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The Study of Relation Between Normal and Higher than Standard Concentration of Sodium of Drinking Water in Ascites Syndrome Declaration in Broiler Chickens

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Abstract: Eight drinking water trials were conducted to study the tolerance of young chickens to various concentrations of sodium of drinking water. Chickens tolerated 40 ppm of sodium of drinking water from hatch to seven weeks of age without harmful effects as judged from the data on mortality rate, feed intake, body weight, ascite fluid presence, heart injuries and plasma sodium and protein amounts. High mortalities with ascites and heart injuries occurred in chickens that drank drinking water which its sodium concentration was 350 ppm. Mortalities had a direct relation with increase in levels of sodium in drinking water.

Key words: Ascites, sodium, drinking water and broiler chickens

Introduction

Ascites syndrome is a condition caused by fluid retention in hepato - peritoneal cavity with congestion and dilation of venous blood vessels (Bezuidenhout, 1988; Sakumi *et al*., 1996) which may be accompanied by hydropericardium, hydrothorax, subcutaneous edema or visceral gout (Gibson, 1957). This syndrome can result in flock mortality and has been reproduced experimentally in broiler chickens (Owen *et al*., 1990; Witzel *et al*., 1990; Yersin *et al*., 1992). Ascites syndrome has a worldwide distribution (Buys and Barnes, 1987) and its appearance has multifarious causes (Sholsberg *et al*., 1992). Some main causes of ascites syndrome deceleration in broiler chickens are poor ventilation, cold exposure, low temperatures, type of feed, bird's sex and excess of sodium in diet or drinking water (Julian, 1993). Ascites and cardiac dilation were also seen in chickens on high salt intakes in practical conditions (Morrison *et al*., 1975; Pang *et al*., 1979). Food and drinking water are sources of excess sodium but between these two sources, drinking water is more important because on a weight basis, broiler chicks usually consume water up to 2. 4 time more than feed (Williams, 1996). Salt is the main source of sodium in water or diet. In practical conditions, excessive salt intake could occur if young chickens were offered salty well water or consumed feed accumulating with salt (Pang *et al*., 1982). Chickens up to about 21 day of age are very sensitive to excess of sodium (Julian *et al*., 1992) and sodium toxicosis is a usual finding at these periods (Julian, 2002). Feed and water are usually analyzed for this compound to evaluate their role as possible etiologic factors. Because of importance of sodium as a main factor of acites declaration, we decided to study the tolerance of young chickens to varying levels of sodium of drinking water.

Materials and Methods

Two hundred 1 - day - old broiler chickens (Ross strain) which consist of male sex were obtained from a commercial hatchery with a good reputation of producing diseases free chickens. The chickens were allotted at random to 8 pens with the same food and standard rearing condition. Eight treatments were randomly assigned to these pens. The control and treatment groups (A, B, C, D, E, F and G) were received drinking water which contained 0 (distilled water), 40, 100, 150, 200, 250, 300 and 350 ppm of sodium respectively from day 0 to slaughter time. All aspects of diet had absolutely balanced and all groups received same diet which had 0.4% sodium. The birds were given free access to feed and water. Mortality and incidence of ascites in the dead chickens which showed abnormal accumulations of fluid in the abdominal cavity were recorded every day. In order to prevent outbreak of newcastel disease (ND), all chickens were vaccinated at 3 day-old with ND oil-emulsion vaccine. Water and feed intakes were respectively measured every day and every seven day. At seven weeks of age, ten chickens from each group were randomly selected and after a heparinized blood sample from the right wing vein, were killed and their body and heart were determined. For each blood sample, packed cell volume (PCV) was determined by a microhematocrit technique. For each sample, sodium concentration of plasma was determined by flame - photometer apparatus. Total protein amount, for each plasma sample, was determined with Kjeldahl method (White *et al*., 1978).

Data from this study were subjected to analysis of variance (ANOVA) and Duncan multiple range test (Snedecor and Cochran, 1980). The level of significance was set at 5%.

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Table 1: Levels of sodium of drinking water are shown in parenthesis. All chickens that died from ascites syndrome and incidence time of first mortalities at treated groups.

Treatment	Ascites mortality	Incidence time of first mortality (day)
Group B (100 ppm)	1/23	38
Group C (150 ppm)	5/24	30
Group D (200 ppm)	8/23	27
Group E (250 ppm)	11/25	19
Group F (300 ppm)	11/23	15
Group G (350 ppm)	14/25	12

Results

Between control and A and B groups, cumulative feed intakes had not significant deference ($p > 0.05$) at all age periods. Compared with control group, significant decrease in cumulative feed intakes was seen at group C older than two weeks of age ($p < 0.05$) and D, E, F and G groups at all age periods ($p < 0.05$).The body weights had not significant deference between control, A and B groups ($p > 0.05$) but there was an obvious deference between body weights of control and A groups with C, D, E, F and G groups ($p < 0.05$).

