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Research Article

Turk. J. Vet. Anim. Sci.
2011; 35(3): 143-148
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doi:10.3906/vet-0708-14

Acetic acid modulates induction of pulmonary hypertension in broiler chickens: based on electrocardiographic parameters

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Received:13.08.2007

Abstract: To clarify the effect of acetic acid on the electrocardiographic parameters of pulmonary hypertensive broilers, chicks were reared at high altitude and treated with acetic acid (0.4 and 0.9 g/L) for 45 days. The right ventricle-to-total ventricle ratio was noted as an index of pulmonary hypertension that was significantly ($P < 0.05$) decreased in the treated groups. S amplitudes were significantly decreased only at 36 days (leads II and III) and 45 days (lead aVF) in many treated groups. There were significant ($P < 0.05$) reductions of T amplitudes at 28 days (leads aVR and aVL) and 36 days (lead aVL), and significant elevations of QRS (leads II, III, and aVF) and QT intervals (leads II, aVR, aVL, and aVF) at different ages of many treated groups. R amplitudes were significant ($P < 0.05$) at 28 days (leads aVL and aVF, at both doses of acetic acid), 36 days (leads aVR and aVL, at 0.4 g/L of acetic acid), and 45 days (lead aVR, at both doses of acetic acid). The RR interval also significantly increased at 28 days (leads aVR and aVL) and 45 days (lead aVL). It was concluded that acetic acid modulates induction of ventricular hypertrophy, dilation, and arrhythmia. These effects are detectable in many electrocardiographic parameters.

Key words: Acetic acid, electrocardiographic parameters, broiler, altitude, pulmonary hypertension

Introduction

Among chickens, meat-producing broiler strains are highly prone to severe pulmonary hypertension and congestive right heart failure. The increased susceptibility of the broiler chicken to pulmonary hypertension is believed to be due to increased metabolic rates and high oxygen requirements causing increased cardiac output, in conjunction with restricted vascular space in the lungs that results in pulmonary hypertension. However, growth rate, oxygen requirements, organ size and capacity, hematological parameters, and cellular responses can all determine how resistant or susceptible a broiler is to pulmonary hypertension syndrome. In addition, environmental causes such as altitude, cold stress, and rearing conditions, such as feed, lighting, air quality, and ventilation, have all been implicated in pulmonary hypertension development (1). Decreasing the oxygen level to below normal could cause the broiler to become more susceptible to pulmonary hypertension (2). The incidence of pulmonary hypertension has been reported to be greatly increased at altitudes greater than 1300 meters above sea level, presumably because of low oxygen concentration (3).

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Short-chain or volatile fatty acids (acetate, butyrate, and propionate) have been used as feed additives for poultry for many years. Volatile fatty acids are bacteriostatic or bactericidal in vitro for gram-negative bacteria (4). Van Der Wielen et al. (5) showed a correlation between pathogen control and the presence of undissociated levels of acetate, propionate, and butyrate in the cecum. In addition, butyrate appears to play a role in the development of the intestinal epithelium and thus the better absorption of food (6). It has been indicated that acetate is a vasodilator and a suppressor of arterial pressure (7). Our goal in this study was to evaluate the acetate effect on the heart based on electrocardiographic changes in broiler chickens exposed to pulmonary hypertension due to high altitude.

Materials and methods

Animals, management, and treatments

A total of 162 broiler chickens 1-day old from the Ross 308 breed were randomly divided by a poultry net fence into 3 equal groups (1 control and 2 acetic acid-treated groups) with 3 replicates per group (54 birds per group, 18 birds per replicate) and were reared in floor pens on shavings and sawdust litter at a high altitude (2100 meters) for 45 days. From 1 day of age, all chickens were kept under a 24-h light regimen and provided ad libitum access to water and a standard ration (starter: 3200 kcal metabolizable energy/kg of diet, 23% crude protein; grower: 3200 kcal metabolizable energy/kg of diet, 20% crude protein; finisher: 3200 kcal metabolizable energy/kg of diet, 18% crude protein) formulated to meet the requirements of the National Research Council for broilers (8). Acetic acid was purchased from Merck Chemical Co. (Bubendorf, Germany) and used from 1 day of age at 2 doses, 0.4 and 0.9 g/L, by dissolving it in drinking water.

