Global Veterinaria 9 (3): 262-265, 2012
ISSN 1992-6197

DOI: 10.5829/idosi.gv.2012.9.3.6528

Comparative Effect of Vitamin E and Vitamin E-Selenium Compound on T4-Induced Ascites Syndrome (By T4-Suplementation of the Diet) in Broiler Chickens

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Abstract: 120, 1-day-old Ross 308 broiler chickens were randomly divided into 4 experimental groups A, B, C and D. Group A as control negative group,. Beginning of the breeding period to drinking water in all groups except Group A, Group B, as control positive group, Thyroxin (T4), (1mg/L) was added to a higher incidence of ascites. Group C, (400 IU/L) vitamin E and Group D combination of selenium-vitamin E (0.5 ml/L) received. Production factors such as weight per groups, food intake, feed conversion ratio (FCR), average weight of chickens were measured weekly. Weekly mortality was calculated. Factors related to carcass ratio (RV/TV), Bursa of Fabricius weight, liver weight and spleen weight was recorded at the end of the growing period. T4 diet led to an increase in ascites. Add the combination of vitamin E-selenium and vitamin E reduced mortality from ascites. Vitamin E and E-selenium combination were not affecting on performance of production factors. None of the groups in factors related to the carcass was not affected. According to the results obtained by adding compounds such as vitamin E and vitamin E-selenium combination, can be control and prevention of ascites syndrome and is effective in improving the productive factors.

Key words: Broilers · Ascites · Selenium · Vitamin E · Thyroxin

INTRODUCTION

The incidence of high altitude disease or ascites resulting from pulmonary hypertension (PH) and. Consequently, cardiac hypertrophy, heart failure and oedema, in broiler chickens kept at altitudes of approximately 3000m, have been described by Walton *et al*. [1]. More recently, similar sings and mortality rates were reported in broilers reared at lower altitudes of 1000 to 2000m and at altitudes down to sea level Decuypere *et al*. [2]. Ascites is a complex syndrome for which the major inducing factor is thought to be a lack of oxygen. Decreased oxygen tensions and/or increased oxygen requirements can create hypoxic conditions at the tissue level and this may seriously influence PH. Many questions remain to be answered concerning the factors leading to hypoxia and the pathophysiological effects of hypoxia on the chicken. Histopatologhy studies by Maxwell *et al.* [3] indicated that a large number of inflammatory cells were observed in numerous tissues of ascetic birds and in the hearts of experimentally-induced hypoxic birds. Also, Enkverchakul *et al*. [4] pointed out that activated white blood cells can generate a variety of reactive oxidant status. In addition, dietary vitamin C has been reported to improve resistance to a variety of stressors, including toxic salt and hypoxia Al-Taweil and Kassab [5]. The primary cause of ascites in broilers is hypoxia-induced PH, but the exact biochemical mechanisms responsible for producing the disease have not been identified. There are several reports Al-Taweil and Kassab, Enkvetchakul *et al*. [5, 4] suggesting that free radical-mediated mechanisms may be involved in the etiology of PH syndrome (PHS). Bottje & Wideman [6] recorded that production of free radicals is contributed to by systemic hypoxia, inflammation and thyroid hormones. The potential of oxygen-derived free radicals to cause cytotoxic damage was also proposed by Maxwell *et al*. [3]. They suggested that together whit granulocytes,

