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Study of Blood Cells, Blood Gases and Thyroid Hormones in Broiler Chickens Suspected of Ascites Syndrome

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Abstract: Ascites is defined as an accumulation of excessive amount of serous fluid in the abdominal cavity that leads to increased mortality and slaughter carcass condemnation in broiler chickens. Its occurrence has frequently been regarded as the result of some metabolic and physiologic disorders due to rapid growth of chickens.In this study, 100 chickens suspected of ascites, as the experimental group and 25 healthy chickens, as the control group, from 10 different broiler chicken flocks were collected and studied. The two groups of broilers had been raised in almost identical conditions. The test blood samples were procured from the wing vein and cervical aorta to assess the vein and arterial blood gases, the cell blood count (CBC),T3 and T4. The data were analyzed by SPSS statistical software. WBC, RBC and Hematocrit of the experimental group were significantly higher than those of the control group ($P<0.05$), but hemoglobin changes were not significant. There were no significant differences in MCV, MCH, MCHC, Pco2 and Po2 of the arterial blood and HCO3- of the venous blood samples between the experimental and control groups. While the amount of saturated Po2 in the arterial blood of the experimental group was lower than that of the control group ($P<0.05$). the Pco2 of the vein blood increased in the ascetic broilers ($P<0.05$). Significant reduction of Po2, pH and Po2 in saturated blood were observed in the experimental group ($P<0.05$). These results seem to support the hypothesis that analyzing blood parameters seems to be the most useful procedure for early detection of ascites and its prevention.

Key words: Broilers · Ascites · Blood cells · Blood gases · Thyroid hormone

INTRODUCTION

Ascites syndrome is defined as an accumulation of excessive amount of serous fluid in the abdomen that leads to higher mortality, an increase in feed conversion rate in broiler chickens and condemnation of chickens′ carcass [1]. The ascites syndrome in broiler chickens is composed of a collection of clinical features resulting from a pathophysiological progression from primary pulmonary hypertension to right-sided congestive heart failure, central and portal venous congestion, hepatocellular damage and transudation of fluid into the abdominal cavity [2]. This syndrome has worldwide distribution and over the years, many studies have addressed its multifarious facets. Yet there exists many unresolved issues concerning this syndrome [3]. In view of great improvements in various fields of science and technology and over half a century after the first recognition of the ascites syndrome, there still aren't any absolute solutions that can prevent the widespread occurrence of this syndrome in poultry flocks. This syndrome is multi-factorial and is capable of inflicting various damages to different organs. Therefore, this study tried to focus on the variation observed in blood parameters and thyroid hormones in the broiler chickens in Chahar-mahal va Bakhtiyari province, in the western part of Iran, where its natural conditions and climate for the occurrence of the syndrome appears to be suitable.

MATERIALS AND METHODS

In this study, first, broiler poultry houses in Chaharmahal-va-bakhtiyari province that suffered from ascites were identified. After receiving a complete history, the required samples were derived from the broilers. All broiler chickens were 45 days old. The broilers were

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raised in commercial poultry houses and fed according to NRC 1994 and received food ad libitum. All commercial chickens were from the Ross 308 strain breed, nurtured in a high altitude region (2100 meters above sea level) in Chaharmahal-va-bakhtiyari province, in the west of Iran. From 100 chickens afflicted to ascites from 10 different Flocks, 100 serum and blood samples were collected. Also, 25 samples from healthy chickens, all from the same farms, were studied. The collected samples included serum samples for appraising T4 and T3, wing vein blood containing EDTA anticoagulant substance for estimating cell blood count (CBC), wing vein blood containing Heparin anticoagulant substance for assessing VBG (vein blood gases) and aorta arterial blood consisting Heparin anticoagulant substance for measuring ABG (Arterial blood Gas).

In CBC test, the different parameters that were measured included:

- Counting of white blood cells (WBC)
- Counting of Red blood cells (RBC)
- Measurement of blood hemoglobin
- Measurement of blood hematocrit
- Measurement of average red blood cell size (MCV)
- Hemoglobin amount per red blood cell (MCH)
- The measurement of hemoglobin concentration per red blood cell (MCHC)

To count the red blood cells, symex k100 cell counter unit (TOA Medical Electronic Com.) was utilized. The operative basis of this unit was volumetric impedance.