Electrocardiographic recordings

At days 28, 36, and 45, 9 chickens from each group were randomly selected, and electrocardiograms were recorded with an automatic recorder (Cardiomax FX-2111, Fukuda, Japan) and standardized at 10 mm = 1 mV with a chart speed of 50 mm/s. Leads I, II , III, aVR, aVL, and aVF were recorded for every chicken. The amplitude of T, R, and S waves; the intervals of QRS, QT, RR, and ST; and the mean electrical axis (MEA) were measured.

Dissection and assessment of right ventricle hypertrophy

After the recording of the electrocardiograms, the chickens were killed by decapitation and then right ventricle hypertrophy was determined, as previously described by Cueva et al. (9). The heart was dissected and the atria were removed to the plane of the atrial-ventricular valves, and then the total ventricles (TV) were weighed. The right ventricular (RV) wall was then dissected free of the left ventricle (LV) and septum. The RV was weighed and the RV-to-TV ratio was calculated. Pulmonary hypertension syndrome was defined as a RV-to-TV ratio greater than 0.28 during the experiments (10).

Statistical analysis

All results are represented as mean ± SEM. Comparisons were made by one-way ANOVA using SPSS 14.0, with $P < 0.05$ accepted as significant.

Results

Assessment of right ventricle hypertrophy

The RV-to-body weight ratios in the acetic acid-treated groups decreased at different ages and were significant at 45 days (Table 1). The RV-to-TV ratio decreased in all ages of acetic acid-treated groups and was significant at 28, 36, and 45 days when compared to the control groups ($P < 0.05$) (Table 1). This decrease was 28.8% in the 28-day groups, 22.4% in the 36-day groups, and 24.2% in the 45-day groups. The TV-to-body weight ratio decreased in acetic acid-treated groups but was not significant. The LV-to-body weight ratio was also not significant (Table 1). Statistical analysis showed no significant differences among the treated groups for any of the other measured ratios.

Electrocardiographic parameters and MEA

S, T, and R wave amplitudes: There were reductions of the S wave amplitudes at 28, 36, and 45 days for both doses of acetic acid (leads II, III, and aVF), but this was only significant ($P < 0.05$) at 45 days (lead aVF, 0.4 g/L of acetic acid) and 36 days (leads II, III, and aVF, 0.4 g/L of acetic acid) compared to the controls. Variations in other leads were not significant. T wave amplitudes were decreased at most leads but were only significant ($P < 0.05$) at 28 days (lead aVR, 0.4 g/L acetic acid; lead aVL, 0.4 and 0.9 g/L acetic acid)

Table 1. Cardiac indices and mean electrical axis (MEA) in different groups.

% TV/BW	% RV/BW	% LV/BW	RV/TV	MEA	Group	Age (days)
0.43 ± 0.01	0.14 ± 0.01	0.29 ± 0.01	0.33 ± 0.02	156.3 ± 44.0	C	
0.41 ± 0.02	0.10 ± 0.01	0.31 ± 0.02	0.24 ± 0.01*	253.4 ± 21.4	T (0.4 g/L)	28
0.39 ± 0.03	0.09 ± 0.01	0.30 ± 0.03	0.23 ± 0.01*	125.1 ± 25.4	T (0.9 g/L)	
0.38 ± 0.00	0.11 ± 0.02	0.26 ± 0.02	0.29 ± 0.02	245.5 ± 22.8	C	
0.36 ± 0.02	0.09 ± 0.01	0.27 ± 0.01	0.23 ± 0.02*	115. 6 ± 35.1	T (0.4 g/L)	36
0.35 ± 0.02	0.08 ± 0.01	0.27 ± 0.02	0.22 ± 0.02*	118.3 ± 24.5	T (0.9 g/L)	
0.35 ± 0.02	0.11 ± 0.01	0.24 ± 0.02	0.31 ± 0.02	194.5 ± 24.9	C	
0.33 ± 0.02	0.07 ± 0.00*	0.24 ± 0.01	0.24 ± 0.01*	176.5 ± 31.9	T (0.4 g/L)	45
0.30 ± 0.01	0.07 ± 0.00*	0.23 ± 0.01	0.23 ± 0.01*	204.0 ± 44.7	T (0.9 g/L)	

* Significantly different vs. corresponding control (P < 0.05).

and 36 days (lead aVL, 0.4 and 0.9 g/L) in the treated groups (Table 2) (Figure). Decreases of the R wave amplitudes were significant (P < 0.05) at 28 days (leads aVL and aVF, for both doses of acetic acid), 36 days (leads aVR and aVL, 0.4 g/L of acetic acid), and 45 days (lead aVR, for both doses of acetic acid).