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mitochondria in ascetic and hypoxic birds may be a source of oxygen-free radicals in injured myocardial cells and that these components may play an important role in the synthesis of tissue damage in broilers. They also argued that oxygen-free radicals cause tissue damage through lipid peroxidation of cell membranes which leads to increased membrane permeability. These reviews support the idea that oxygen- derived free radicals are generated, rather than ischemia in hypoxic birds. Because oxygen-derived free radicals play an important role in the genesis of tissue damage during inflammatory reactions and ischemia in humans, Halliwell and Gutteridge [7], it is reasonable to hypothesis that the development of ascites may be due in part to free radical generation from mitochondria of cardiomyocytes of ascetic and hypoxic birds, with subsequent depletion of tissue anti oxidants Maxwell *et al*. [8]. Earlier studies showed that dietary vitamin E can be effective in reducing mortality in reared under environmental stress, Ruiz-Feria [9]. concluded that, although vitamin E is not an essential for chickens maintained in optimal conditions, it may become an essential vitamin for birds under environmentally, pathologically stressful conditions. This experiment was conducted to explore the effect of vitamin E and vitamin E-selenium combination on the mortality of ascites and performance of some of productive and carcass parameters in commercially grown broilers fed with Thyroxin (T4). Considering that T4 increases the mortality of ascites, the 400IU / L vitamin E or 0.5 ml/L vitamin E-selenium in drinking water can show difference mortality between ascites in birds fed with vitamin E and vitamin E-selenium diet and birds fed normal and T4 that were used as positive control groups.

MATERIALS AND METHODS

120 chickens 1- day- old male Ross 308 were divided to 4 groups A, B, C and D. Each group was trained in 3 repeated in separated Penn under identical environmental conditions. Group A (gpA) as control negative group. Group B (gpB), as control positive group, Thyroxin (T4) (1 mg / L) was added to a higher incidence of ascites. Vitamin E and vitamin E-selenium, as Thyroxin (T4) was added to the water birds. Group C (gpC), 400 IU/L vitamin E, GroupD(gpD)combination of vitamin E and selenium (0.5ml/L) and Production factors such as weight per groups, food intake, feed conversion ratio (FCR), average weight of chickens were measured weekly. Weekly mortality was calculated. Factors related to carcass ratio (RV/TV), Bursa of Fabricius weight, liver weight and spleen weight was recorded at the end of the finisher period. Throughout the study mortality was recorded daily. At the termination of the experiment, ten chickens from each group were randomly selected and slaughtered. These birds and the broilers which died during the experimental period were examined for lesions of heart syndrome and ascites. The heart was removed and the atria, major vessels and fat were trimmed off. The right ventricle (RV) was carefully cut away from the left ventricle and septum. The right ventricle was weighted, the left ventricle and septum were added and the total ventricle (TV) was weighted. Birds having a RV/TV ratio of over 0.299 were classified as suffering from right ventricular failure, Julian [10]. Statistical analysis was performed using the 'LSMeans procedure', SAS [11].

RESULTS

Ascites Mortality and RV/TV ratio: The number of broiler that developed right ventricular hypertrophy and Ascites in the different age and treatment groups and the ratios of RV/TV are shown in Table 1. During the experiment 5 (6.16%) of the 120 birds died. All of them showed RVH and ascites up to 5 weeks of age. Dietary T4 markedly increased ascites mortality (mean RV/TV ratio = 0.37). There was no difference in mean RV/TV ratio of surviving

Table 1: Ascites mortality and corresponding RV/TV ratio in commercial broilers that don't give any supplemented (control) and groups that given T4 supplemented with vitamin E and vitamin E - selenium compound

Dietary supplement	---------- Ascites mortality ----------							RV/TV	Ascite cases without mortality
NonegpA	1wk	2wk	3wk	4wk	5wk	6wk	Total	.031	2
T4 gpB	0	0	0	0	0	0	0	0.32	9
T4 and Vitamin EgpC	0	0	0	1	1	3	5	0.30	4
T4 and vitamin E-selenium compoundgpD	0	0	0	0	0	0	0	0.29	0
Total	0	0	0	1	1	3	5	0.32	15

Ascites mortality occurred from 3 weeks of age onward and the rate increased considerably between week 4 and 6. In the dietary T4 groups, 30 birds which had been T4 only died due to RVF induced ascites, while the mortality of Ascites was clearly reduced (100%) bye the effect of vitamin E (400IU/L) and Vitamin E-selenium compound (0.5ml/L) supplementation in the T4 treatment group.

