Histological Changes in Broiler Chickens Livers and Kidneys Fed Diet with Different Levels of Boron

Sabah S. Al-Tekrity

Department of Physiology, Biochemistry and Pharmacology, College of Veterinary Medicine, Tikrit University, Tikrit, Iraq <u>Vet.sabah@yahoo.com</u>

(Received: 13 / 7 / 2011---- Accepted: 27 / 9 / 2011)

Abstract

Forty, Ross broiler chickens (21 days old) were allocated into four groups, fed diet supplemented either with 0,100,200,or300 mg boron/kg diet, for 30 days. At the end of the experimental period, chickens were slaughtered, livers and kidneys were examined histologically. Results indicated that boron in the doses over 100mg/kg diet caused different kind of histological changes in livers and kidneys. Changes appeared dose-dependent. Changes resulted from this experiment indicated concentration of boron over 100mg/kg diet could be toxic to chickens.

Key words : Boron, chickens liver, chickens kidney .

Introduction

Boron is an essential element for plant [1]. Experiments in animals indicated that boron may indirectly influence minerals metabolism cholicalceferol metabolism, and utilization of several energy substrates, in a way that is beneficial to animals, [1-3] Moreover, results from other experiments indicated that boron affect the composition and or functions of several organic system, including brain, skeleton, bone density, immune system, cardiovascular system, and its effect on mineral metabolism, energy substrate metabolism, and membrane function, [4-5]. In contrast to the beneficial effect of boron typical toxicity symptoms may occur, although boron is well tolerated element as more than 300 mg per kg dry matter produce the first signs of toxicity in poultry,[6] these signs include loss of appetite, body weight and reduction in hatchability of hens eggs. Gametogenesis alteration also noticed as a result of chronic boron toxicity in animals [7].

Liver is the largest gland of the body receiving double blood irrigation from hepatic artery and nutrients rich blood from portal vein [8], and being the first organ in receiving the metabolic substances and nutrients , which make it exposed to toxic substances that are absorbed, degraded, and conjugated, [9] in the meantime kidneys are the main site of metabolic waste products elimination. Numerous studies have been shown that boric acid and borax (boron compounds) are absorbed from gastrointestinal tract as well as inhalated and absorbed from the respiratory tract, as indicated by increasing boron concentration in blood, tissues or urine, or by systemic toxic effect of exposed individuals or laboratory animals, [10] With all the documentary results for boron essentiality in physiological amounts, and its toxicity in human and animals, nevertheless little information exist about its effect on hepatic and kidney tissues. This experiment was performed to elucidate the histopathological changes in liver and kidney tissues of poultry after boron administration in doses over the physiological amounts .

Materials and Methods

This study was conducted in the poultry field of the department of animal resources, College of Agriculture / University of Tikrit, from the period 6th January to 6th February' 2011. Forty Ross broiler 21 days old chickens were allocated randomly into four groups, 10 chicks each (T1, T2, T3, and T4) using batteries system . Each group was given traditional diet (Table 1) supplemented with four different boron concentrations (0, 100 mg/kg, 200 mg/kg and 300 mg/kg) for the groups T1, T2, T3 and T4 respectively. The chicks were slaughtered according to the Islamic method by cutting the jugular veins and the spinal cord, at the end of the last day of the experiment. Kidneys and livers of the animals were rapidly removed and micro dissected to obtain tissue samples for histological examination. Blocks of tissues were immediately fixed in 10% neutral buffered formalin, dehydrated with graded series of ethyl alcohol and embedded in paraffin. Sections of 5 micrometers were cut and processed histologically and stained with eosin and hemotoxylin according to [11] . Photomicrographs of the stained slides were taken using digital camera attached to light microscope. The whole treated specimens were examined and compared with those of control specimens of liver and kidneys slides of control group (T1).

Table 1 Compositions of the poultry diet	
Ingredient	Percentage %
Corn	30
Wheat	10
Barley	20
Soybean	24
Diet Animal Protein	8
Wheat Bran	6
Vitamins and Minerals	2
Calculated Crud Protein	20

Table 1- Compositions of the poultry diet

Results

Liver sections from the control group (T1) of the present study showed normal lobular architecture of the liver tissues, the hepatocytes with normal spherical nuclei in the center of each cell and the sinusoids appeared white color in between the hepatocytes (Photo 1).

