Effects of long-term calcium deficiency on the electro-and echocardiography in laying hens

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Abstract:

BACKGROUND: Ca is the most important minerals in the body that plays a key rols in the physiological activities, anzymatic reaction and the regulation of myocardial contraction and relaxation. Ca deficiency causes the heart failure and decrease cardiac contractility. **OBJECTIVES:** To determine the effects of long-term dietary calcium deficiency on the heart function of layer hens based on the electro- and echocardiography. **METHODS:** Ninety Hyline W36 hens were kept for 21 weeks and fed by rations with different amounts of calcium. At 20, 28 and 36 weeks of age, electrocardiographic, echocardiographic and post-mortem left ventricular parameters were assessed. **RESULTS:** S wave amplitude was significantly ($p<0.05$) increased in the Ca-deficient group II (in lead II) at 36 weeks of age in compared to control. There were also elevations of the QRS wave amplitudes in 28 and 36 weeks of age at two Ca-deficient groups (leads II, III, aVR, aVL and aVF) but were only significant ($p<0.05$) in Ca-deficient group II (leads II and aVF) in compared to control. Left ventricular free wall diameter at end-systole were significantly ($p<0.05$) increased in the Ca-deficient group II at 28 and 36 weeks of age compared to the control group. Inter-ventricular septal diameters at end-systole were decreased in two Ca-deficient groups at 28 and 36 weeks ($p<0.05$). Left ventricular free wall diameter at post-mortem were significantly ($p<0.05$) increased in two Ca-deficient groups at 36 weeks of age compared to the control group. **CONCLUSIONS:** From these results, it can be suggested that long-term dietary calcium deficiency alters in electro- and echocardiographic parameters, which could reflect pathological left ventricular hypertrophy in the laying hens.

Introduction

The increased incidence of cardiomyopathies in the broiler and layer chickens population has been attributed to selective breeding for rapid growth, feed efficiency, high egg production and muscle development (Baghbanzadeh and Decuypere, 2008; Martinez et al., 1997; Olkowski and Classen, 1995). On the other hand, in recent years, the increased productivity of broiler and layer chickens due to changes in genetic potential of poultry and partly due to improved management practices, have caused that the genetic make-up of the bird influences the utilization of Calcium (Ca^{2+}) (Hurwitz et al., 1995; Shafey et al.,

1990) and thereby its requirement (Rama Rao et al., 2003).

Ca^{2+} plays a central role in the regulation of myocardial contraction and relaxation. During each cardiac beat, a small leak of calcium through the voltage-dependent L-type calcium channels (VDLC) triggers massive calcium release from an internal storage pool in the sarcoplasmatic reticulum (SR) through SR-bound ryanodine receptors (RyR) into the cytoplasm of heart muscle cells, which triggers muscle contraction. Cardiac relaxation is initiated by the concerted action of the sodium/calcium exchanger (NCX) and muscle-specific SR calcium ATPase-2 (SERCA2), which resequesters calcium into the internal calcium storage pool, allowing for the next quantal release of calcium. SERCA2 thereby promotes both cardiac relaxation and contractility (Franzini-Armstrong et al., 2005). On top of this elaborate system of calcium pumps, the cardiac β-adrenergic receptor system controls the magnitude of the calcium transient by means activating protein kinase A (PKA), a kinase that by virtue of its direct phosphorylation of the VDLC, RyR, SERCA2 and phospholamban allows for intracellular calcium transients with higher amplitude and faster reuptake into the SR calcium storage. Specifically, PKA-mediated phosphorylation of phospholamban dissociates the latter from SERCA2, allowing for faster calcium reuptake into the SR (Armand and De Windt, 2004).

Ca^{2+} plays a key role in both the short- and long-term properties of cardiac cells and is thus involved in the development of arrhythmias. The notion that these ions do play a key role in the development of arrhythmias is not surprising. The structure of cardiac cells enables rapid electrical conduction as well as rapid activation of the contractile system even though diffusion of Ca^{2+} is slow. Nature has, therefore, provided amplification stations between the sarcolemma and the myofibrils so that both the delivery and the removal of Ca^{2+} are accelerated. Simultaneously, the Ca^{2+} sensitivity of many proteins in the cardiac cell is so high such that activation of contractile proteins occurs at Ca^{2+} only slightly above the diastolic level. Furthermore, Ca^{2+} affects the electrical processes at the surface membrane profoundly. It is therefore plausible that instability of the Ca^{2+} transport systems is involved in the mechanisms that lead to overall instability of the tissue during arrhythmias (Eisner et al., 2006; Ter Keurs and Boyden, 2007).

Techniques such as electrocardiography and echocardiography have been used to perform repeated evaluations of cardiovascular function in chickens (Hassanpour et al., 2009; Odom et al., 1992; Owen et al., 1995). These techniques, however, assess cardiac function indirectly, and noninvasive means to directly and repeatedly monitor cardiac morphology and function in the chicken are needed. Echocardiography is a noninvasive technique used for the evaluation of cardiac structure and function in both experimental and clinical settings. The unique feature of this technology is real time visualization of the living, beating heart. With this technique, reliable heart mass, chamber dimensions, and systolic function have been obtained (Martinez-Lemus et al., 1998).