The function of Avl 995 unit (Hornocout com.) in measuring ABG and VBG and assessing blood gases consisted of direct measurement of PH, PO_2 and PCO_2 and indirect measurement of HCO_3, Basic buffer (BB), BE, BEECF and O_2 saturated blood.

The measurement of PH was determined through the H^+ ion concentration difference created on the two electrodes and measured by a voltmeter.

The electrode responsible for measuring gases, followed the same principles as was utilized for the measurement of PH. That is, variation in CO_2 pressure of the sample causes a variation in PH and a H^+ ion concentration difference.

The measurement of Thyroid Hormones was done by gama kit (Bio-source Europe Com.). To separate the serum from the samples, the clotted blood was centrifuged twice in 5000 rpm for two minutes. Then 25 microliters of the sample were isolated for T3 test and 200 microliters of I^{125} iodine were added to it.

Table 1: Mean differences between measurement parameters in the experimental and control group (E g and C g)

	Factor	*Mean±SEM*	*P-value*
C g	*WBC*	*205400±346*	*P<0.05*
	E g	*237305±1811*	
RBC	*C g*	*2.93±0.031*	*P=0.05*
	E g	*3.58±0.128*	
HG	*C g*	*11.36±0.033*	*p>0.05*
	E g	*12.99±0.454*	
HAEMATOCRITE	*C g*	*37.46±0.2*	*P=0.05*
	E g	*47±1.3*	
MCV	*C g*	*128.7±0.286*	*p>0.05*
	E g	*130±1.07*	
MCH	*C g*	*39.2±0.351*	*p>0.05*
	E g	*39.81±0.185*	
MCHC	*C g*	*30±0.26*	*p>0.05*
	E g	*30.48±0.24*	
ABG PH	*C g*	*7.19±0.025*	*p>0.05*
	E g	*7.25±0.017*	
ABG Pco2	*C g*	*39.66±6.89*	*p>0.05*
	E g	*45.3±1.47*	
ABG BB	*C g*	*35.22±2.22*	*p>0.05*
	E g	*16.97±8.36*	
ABG Hco3	*C g*	*14.96±2.51*	*p>0.05*
	E g	*18.52±0.54*	
ABG Po2	*C g*	*59.9±8.17*	*p>0.05*
	E g	*49.2±2.38*	
ABG Po2sat	*C g*	*80.23±4.41*	*P=0.05*
	E g	*61.72±3.09*	
T4	*C g*	*1.67±0.012*	*P=0.05*
	E g	*0.92±0.059*	
T3	*C g*	*100.67±2.18*	*p>0.05*
	E g	*106.2±6.29*	
VBG PH	*C g*	*7.35±0.017*	*P=0.05*
	Eg	*7.26±0.129*	
Pco2 VBG	*C g*	*35.4±3.4*	*P=0.05*
	Egt	*46.9±1.65*	
VBG B	*C g*	*42.7±0.91*	*p>0.05*
	E g	*42.2±0.62*	
VBG Hco3	*C g*	*17.6±0.67*	*p>0.05*
	E g	*19.6±0.53*	
VBG Po2	*C g*	*57.4±2.31*	*P=0.05*
	E g	*35.4±2.12*	
VBG Po2 sat	*C g*	*87.2±1.8*	*P=0.05*
	E g	*50.2±4.28*	

After being incubated for one hour in the shaker unit, it was perused by the gama kit. For T4 experiment, 500 microliters of I^{125} iodine was added to 20 microliters of the sample. Then the mixture was incubated in the shaker unit for one hour and finally read by the gama kit.

The data and the statistical procedures thereof were analyzed using the SPSS15 software and comparison of the results was done by applying the *Mann-Whitney* non-parametric test.

RESULTS AND DISCUSSION

The results achieved through this study indicated that there were significant changes in the blood factors of the broilers suffering from the ascites syndrome. It may be inferred that the measurement of blood gases and the lab analysis thereof can be employed as an effective forewarning method before the occurrence of ascites in the broilers brings about greater losses or mutilation.

The blood gases present in the veins seemed to be very similar to O_2 pressure and CO_2 pressure found in the tissue.

One report alleged that Vein blood PCO_2 in chickens sensitive to ascites was more than that of chickens resistant to ascites [3].

The vein PCO_2 increase and vein PO_2 decrease can serve as a sign of chronic lung failure that is triggered by reabsorption of kidney Bicarbonate [4]. So, Hypoxia and PCO_2 increase can be regarded as the main impairments of chickens suffering from ascites.