Liver sections from group (T2) showed minor degenerative changes in the liver cells distributed in liver tissues associated with mild dilatation of the sinusoids, (Photo 2) while liver sections from group (T3) showed the presence of hemosidrin in the sections (Black dotes) with abnormal distribution of hepatocytes cord in the parenchyma of the liver intermingled with network of sinusoid and hemosidrin granules (Spots) could be recognized easily. (Photo 3) .Sections from group (T4) showed hypertrophy of some of the hepatocytes associated with swelling of the nuclei and the presences of black dotes may reflect a case of hemosidrosis, the hepatocytes in the parenchyma of the liver showed degenerated and/or atrophied and the nuclei are demonstrated different sizes and shapes (spindle, elongated, and swollen). The sinusoid appear dilated (Photo 4&5).

The kidney of the control group (T1)(photo,6 &7) showed normal glomeruli and normal proximal convoluted tubules. The lumens were rarely seen due to the presence of microvilli. The medulla contained well recognized collecting tubules. Group (T2) (photo 8) showed that the glomeruli in the cortex were containing high density of cells which could be lymphatic aggregation, and the capsular space rarely seen, while the proximal convoluted tubules lumens were easily recognized which means lower lumping cells than that found in the control group, and the cytoplasm contained vacuoles. Distal convoluted tubules contained wider lumen than the proximal convoluted tubules, and the cells compressed near the basement membrane. The medulla was occupied by collecting tubules of desquamated lining cells. As the dose of boron increased group (T3) (photo 9) showed aggregation inside the glomeruli and the glomeruli were hypertrophied. Proximal tubules contained vacuoles in the cytoplasm and some cells showed necrosis. Distal convoluted tubules also containing necrotic cells. Amyloidal materials were present in the medulla, and the epithelial cells lining the collecting tubules showed necrosis too. When the dose of boron increased to 300 mg per kg diet histological alterations in the kidney get more severe in that group T4 (photo 10) showed that the glomeruli were packed with cells, atrophied and some of them were displaced peripherally, convoluted tubules appeared containing degeneration of some epithelial cells and even some of cells were displaced from the

basement membrane, and infiltration of lymphocytes in the interstitial tissues were well recognized. Distal convoluted tubules appeared atrophied and their cell became sequamus like cells instead of cuboidal.

Discussion

Boron supplementation in broiler feed was studied by many investigators .

Evidences of boron in trace amount are essential to bone formation and other minerals metabolism were well documented [12-15]. Experiments made in mice by **Bustos-Obregon et al,**[16] demonstrated possible poisonous effect of boron on liver tissues in that the hepatocytes in the perivenous zones of the hepatic lobules are diminished in sizes with nuclei appear heterochromatic and some with marginalization of chromatin. **Gonzalez et al,** [17] mentioned that the cellular death by necrosis causes massive damage and alteration in the cellular homeostasis with decrease adenosine triphosphate (ATP) and loss of the cell membrane integrity and genes expression, which related to glycogen ill-synthesis due to boron toxicity which lead to fibrosis.

Bustos-Obregon, et al [16] concluded that boron in toxic doses increases binucleated cells as an attempt to restore the interrupted equilibrium after elevation of the apoptosis of the hepatocytes. In the mean time there were alteration in the glucose metabolism by considerable diminution of glycogen storage in the liver and boron impact on hepatic connective tissue. Likewise Geyikogglu and Turkez [18] reported that boron toxicity from boric acid in breast muscles of poultry decreased metabolic concentration of glucose and glycogen with mitochondrial damage (cristae dissolution).

Histological alteration resulted from the present experiment in both liver and kidney tissues of broiler chickens, demonstrate variety changes. These changes were in accordance with the magnitude of boron concentration in the diet. These results were in agreement with those results obtained earlier in rats kidneys, [19]. These changes could be attributed to many metabolic disorders. [16, 20].