At present study, both electrocardiography and echocardiography were used to investigate effects of long dietary calcium deficiency on cardiac function in the laying hens.

Materials and methods

Animals, management and treatments: The Research Animal Ethic Committee of Shahrekord University approved this experimental protocol. Ninety commercial White Leghorn pullets (Hyline W36) aged 15 weeks were randomly and equally allotted into three groups (30 pullets in each group) with three replicates in each group (ten birds per replicate). The birds were housed in individual cages (40 × 47 cm). The experiment lasted 21 weeks during which water and food were supplied ad libitum. They were exposed to 16 h of light per day during the laying period of the experiment. Room temperature in this system was controlled to 21 ± 2.0^{o}C. The experimental diets were formulated according to the requirements for laying hens suggested by National Research Council (1994). Three experimental diets were formulated to contain 35.5 (as control), 20.7 (as Ca-deficient I) and 6 (as Ca-deficient II) g Ca / kg diet utilizing common feed ingredients. The levels of metabolizable energy (2950 kcal / kg), crude protein (15.5%), lysine (0.9%), methionine (0.52%) and methionine + cysteine (0.82%) were kept constant in

all diets.

Electrocardiography: From each group 10 chicks were randomly selected at weeks 20, 28 and 36, and electrocardiograms were recorded by an automatic recorder (Cardiomax FX-2111, Fukuda, Japan) while standardized at 10mm = 1mV with a chart speed of 50mm/s. Leads II, III, aVR , aVL and aVF were recorded for each bird . Then, the amplitude of T, R, S, QRS waves, the intervals of QT, ST were measured (Martinez et al., 1997).

Echocardiography: Echocardiography in the chickens were performed as described by Martinez-Lemus et al., 1998. Images were obtained while birds were minimally restrained in a standing position. A probe (7.5-9 MHz) placed in a parasternal position was used to generate the images. The probe was positioned 1 to 2 cm dorsal to the ventral midline just in front of the stifle joint and angled steeply cranially. Left ventricular free wall and inter-ventricular septal thicknesses measurements, as well as left ventricular internal diameter measurements were made at the time of echocardiographic examination. Images were optimized for detection of endocardial surfaces in both M- and B- mode examinations. Measurements were done by M mode. Diameters were measured between the atrioventricular valves and the papillary muscles for the left ventricle, and at the septal insertion of the atrioventricular valve for the right ventricle at both, end-systole and end-diastole. Accurate short axis cross-section of the ventricles was verified using two-dimensional images, checking for an essentially circular structure of the left ventricle. End-systole and end-diastole were identified as the smallest and largest distance between the endocardial surfaces of the ventricles during the cardiac cycle. Fractional shortening (end-diastolic diameter minus end-systolic diameter divided by end-diastolic diameter) was calculated for both right and left ventricles. At 20, 28 and 36 weeks of age, 10 birds from each group were randomly selected and studied echocardiographically. All in vivo cardiac evaluations were performed using a Medison EX 8000 CE color flow mapping echocardiograph.

Dissection and assessment of post-mortem left ventricle: After electroechocardiography, the selected birds were euthanized by cervical dis-location at 20, 28 and 36 weeks of ages. Hearts were removed via thoracotomy. After removal of the vascular trunks at their insertion with the myocardium and without the pericardial sac, the ventricles were dissected from the atria, and left ventricular diameters, weight of left ventricle to total ventricles, inter-ventricular septal thicknesses and left ventricular free wall thicknesses were measured between the endocardial insertion of the left atrioventricular valve and the papillary muscles. All post-mortem measurements were obtained using a Vernier caliper.

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

Results

Electrocardiographic parameters: Electrocardiographic parameters (i.e. R, S, T, QRS waves) are shown in the tables 1 and 2 and figure 1. S wave amplitude was significantly ($p<0.05$) increased in the Ca-deficient group II (in lead II) at 36 weeks of ages in compared to control. R wave amplitude was also increased at most leads at two Ca-deficient groups in 28 and 36 weeks but were not significant ($p<0.05$) (Table 1). There were elevations of the QRS wave amplitudes in 28 and 36 weeks of ages at two Ca-deficient groups (leads II, III, aVR, aVL and aVF) but were only significant ($p<0.05$) in Ca-deficient group II (leads II and aVF) in compared to control and Ca-deficient group I. There were not significant differnces in T wave amplitudes among Ca-deficient and control groups (Table 2). Variations in QT and ST intervals were also insignificant in Ca-deficient groups compared with control (data not shown).

Echocardiographic and post-mortem left ventricular parameters: Left ventricular parameters of echocardiography are shown in the table 3 and figure 2. Left ventricular left free wall diameter at end-systole (LFWDS) were significantly ($p<0.05$) increased in the Ca-deficient group II at 28 and 36 weeks of age compared to the control group. Inter-ventricular septal diameters at end-systole (IVSDS) were decreased in two Ca-deficient groups at 28 and 36 weeks ($p<0.05$). Other left ventricular parameters of echochardiography i.e. left ventricular diameter at end-diastole (LVDD), left ventricular diameter at end-systole (LVDS), left ventricular free wall diameter at end-diastole (LFWDD), inter-ventricular

Table 1. Amplitude of S and T waves at different groups. [a,b,c] Means with the different indices between groups (within the same times) are significantly different for p<0.05.