At control, B, D and F groups, two chickens from each group and at A and C groups, one chicken from each group died at younger than four days of age. These chickens had yolk sac infection which *Escherichia coli* were isolated from them.

Mortalities with ascites were observed in B, C, D, E, F and G groups. Incidence of mortality increased with increase in levels of sodium in drinking water (Fig. 1).

There was a decrease in the time of first mortality with increase in the levels of sodium in drinking water because first ascites mortality of group B was happen at 38 days old but first mortalities of other groups were happen before group B. There was no ascites mortality at control and A groups. At group B, there was only 1 scites case but at other groups there were many ascites cases which with increase in the level of sodium in drinking water, the rate of mortalities were increased (Table 1). Incidence time of ascites mortalities was mainly between 14 - 33 days of age.

None of control and group A chickens which killed at seven weeks of age had ventricle dilatation and there was no grossly observable fluid retention in these chickens. Six out of 10 chickens of group B which were slaughtered at seven weeks of age had right ventricular enlargement and 2 of them had biventricular dilation but no carcasses had any grossly observable ascites. At the other groups there was obvious sign of fluid retention, varying extents, in the hepato - peritoneal cavity of slaughtered chickens.

PCV and Plasma sodium contents in B, C, D, E, F and G groups were significantly higher than control and A groups ($p < 0.05$). Plasma protein amounts were significantly higher in the control and A groups than other groups ($p < 0.05$).

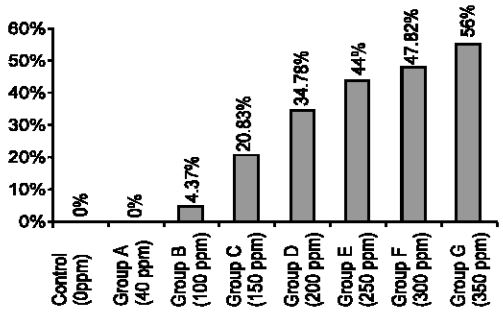

Fig. 1: Mortality percents from ascites in treated groups with variable concentrations of sodium in drinking water (numbers in parentheses indicate the sodium concentration of drinking water).

Discussion

The detrimental effects of excess water salt in poultry which are consist of generalized venous congestion, accumulation of fluid in peritoneal and pericardial cavities and right ventricular dilation and failure has been known for many years (Selye and Stone, 1943; Julian, 1987). Ascites from any cause also occur in fast-growing meat- type chickens with primary pulmonary hypertension (Julian, 1993). Pulmonary hypertension is the main cause of the right ventricular failure which is induced by many factors such as salt (Julian, 1987). In chicken, pathophysiology of salt which induces pulmonary hypertension is not completely understood. Reduction of erythrocyte deform ability has been shown with excess dietary salt in broiler chickens and this may affect blood flow dynamics and cause pulmonary hypertension (Mirsalimi *et al*., 1992). Young chickens are more susceptible to salt than older birds. Water and dietary salt greatly increases plasma sodium concentration in younger chickens but young chicks are unable to regulate the higher plasma sodium level and therefore, have a greater blood volume expansion than older chickens (Morley *et al*., 1980).

In chicken, standard amount of salt (as main source of sodium) in water is 40 ppm (Shanawany *et al*. 1989).The avian blood capillaries are small and can dilate only very little to accommodate the extra blood volume produced by excess sodium (Julian, 2002) and high sodium intake may cause arteriolar vasoconstriction (Mulvany, 1984) which will finally cause ascites. This study demonstrated that excess amount of sodium of drinking water (especially greater than 100 ppm) produced variable degrees of pulmonary hypertension, right ventricular hypertrophy, biventricular dilation, right ventricular failure and ascites in broiler chickens and with increase in levels of sodium in drinking water, blood volumes were significantly increased. This added volume would increase the workload on the heart and produce the ascites.

Observation made from the present study confirmed the previous findings that incidence of mortality from ascites occurred mainly between two and four weeks of age and these moralities increased with increase in drinking water levels of sodium (Pang *et al.,* 1980; Morrison *et al.,* 1975) .

Results of this study indicate that for accessing to better flock performance, notification to sodium amounts of drinking water as one of the main factors of acsites declaration syndrome is very important.

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