HISTOPATHOLOGY CHANGES OF HEART, LUNG, LIVER AND KIDNEY ON BROILERS UNDER CHRONIC HEAT STRESS

by Boedi Setiawan

Submission date: 14-Apr-2020 10:21AM (UTC+0800)
Submission ID: 1297015385
File name: Histopathology_Changes_of_Heart.....pdf (2.72M)
Word count: 1610
Character count: 9227

HISTOPATHOLOGY CHANGES OF HEART, LUNG, LIVER AND KIDNEY ON BROILERS UNDER CHRONIC HEAT STRESS

Djoko Legowo* ; Arimbi* ; Boedi Setiawan**
*Department of Veterinary Pathology ; ** Department of Veterinary Clinic
Faculty of Veterinary Medicine - Airlangga University

ABSTRACT

This experiment was conducted to investigate the histopathologic changes of heart, lung, liver and kidney of broilers under chronic heat stress. Fifteen birds (n = 15), twenty-one days old were kept at controlled temperature chamber ranged between 34 and 35°C for 20 days. At 41 days of age the birds were sacrificed for data collection. Under microscopic examination of heart, massive myofibrillar degeneration with hemorrhage, general fatty degeneration of myofibers and diffused myocarditis containing organisms were found in 10 out of 15 broilers (67%). The major histopathologic changes of lung were related to vein and arteriole massive congestion with interalveoli septum hemorrhage and edema. These changes were observed in 13 out of 15 broilers (87%). Almost all liver showed fatty degeneration with narrowed sinusoid. Congestion of liver central vein were found around 50% of samples, beside necrosis with heterophils and lymphocytes that was observed in some parts of the liver, especially in the portal region in 8 out of 15 broiler (53%). In the kidney, the most histopathologic changes was hidropic degeneration (70%), which largely found in renal tubular epithelia cells, beside hemorrhage and accumulation of heterophils in many inflammatory areas.

Keyword : Chronic Heat stress ; Broiler ; Histopathology of Heart, Lung, Liver and Kidney

INTRODUCTION

One of the major problem challenging poultry industry in Indonesia is the ambient temperature, which persists for about six months of the year (April to October) in most agro ecological zones of this country. During August to October, there is high temperature and low relative humidity, which results in hot humid climate, causing severe heat stress and economic loses, especially on fast growing broiler. Unfortunately information about pathologic changes of broilers in response to heat stress in Indonesia has not been reported.

High ambient temperatures can be devastating to commercial broilers; coupled with high humidity they can have an event more harmful effects. Heat stress interferes with the broilers comfort and suppresses productive efficiency. During periods of heat stress the broiler has to make major thermoregulatory adaptations in order to prevent death from heat exhaustion. The result is that the full genetic potential of the broiler is often not achieved (Butcher and Miles, 2003). During heat stress, behavioral, physiological, hormonal, and molecular changes occur on boiler (Moraes *et al.*, 2003).

Optimal temperature for efficient production on broiler ranges from 18 – 21°C. When ambient temperature rises, the body temperature tends to rise due to imbalance of thermal equation (heat produced more than heat loss) (Mehta and Shingari, 1999). During heat stress, catecholamines are released from the adrenal medulla (Gregory, 1998) due to the effect of accelerated heart rate (Ewing *et al.*, 1999); moreover this condition stimulated parabrachial nucleus causing increase respiration (Gregory, 1998). Packed cell volumes (PCV) and hemoglobin concentration (Hb) decreased in broilers under chronic heat stress (Altan *et al.*, 2000) and might cause hypoxia.

Generally, oxygen deficiency is one of the most common causes of tissue injury when combined with an increased body temperature, hypoxia becomes a potent cause of death. Heart related mortalities are observed predominantly in fast growing

broiler chickens, with edema and sudden death syndrome (SDS) being the most common heart related conditions in modern broiler flocks (Olkowski, 2007).