Table 2: Mean body weight gain (g/chicken/week), feed intake (g/chicken/week) and feed conversion ratio in commercial broilers that divided to 4 groups A, B, C and D. Group A as control group. Group B given T4, group C given T4 and vitamin E and group D given T4 and vitamin E-selenium combination

		Age (week)					
Dietary supplement		1	2	3	4	5	6
Body weight gain							
gpA	None	97a	258a	53a	957a	1421a	1850a
gpB	T4	94a	245a	559a	598a	1413a	1840a
gpC	T4 & vitamin E	94a	225a	524a	927a	1398a	1780a
gpD	T4 and vitamin E-selenium combination	94a	237a	533a	963a	1446a	1850a
Feed intake							
gpA	None	813b	3348a	8311a	16513a	26754a	38569a
gpB	T4	750c	3399a	7899a	15894a	26530a	38100a
gpC	T4 & vitamin E	900a	3200a	8158a	15934a	26674a	38352a
gpD	T4 and vitamin E-selenium combination	749c	3166a	8120a	15989a	25919a	37990a
FCR							
gpA	None	1.66a	1.61a	1.76a	1.87a	2.00a	2.10a
gpB	T4	1.56a	1.64a	1.58a	1.78a	2.08a	2.80a
gpC	T4 & vitamin E	1.65a	1.76a	1.70a	1.80a	1.97a	2.07a
gpD	T4 and vitamin E-selenium combination	1.51a	1.73a	1.72a	1.79a	1.98a	2.08a

a,b,c Means with the same indices within a column are not significantly different for P<005

birds which were killed randomly at 42 days of age. In ascites chickens the heart was flaccid due to right ventricular hypertrophy and dilation. The lungs and kidneys were congested and sometime hemorrhagic. The liver was swollen or shrunken.

Growth: T4 did not decrease the growth the same as vitamin E and vitamin E-selenium combination. (Table 2)

Feed Intake: Vitamin E and vitamin E-selenium combination did not cause any effect on feed intake. (Table 2).

FCR: Vitamin E and vitamin E-selenium did not change FCR. (Table 2).

DISCUSSION

This study suggests that in birds reared whit T4 supplementation the addition of vitamin E (400IU/L) and vitamin E-selenium combination (0.5ml/L) in the diet of broilers has no effect on growth, feed intake and feed conversion rate. Autopsy on birds which died during the experiment revealed that the clinical signs, lesions and RV/TV ratio in ascites birds correspond with those reported at high altitude, Julian [12]. The highest mortality due to ascites was found in the six week which corresponds with the period of maximal weight gain. Dietary T4 can be used as a model to increase the incidence of ascites and discriminatory power for studying other factors involved in ascites, Buys *et al*. and Decuypere *et al*. [2, 13-15]. Vitamin E and vitamin E-selenium combination, decreased Ascites incidence both in T4 supplemented feed and this reduction of ascites in vitamin E and vitamin E-selenium treated bird could be a consequence of the reduction of blood flow resistance, especially in the narrow capillaries of the lungs, Julian [16]. Our results confirm the earlier finding of ascites, who observed that Vitamin E and vitamin E-selenium supplementation of broiler drinking water reduced ascites incidence Ruiz-Feria, [9]. Enkvetchakul and Al-Taweil and kassab [4,5] suggested that the reduction of ascites mortality may be a consequence of depression in antioxidant status by vitamin E and vitamin E-selenium supplementation. This study showed in birds that Ascite induced, adding vitamin E and vitamin E-selenium combination had no effect on growth, feed and feed conversion ratio and it can be concluded that the addition vitamin E and vitamin E-selenium combination to the diet clearly counteracts the increase in ascites mortality due to dietary T4 supplementation. Further studies are needed to elucidate the exact underlying physiological mechanism.

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