QRS, QT, RR, and ST intervals and mean electrical axis (MEA): QRS intervals were increased in all treated groups at leads II, III, and aVF, but were not significant at lead III (28 days) in the treated groups in comparison to the control (Table 3). RR and QT intervals also relatively increased in all ages of the treated groups. QT intervals were significant (P < 0.05) at 36 days (leads aVR, II, aVL, and aVF) and 45 days (lead aVL) for 0.4 or 0.9 g/L of acetic acid, and RR intervals were also significant (P < 0.05) at 28 (leads aVR and aVL) and 45 (lead aVL) days compared to the controls (Table 3) (Figure). MEA (Table 1) and ST intervals (not shown) did not significantly change in the treated groups at different ages when compared to the controls. Electrocardiographic parameters and MEA variations were not statistical significant among the treated groups.

Table 2. Many electrocardiographic wave amplitudes in different groups.

Age (days)	Lead Group	S			T		R		
		II	III	aVF	aVR	aVL	aVR	aVL	aVF
28	C	0.25 ± 0.04	0.16 ± 0.02	0.21 ± 0.03	0.16 ± 0.02	0.08 ± 0.03	0.18 ± 0.03	0.09 ± 0.02	0.18 ± 0.02
	T (0.4 g/L)	0.20 ± 0.04	0.14 ± 0.03	0.16 ± 0.03	0.10 ± 0.00*	0.02 ± 0.00*	0.08 ± 0.02	0.02 ± 0.00*	0.07 ± 0.02*
	T (0.9 g/L)	0.15 ± 0.01	0.11 ± 0.02	0.14 ± 0.02	0.12 ± 0.02	0.02 ± 0.01*	0.11 ± 0.00	0.03 ± 0.01*	0.08 ± 0.03*
36	C	0.34 ± 0.06	0.25 ± 0.05	0.29 ± 0.06	0.13 ± 0.02	0.12 ± 0.01	0.22 ± 0.04	0.08 ± 0.02	0.25 ± 0.04
	T (0.4 g/L)	0.13 ± 0.04*	0.07 ± 0.02*	0.10 ± 0.03*	0.09 ± 0.02	0.02 ± 0.02*	0.08 ± 0.03*	0.02 ± 0.01*	0.17 ± 0.04
	T (0.9 g/L)	0.19 ± 0.05	0.12 ± 0.04	0.11 ± 0.03	0.10 ± 0.02	0.03 ± 0.01*	0.13 ± 0.02	0.04 ± 0.16	0.17 ± 0.05
45	C	0.31 ± 0.06	0.18 ± 0.04	0.24 ± 0.04	0.13 ± 0.01	0.03 ± 0.03	0.24 ± 0.02	0.05 ± 0.06	0.25 ± 0.04
	T (0.4 g/L)	0.15 ± 0.05	0.08 ± 0.03	0.11 ± 0.03*	0.10 ± 0.02	0.02 ± 0.00	0.10 ± 0.03*	0.02 ± 0.00	0.16 ± 0.05
	T (0.9 g/L)	0.16 ± 0.02	0.09 ± 0.03	0.13 ± 0.03	0.11 ± 0.01	0.02 ± 0.00	0.14 ± 0.02*	0.02 ± 0.00	0.13 ± 0.02

* Significantly different vs. corresponding control (P < 0.05).