Normally, the function of cardiac muscle is to extract oxygen from blood efficiently, the failure of which causes the lack of oxygen in the heart muscle and results in hypoxic damage and finally heart failure. Mammalian skeletal muscle uses about 40% of oxygen in the blood that circulates through the muscle. In contrast, cardiac muscle uses nearly 100% of the oxygen from the blood circulating through the myocardial capillaries. This high oxygen demand makes myocardium susceptible to systemic hypoxia. Lack of oxygen content known caused in centrolobular areas of the hypoxic liver, and . proximal convoluted tubules on cortex renal that known highly sensitive to hypoxia (Cheville, 1999).

At present, information about pathologic conditions of broilers in response to heat stress in Indonesia has not been reported. Therefore, the objective of this experiment was to study the effect of chronic heat stress on pathogenic lesions of heart, lung, liver and kidney of broilers. This pathogenesis may explain the phenomenon of body changes and cause of death under this condition.

MATERIALS AND METHODS

Fifteen infectious disease-free broilers were obtained from a commercial hatchery and brooded for 21 days before being placed in experiment chamber. Chicks were fed on standard broiler starter in continuous light and water supply. At 22 days of age, they were transferred into an environmentally controlled chamber under heat stress condition with temperature ranged between 34-35^{o}C. All broilers were subjected to a 5-h episode of heat stress at 34-35^{o}C environmental temperature each day. During the experiment the birds were fed and drink *ad libitum* with uncontinuous light. Body temperature and respiratory rate and behavior were investigated and observed. On day 41 of the experimental period (20 days heat exposure), all birds were sacrificed by cervical dislocation. Several visceral organs (lung, liver, kidney and heart) of broilers collected and fixed in 10% buffered formalin and then proceed with routine method (H&E) for microscopic examination (magnified 100x and 400x).

RESULTS AND DISCCUSION

Lung: Congestion, edema and hemorrhage of lung were observed in 13 out of 15 broilers (87%). Commonly histopathologic changes in the lung were related to veins and massive congestion of veins (arrow) and arterioles (arrowhead) (Figure 1A.) which could be followed with massive hemorrhage (arrow) and edema. These changes were mainly observed in inter septum alveoli (arrowhead) (Figure 1B.).

Liver: Almost all liver of broiler showed fatty degeneration with narrowed sinusoid (arrow) (figure 2A/2B). Congestion (arrow head) (figure 2A) of liver central vein were found around 50% of samples, beside necrosis with heterophils and lymphocytes that was observed in some parts of the liver, especially in the portal region in 8 out of 15 broiler (53%) (figure 2B).

Kidney: In the kidney, the most histopathologic changes was hidropic degeneration which largely found in renal tubular epithelial cells and narrowed renal tubular (arrow) (figure 3A/3B) (70%), beside congestion (arrowhead) (figure 3B), hemorrhage (arrowhead) (figure 3A).and accumulation of heterophils in many inflammatory areas.

Heart: Under light microscope examination ; massive myofibrillar degeneration with hemorrhage (Figure 4A.), fatty degeneration and vacuolation of myofibers (Figure 4B), and in some cases generalized and diffuse myocarditis containing heterophil infiltration were observed in 10 out of 15 broilers (67%).

Heat stress on broiler might be challenges the haemodinamic state. Thus the stress response includes complex responses to maintain a steady state. Prime examples of such response were increased heart rate and increased blood flow to muscle, brain, and heart. This might cause congestion in kidney, liver and lung. Effects of heat stroke on the

gross lesions were dominated by severe and generalized hyperemia, which was most severe in the respiratory tract, especially in the lung, tracheal and bronchial mucosae (Ewing *et al.*, 1999). Lungs may also be edematous and occasionally contain focal consolidations of bronchopneumonia. Other organs, such as heart, kidney, may also be severely congested. The microscopic lesions are compatible with those seen grossly. The vasculatures of the lung are severely engorged and edema are evident in alveoli. Centritubular necrosis, dissociation of hepatocytes and congestion are often found in the liver. Heat stress caused endothelial cell damage through oxidative stress by increasing of free radical (Wideman, 2007). Excess lipids in hepatocytes indicate that sublethal injury has occurred. However, the swollen, yellow, greasy appearance of fatty degeneration is characteristic of liver and less common in kidney and heart. On microscopic examination, lipid accumulation causes cells to be enlarged, pale and lacy, especially in centrolobular zones – areas.