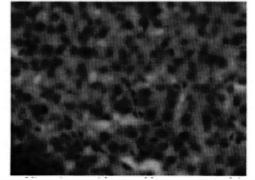


Photo 1: (Group T1) Normal liver tissues with normal hepatocytes, nuclei and sinusoids.(E&H X20)

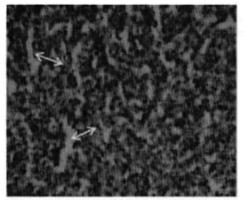


Photo 2: (Group T2) Liver cells with mild degeneration and mild dilatation of the sinusoids E&H X20)

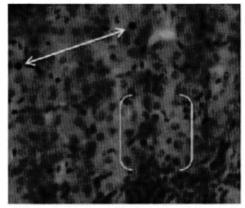


Photo 3: (group T3) Showing abnormal distribution of hepatocytes with the presence of hemosiderin (Black spots) (E&H X20)

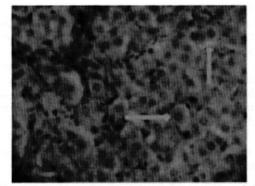


Photo 4:(group T4) Showing hypertrophy of the hepatocytes with swelling nuclei (arrows) and black dotes of E&H X20)

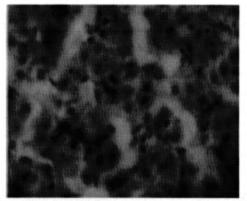


Photo 5: (group T4) showing degeneration of the parenchyma of the liver, atrophied cells, and different sizes and shapes of the nuclei. E&H X40)

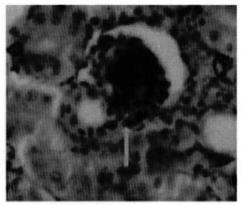


Photo 6: (group T1) showing normal proximal and distal convoluted tubules and the glomeruli appear slightly displaced to urinary pole.(E&H X40)

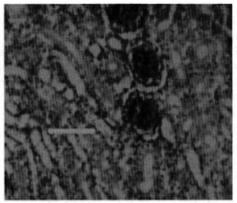


Photo 7: (group T1) kidney section shows distal and proximal convoluted tubules are widely distributed in the cortex and the glomeruli with normal appearance. E&H X20)

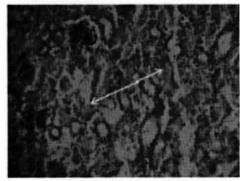


Photo 8: (group T2) showing certain number of proximal and distal convoluted tubules are widen due to sloughing of certain of its epithelial cell lining. (E&H X20)

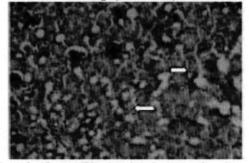


Photo 9: (Group T3) showing degenerative changes of the proximal and distal convoluted tubules .the

References

1- Moseman, R. (1994) Chemical deposition of boron in animal and human. Environmental Health Perspective 102 (7) 113-117.

2- Nielson, F. H. (1985)Ultra trace elements current status, Nutrition Update, 2 106-126.

3- Hunt, C. ; Jol, H.; joseph, P.L. (1994) Dietary boron modifies the effect of vitamin D3 nutrition on indices of energy substrate utilization and mineral metabolism in chickens . J. Bone and Minerals , 9 (2) 171-182.

4- Hunt, C. D. (2003) Dietary boron: an overview of evidence for its role in immune function. J. Trace Elem. Exp. Med. 16 291-301.

5- Nielsen, F. H. (2008). Is boron nutrition ally relevant? Nutrition reviews, 66 (4) 183-191.

6- Puls, R. Mineral levels in animal health, diagnostic data, Sherpa international Clear book. Britsh Columbia.

7- **IPSCS** (1998) Environmental health criteria for boron. Editorial world organization Geneva 204.

8- Garther, L.; and Hiatt, J. (1997) Hitologia Texto Atlos Ed. Mc.Graw Hill. Interamericana. Mexico. Chapter 18 pp. 366-376.