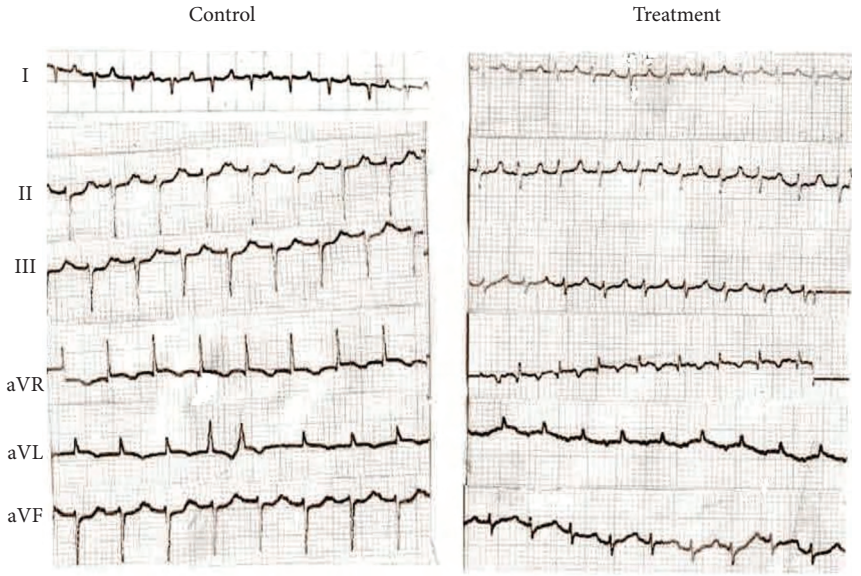

Figure. Samples of different electrocardiographs in 2 groups of hypertensive chickens (control and acetic acid-treated) at 45 days. Standardization, 10 mm = 1 mV; chart speed, 50 mm/s.

Table 3. Many electrocardiographic wave intervals in different groups.

Age (days)	Lead Group	QRS				QT			RR	
		II	III	aVF	aVR	II	aVL	aVF	aVR	aVL
28	C	0.01 ± 0.00	0.03 ± 0.01	0.01 ± 0.00	0.10 ± 0.00	0.10 ± 0.01	0.09 ± 0.02	0.10 ± 0.01	0.01 ± 0.00	0.04 ± 0.05
	T (0.4 g/L)	0.04 ± 0.00*	0.03 ± 0.00	0.04 ± 0.00*	0.11 ±0.00	0.11 ± 0.01	0.12 ± 0.00	0.11 ± 0.00	0.14 ± 0.00*	0.14 ± 0.00*
	T (0.9 g/L)	0.04 ± 0.00*	0.04 ± 0.00	0.04 ± 0.00*	0.12 ± 0.01	0.11 ± 0.01	0.11 ± 0.01	0.11 ± 0.01	0.13 ± 0.00*	0.13 ± 0.00*
36	C	0.02 ± 0.00	0.02 ± 0.00	0.02 ± 0.00	0.09 ± 0.01	0.08 ± 0.01	0.04 ± 0.04	0.09 ± 0.02	0.13 ± 0.01	0.13 ± 0.00
	T (0.4 g/L)	0.04 ± 0.00*	0.04 ± 0.00*	0.04 ± 0.00*	0.11 ± 0.00*	0.11 ± 0.00*	0.11 ± 0.00*	0.11 ± 0.00*	0.13 ± 0.00	0.13 ± 0.00
	T (0.9 g/L)	0.03 ± 0.00*	0.03 ± 0.00*	0.04 ± 0.00*	0.10 ± 0.00	0.10 ± 0.00	0.09 ± 0.00*	0.1 0 ± 0.00	0.14 ± 0.01	0.14 ± 0.01
45	C	0.02 ± 0.00	0.02 ± 0.00	0.02 ± 0.03	0.11 ± 0.00	0.10 ± 0.00	0.04 ± 0.04	0.11 ± 0.00	0.13 ± 0.01	0.05 ± 0.01
	T (0.4 g/L)	0.04 ± 0.00*	0.04 ± 0.00*	0.04 ± 0.02*	0.11 ± 0.00	0.12 ± 0.00	0.11 ± 0.01*	0.11 ± 0.00	0.14 ± 0.01	0.14 ± 0.01*
	T (0.9 g/L)	0.04 ± 0.00*	0.04 ± 0.00*	0.04 ± 0.02*	0.12 ± 0.01	0.12 ± 0.01	0.12 ± 0.01*	0.11 ± 0.01	0.14 ± 0.01	0.14 ± 0.01*

* Significantly different vs. corresponding control ($P < 0.05$).