REFERENCES

Altan, O., Altan, A., Cabuk, M. and Bayraktar, H. 2000. Effect of heat stress on some blood parameter in broilers. Turk Veternerlik Ve Hayvancilik Dergisi. 24 (2): 145 – 148.

Butcher, G., D. and Miles, R. 2003. This document is VM65, one of a series of the Veterinary Medicine-Large Animal Clinical Sciences Department, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida. Original publication date May 1, 1996. Reviewed May 1, 2003. Visit the EDIS Web Site at http://edis.ifas.ufl.edu.

Cheville, N. F. 1999. Introduction to Veterinary Pathology, 2nd edition. Iowa State University Press, Ames, Iowa.

Ewing, S. A., Lay, D. C. Jr. and Borell, E. V. 1999. Farm Animal Well – Being. Simon & Schuster Company, New Jersey.

Gregory, N. G. 1998. Animal Welfare and Meat Science. The University Press, Cambridge. Guyton, A. C. 1966. Texbook of Medical Physiology. W.B. Saunders Company, Philadelphia.

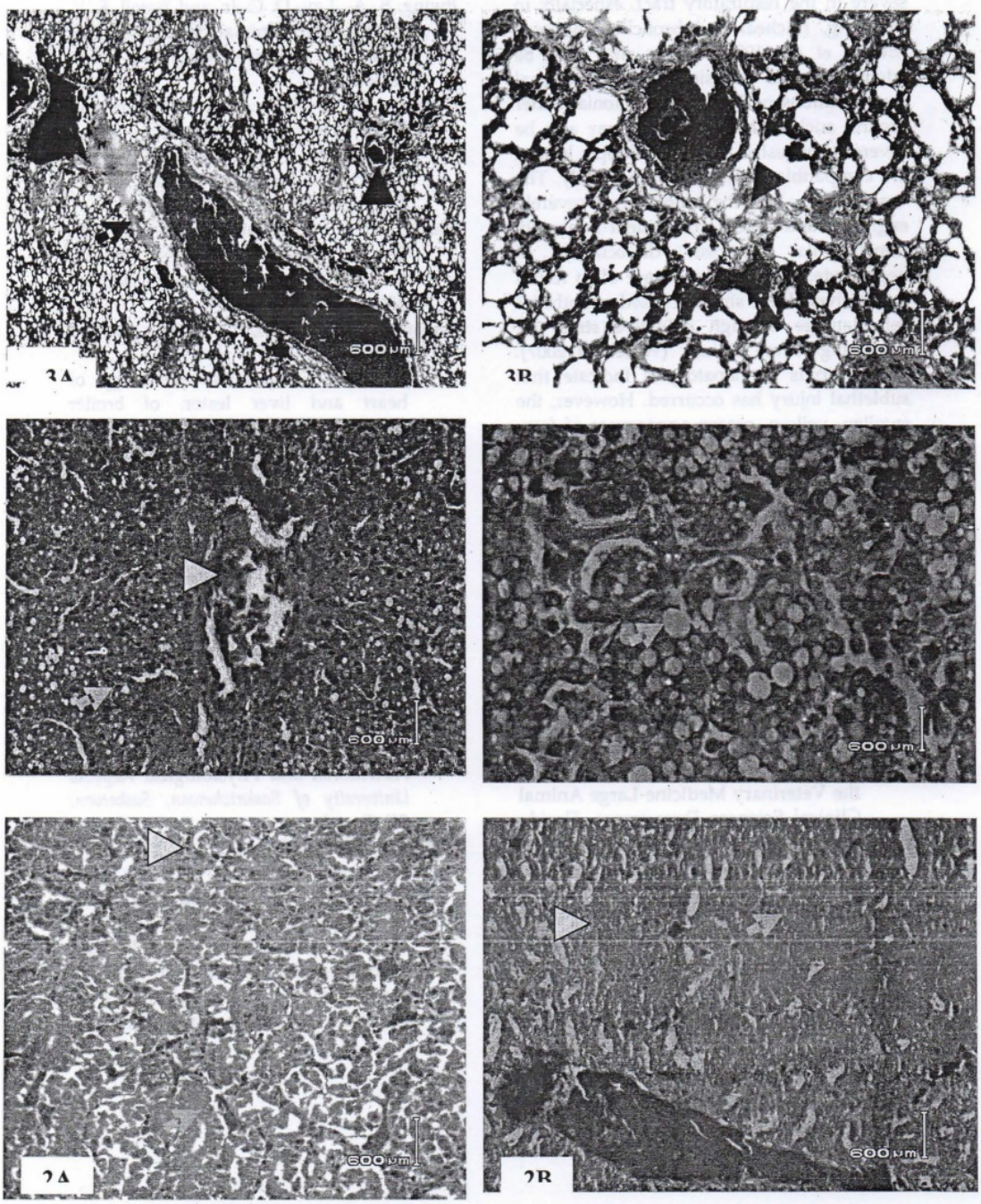
Mehta, R. K. and Shingari, B. K. 1999. Feeding under heat stress. Poultry International: Asia Pacific Edition. 30(9): 68-77.

