toxicology the basic science of poisons. NY, MacMillan. Pp: 190-200.

Gaffin, S. and Hubbard, R.W. 2001. Pathophysiology of heat stroke. In: Pandoll K.B. Burr R. (Ed.) Medical aspect of harsh environment volume, 1 Washington DC, Borden Institute TMM publications.

Hook, J.B. 1980. Toxic response of the kidney. Toxicology. The basic science of poisons. NY, MacMillan. Pp: 232–245.

Kumar, R., Kela, A.K. and Tayal, G. 2010. Effect of acute stress on rat ECG. *International Journal of Pharmacology*, 18 (1):23-34.

Lee, D.H.R. 1965. Climatic stress indices for domestic animals. *International Journal of Biometrology*, 9: 29. Cited from Scott (1981).

Luna, L.G. 1968. Manual of histological staining methods. The armed forces institute of pathology 3rd .ed Mc Graw Hill Book Company.

Maroni, P., Bendinelli P., Tiberio, L., Rovetta, F., Piccletti, R. and Schiaffonati, L. 2003. Heat–shock response in brain: Signalling pathway and transcription factor activation. *Molecular Brain Research*, 119(1): 90-99.

Maritim, A.C. Sanders, R.A. and Watkins, J.B. 2003. Diabetes oxidative stress and antioxidant. A review. *Journal of Biochemistry and Molecular Toxicology*, 17(1): 24 -37.

Morrow, A. 2009. Stress definition. http://dying.about.com/od/glossary/g/stress.htm

Rodrigus, L.O.C., Lima, O.A. and Moreira, C.A. 2003. Fatique was independently affected by increase exercise and thermal environmental stress. *Brazilian Journal of Medicine and biological research*, 36 131–135.

Saunder, R.L. Handerson, E.B. and Harmon, P.R. 1983. Effect of law environmental pH on smolting

of Atlantic salmon. *Canadian Journal of Fish aquatic Science*, 40: 1203- 1211.

Scott, G.H. 1981. what is animal stress and how is it measured. *Journal of Animal science*, 52: 150–153.

Teoh, S.L. and Latiff, A. 2010. Histological changes in the kidneys of experimental diabetic rats fed with *Momordica charrantia* (bitter ground) extract. *Romania Journal of Morphology and Embryology*, 51(1): 91-95.

Voloka, K.Y. 2006. Stress in growing soft tissue. *Acta Biomaterica*, 2: 493–504.

Wang, Y., Zhihui, M. and Hebei. Z.B. 2009. Study of the stress changes of heart and liver tissue in rats after myocardial ischema. *Hebei Medical Journal*, 14.

Yang, C.Y. and Lin, M.T. 2002. Oxidative stress in rats with heat stroke–induced cerebral ischemia. *Stroke*, 33:790-794.