The present study illustrated that the means of WBC, RBC, hematocrit, T4, arterial blood saturated O_2, vein blood PH, vein blood carbon dioxide, vein blood PO_2 and vein blood saturated O_2 between the experimental and control groups were statistically different at the level of $P<0.05$.

Buys *et al*. [5] reported that the amount of saturated hemoglobin with O_2 in cold-stressed ascitic broilers was less than that of chickens resistant to ascites. In that examination, vein blood PH did not show any differences in the experimental and control groups and it could not be considered a good parameter for the prognosis of ascites.

The mean of blood hemoglobin, MCV, MCH, MCHC, tri-iodoteronin amount (T3), arterial blood PH, CO_2 pressure of arterial blood, bicarbonate in arterial blood, O_2 pressure in arterial blood and vein blood bicarbonate between the experimental and control groups showed no significant differences ($P>0.05$).

Research concerning saturated hemoglobin with O_2 showed that the amount of saturated hemoglobin with O_2 in fast growing broilers was less than saturated hemoglobin in slow growing broilers [6]. Also, Julian and Mirsalimi [7] reported that percent hemoglobin oxygen saturation was significantly lower in chickens with RVF than in control chickens impaired with valvular insufficiency. This might be because of high blood pressure in lung capillaries in chickens suffering from ascites that did not let hemoglobin to saturate completely in the company of O2 [8]. Also, the hypoxemia was caused by a diffusion limitation associated with an excessively rapid rate of pulmonary blood flow [9]. In other words, when the RBCs did not reside at the gas exchange surfaces long enough to saturate the hemoglobin fully with oxygen, a diffusion limitation was exposed and hypoxemia ensued. [10]. Chronic hypoxia was caused by pulmonary hypertension leading to the development of ascites in domestic fowls [9].

Hematocrit is another common measure of ascites development [7]. Shlosberg *et al*. [11] reported hematocrit increase in chickens suffering from ascites in comparison with healthy chickens within one or two weeks before death. The reason of hematocrit increase in ascitic chickens was the escalation of metabolism and the high demand of tissues for O2 that was instigated by the diminishing of O2 saturation and the rise of hematocrit [12]. However, these changes occurred prior to gross physical changes and could be used as early markers for ascites incidence [13].

Probably, the cause of WBC increase is the immune response to unusual cases and the reason behind RBC rise is body response to reduce O_2 and compensate it. High CO_2 pressure in vein blood is probably initiated by blood pressure buildup in the lung and gas exchange disability of CO_2 with O_2 or lung disorders like the thickness of lung air capillary wall.

Balog *et al*. [14] reported PCO2 in vein blood of young Ross broiler chickens resistant to ascites to be more than that of chickens sensitive to ascites.

The decrease of T3and T4 is one of the physiologic reactions in the ascites syndrome. Instability of T3 and T4 at the normal level may trigger the ascites process. The increase in metabolism is known to be associated with the secretion rise in T4 as well as T3. T3 is the main hormone that stimulates the body metabolism and is related to body temperature regulation. Moreover, it is known as a main factor in the growth of chickens. This means that concentration of plasma thyroid hormones plays a part in the relationship between the metabolism rise in ascetic chickens and the expansion of ascites incidence [11]. Luger *et al*. [15] showed that T4 concentration in ascitic chickens reduced significantly in one week before death.

The total impact of hypoxemia on cardiopulmonary hemodynamics during the pathogenesis of ascites is complex. In view of the high altitude and low O_2 pressure in Chaharmahal-va-Bakhtyari province, this study, in farm conditions, showed that hematological changes, blood gases and thyroid hormones in farm and practical conditions are similar to each other, irrespective of

the cause of ascites, region, species, food type and other reasons initiating ascites. Therefore, the measurement of these blood parameters might be a useful precautionary measure in prognosticating the syndrome in ascites-suspicious flocks. In this study, since sampling was done in different regions, from different flocks and among different age levels, it seemed evident that the changes in the measured parameters were similar and the effective factors in the syndrome could not have any role in the amount of the above parameters. Thus, it may be safe to say that it is possible to prognosticate and even prevent the ascites syndrome by consecutive sampling before the syndrome's strike in the first stages of the chickens' growth.

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