9- Espinoza, O.:Bustos-Obregon, E.;and Suja, J.A. (2002) effect del parathion sobre el indice de apoptosis en hepatocitos de ratones CFI . Rev.Chil. Anat. 20 (1) 29-36.

epithelial lining are easily recognized.Atrophied glomeruli with widening renal capsular space (Arrows) E&H X20)

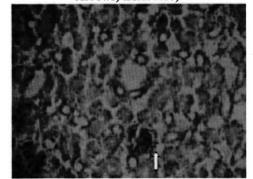


Photo 10: (group T4) Extensive destruction of the renal tubules and atrophy of the glomeruli .(E&H X20)

10- WHO (1998) international program on chemical safety, Environmental health criteria, Ohio, USA 1-201.

11- Luna, L. G. (1968) Manual of histological staining methods, The armed forces instituteof pathology 3^{rd} ed. Mc Graw Hill book company.

12- Hunt, C. and Nielson, F. (198) Interaction between boron and cholicalceferol in chicken. In Gawthorne J. White,c. Eds ,Trace element metabolism in man and animal. Vol.4 Australian academy of science, Canberra. pp. 597-600.

13- Wilson, J. H. and Ruszler, P. L. (1997) Effect of boron on growing pullets Biological trace elements research, 56 (3) 287-294

14- Fassani, E. J.; Bertechini, A. G. ;Brito, J. A. G. ;Kato, P.K.; Fialho, E. T. and Geraido, A. (2004) Boron supplementation in broiler diet. Revista Brasi. Leira de Gencia Avicola, 6 (4) 213- 217. 5-Rodica Diana, Criste.; Doina Valentina Grossu.; Romulus Scorei, and others (2005) New investication on the effect of dietary boron on broilers and layers. Boron and food quality. Arch. Zootechni. 8 65-76.

16- Bustos-Obregon, E.; Belmer, R.H., and Catrino-Galvez, R. (2008) Histopathological effect of boron on mouse liver. Inter. J. Morphology 26 (1) 155-164.

17- Gonzalez 1, M.; Herrera, A.; Dominguez, C.: Coro, R. and Lebredo, I.(2004) Effectos del acoholismo cronico sobre las cacopicas del higad de ratas

adolescents sexton congreso virtual latino-americano de anatomia patologica 1- 31.

18- Geyinkogglu, F. and Turkez, H. (2007) Acute toxicity of boric acid on energy metabolism of breast muscle in broiler chickens. Biologica Heidlberg 62 (10) 112-117.

19-Subuncuoglu, BT.; Kocaturk, PA.; Yaman, O.; Kavas, GO. And Tekelioglu, M. (2006) Effect of

subacute boric acid administration on rat kidney. Clinical Toxicolo. (Philla.) 44 (3) 249-253.

20- Mohora, M. ;Boghianu, L. ;Muscrel, C.; Duta, C. ; and Dumtrache, C. (2002) Effect of Boric acid on redox status in the rat liver. Rom. J. Biophys. 12 (3-4) 77-82.

التغيرات النسيجية في أكباد و كلى الدواجن المغذاة على علف يحوي نسب مختلفة من عنصر البورون

صباح شهاب احمد التكريتي

فرع الفسيولوجي والأدوية والكمياء الحياتية ، كلية الطب البيطري ، جامعة تكريت ، تكريت ، العراق (تاريخ الاستلام: 25 / 9 / 2012 ---- تاريخ القبول: 18 / 1 / 2013)

الملخص

قسم 40 فرخ دجاج من نوع روس بعمر 21 يوم إلى اربعة مجاميع . أضيف إلى غذاؤها ما مقداره صفر أو 100 أو 200 أو 300 ملغرام عنصر البورون . تبين من النتائج أن عنصر البورون بجرعة 100 ملغرام / كيلوغرام علف سبب تغيرات نسيجية مختلفة في كل من الكباد والكلى وا إن التغيرات اعتمدت على مقدار الجرعة. والجرعة بحدود 100 ملغرام/ كيلو علف قد تكون سامة للدواجن .