Discussion

Acetic acid is a volatile fatty acid that naturally enters the metabolism of the body. It is absorbed from the gastrointestinal tract and is completely utilized in oxidative metabolism or in anabolic syntheses. Isotope experiments have shown acetic acids to be utilized in the formation of glycogen, intermediates of carbohydrates, and fatty acid synthesis, as well as cholesterol synthesis. In addition, it participates in the acetylation of amines; it may be converted to alanine by transamination and thence incorporated into the proteins of the plasma, liver, kidneys, gut mucosa, muscle, and brain (11). Acetic acid is apparently metabolized primarily by acetyl-CoA synthase, which is expressed by a number of tissues, including the liver and heart. Starnes et al. (12) observed that isolated hearts perfused with acetic acid as a substrate had increased oxygen consumption and coronary blood

flow as compared with hearts supported by glucose. Based on conventional biochemical measurements of adenine nucleotides, creatine, and creatine phosphate, these authors felt that hearts perfused with acetic acid had a higher phosphorylation potential, suggesting that they were metabolically "healthier." Kiviluoma et al. (13) also examined the effects of acetic acid on cardiac function and metabolism. In an in vivo model, they found decreases in mean arterial pressure and increases in hearts with exposure to intravenous infusions of acetic acid. Suokas et al. (7) showed that, after an infusion period in men, 75 min of treatment by acetic acid increased cardiac output from the baseline by 17%, and decreased peripheral arterial resistance (19%) and diastolic blood pressure (10%). Circumferential fiber shortening velocity was maximally increased during the acetic acid experiment by 7% from the baseline. These data indicate that acetic acid is an arterial vasodilator and a mild diuretic, and it may slightly improve myocardial performance. Amore et al. (14) reported that the vasodilation effect of acetic acid is mediated by nitric oxide. It appears that acetic acid induces the production of cAMP, which is a powerful stimulus of NO synthase (NOS). On the other hand, acetic acid is known to increase adenosine levels in tissues and blood, which induces vasodilation (15).

The most obvious environmental factor that plays a role in pulmonary hypertension development in broilers is high altitude. The effect of high altitude (either natural or simulated) is a decrease in the partial pressure of oxygen. At sea level, oxygen makes up 20.9% of the atmosphere. The equivalent percentage of oxygen drops by approximately 1.0% for every 500 m of rise in elevation (16). The bird's ability to oxygenate the blood depends on the thickness of the blood-gas barrier, the oxygen affinity of the hemoglobin, the transit time in the lungs, and the percentage of oxygen in the air. At altitude, hypoxic stimulation of chemoreceptors increases ventilation, thereby increasing CO_2 washout, reducing P_aCO_2, and increasing pH_a. Although at sea level pH_a is allowed to fluctuate within a rather narrow range of 7.37-7.43, at altitude the mechanisms that titrate pH_a to the normal value are less efficient. Therefore, the blood remains alkaline, perhaps because of slow renal compensation that cannot cope with continuous CO_2 washout from the lungs. It is possible that acetic acid lowers pH in the blood, and the result is probably the offsetting of hyperventilation-induced respiratory alkalosis and the allowance of chemoreceptors to respond more fully to hypoxic stimuli at altitude. Improvement of alkalosis causes the equilibrium of the reaction $HHb+O_2$ Ü Þ HbO_2+H^+, shifting toward the formation of deoxygenated hemoglobin (Hb) upon the increasing acidity of the medium, the well-known Bohr effect (17,18).

It seems that these advantages of acetic acid decrease the development of pulmonary hypertension in broiler chickens, as shown in our study with the decreasing RV-to-TV and RV-to-body weight ratios in treated groups.

Our previous study on the electrocardiographic parameters of cold-induced pulmonary hypertensive broilers determined that T and S waves had significant elevations, in agreement with the reports of Odom et al. (19,20), Owen et al. (21), Wideman and Kirby (22-24), and Martinez et al. (25). Those results could also be used as evidence that dilation and hypertrophy of ventricles were the primary cause of the increased amplitude of the S waves (long ventricle depolarization) (26). In the present study, it was shown that T and S waves decreased during administration of acetic acid in the various age groups; therefore, it is suggested that broilers supplemented with acetic acid had lower rates of hypertrophy and dilation of the ventricles. QRS, QT, and RR intervals were also increased in many of the age groups, evidence for the inhibition of tachycardia in acetic acid-supplemented chickens exposed to pulmonary hypertension (27,28).

It is concluded that supplementation of acetic acid can modulate induction of pulmonary hypertension, hypertrophy, and dilation and arrhythmia of ventricles due to high altitude at 28, 36, and 45 days of age, and these effects are detectable in some electrocardiographic parameters, such as the decreasing of T and S wave amplitudes and the increasing of QRS, QT, and RR intervals.

Acknowledgements

This work was supported by funds granted by the Vice Chancellor for Research of Shahrekord University.

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