Nakamura, K., Ibaraki, Y., Mitarai, Z., and Shibahara, T. 1999. Comparative pathology of heart and liver lesion of broiler chickens that died of ascites, heart failure, and others. Avian Dis. 43: 526 – 532.

Moraes, V. M. B. de Borges, S. A., Ariki, J.,. and. Martins, C. L 2003. Potassium chloride supplementation in heat stressed broilers. Revista Brasileira de Zootecna, 28(2):313 – 319.

Olkowski, B ., 2007. Patho-physiology of heart failure in broiler chickens: Structural, biochemical, and molecular characteristics. A. Metabolic Disease Symposium: Metabolic and Cardiovascular Diseases in Poultry: Nutritional and Physiological Aspects *University of Saskatchewan, Saskatoon, SK, Canada.*

Wideman , R., F. 2007. Inadequate pulmonary vascular capacity and susceptibility to pulmonary hypertension syndrome in broilers. Metabolic Disease Symposium: Metabolic and Cardiovascular Diseases in Poultry: Nutritional and Physiological Aspects. *University of Arkansas, Fayetteville.*


600 µm
600 µm
600 µm
600 µm
600 µm

HISTOPATHOLOGY CHANGES OF HEART, LUNG, LIVER AND KIDNEY ON BROILERS UNDER CHRONIC HEAT STRESS

ORIGINALITY REPORT

13%	11%	7%	0%
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

PRIMARY SOURCES

1	www.ijpba.info Internet Source	5%
2	docplayer.net Internet Source	3%
3	edis.ifas.ufl.edu Internet Source	2%
4	www.wattnet.com Internet Source	1%
5	Zhu, W., W. Jiang, and L. Y. Wu. "Dietary L-arginine supplement alleviates hepatic heat stress and improves feed conversion ratio of Pekin ducks exposed to high environmental temperature", Journal of Animal Physiology and Animal Nutrition, 2014. Publication	1%
6	www.tandfonline.com Internet Source	1%
7	Abdulwahab Kammon, Samia Alzentani, Omar	

Tarhuni, Abdulatif Asheg. "Effect of Some Organic Acids on Body Weight, Immunity and Cecal Bacterial Count of Chicken during Heat Stress", International Journal of Poultry Science, 2019

Publication

<1%

Exclude quotes	Off	Exclude matches	Off
Exclude bibliography	On		