Ages	groups	n	S wave (mV)				
			II	III	aVR	aVL	aVF
	Control	10	0.05±0.005	0.05±0.005	0.03±0.003	0.01±0.001	0.04±0.007
20 weeks	Ca-def-I	10	0.05±0.004	0.05±0.003	0.03±0.004	0.01±0.001	0.05±0.004
	Ca-def-II	10	0.04±0.004	0.04±0.004	0.02±0.003	0.01±0.005	0.04±0.004
	Control	10	0.47±0.027	0.49±0.038	0.27±0.019	0.01±0.002	0.48±0.027
28 weeks	Ca-def-I	10	0.58±0.055	0.54±0.057	0.32±0.031	0.01±0.001	0.55±0.059
	Ca-def-II	10	0.55±0.036	0.50±0.032	0.29±0.031	0.01±0.004	0.53±0.034
	Control	10	0.46±0.030a	0.49±0.049	0.22±0.040	0.01±0.001	0.47±0.040
36 weeks	Ca-def-I	10	0.56±0.030b	0.51±0.043	0.28±0.020	0.03±0.009	0.53±0.033
	Ca-def-II	10	0.61±0.023b	0.51±0.022	0.32±0.023	0.01±0.009	0.54±0.022
			T wave (mV)				
	Control	10	0.02±0.009	0.01±0.001	0.01±0.002	0.01±0.002	0.01±0.001
20 weeks	Ca-def-I	10	0.01±0.002	0.01±0.001	0.01±0.001	0.01±0.001	0.01±0.001
	Ca-def-II	10	0.01±0.002	0.01±0.001	0.01±0.001	0.01±0.001	0.01±0.002
	Control	10	0.10±0.011	0.14±0.015	0.07±0.014	0.07±0.010	0.11±0.018
28 weeks	Ca-def-I	10	0.12±0.008	0.10±0.003	0.07±0.007	0.06±0.005	0.10±0.005
	Ca-def-II	10	0.14±0.018	0.13±0.022	0.09±0.011	0.07±0.015	0.11±0.007
	Control	10	0.11±0.019	0.13±0.021	0.12±0.026	0.09±0.010	0.12±0.015
36 weeks	Ca-def-I	10	0.11±0.011	0.12±0.011	0.09±0.005	0.09±0.008	0.10±0.004
	Ca-def-II	10	0.12±0.013	0.10±0.003	0.09±0.009	0.07±0.008	0.10±0.011

Table 2. Amplitude of R and QRS waves at different groups. [a,b,c] Means with the different indices between groups (within the same times) are significantly different for p<0.05.

Ages	groups	n	R wave (mV)				
			II	III	aVR	aVL	aVF
	Control	10	١00.0±١0.0	0.01±0.001	0.01±0.001	0.02±0.003	0.01±0.001
20 weeks	Ca-def-I	10	0.01±0.007	0.01±0.002	0.01±0.003	0.02±0.002	0.02±0.009
	Ca-def-II	10	0.01±0.005	0.01±0.001	0.01±0.004	0.01±0.004	0.00±0.001
	Control	10	0.06±0.034	0.01±0.008	0.02±0.015	0.23±0.021	0.01±0.009
28 weeks	Ca-def-I	10	0.07±0.035	0.01±0.001	0.04±0.027	0.29±0.022	0.03±0.016
	Ca-def-II	10	0.14±0.052	0.03±0.009	0.09±0.035	0.27±0.021	0.04±0.009
	Control	10	0.09±0.044	0.01±0.003	0.06±0.044	0.23±0.021	0.01±0.009
36 weeks	Ca-def-I	10	0.05±0.026	0.01±0.001	0.05±0.023	0.23±0.051	0.01±0.003
	Ca-def-II	10	0.09±0.045	0.01±0.001	0.07±0.036	0.23±0.011	0.03±0.009
			QRS wave (mV)				
	Control	10	0.05±0.005	0.05±0.005	0.03±0.003	0.02±0.003	0.04±0.007
20 weeks	Ca-def-I	10	0.06±0.009	0.05±0.004	0.03±0.004	0.02±0.002	0.06±0.004
	Ca-def-II	10	0.05±0.004	0.04±0.003	0.03±0.003	0.02±0.002	0.05±0.009
	Control	10	0.52±0.042	0.50±0.039	0.28±0.027	0.23±0.020	0.50±0.031
28 weeks	Ca-def-I	10	0.65±0.047	0.54±0.057	0.36±0.032	0.29±0.024	0.57±0.054
	Ca-def-II	10	0.69±0.073	0.56±0.039	١٥0.0±8٤.0	0.27±0.023	0.64±0.065
	Control	10	0.51±0.039a	0.41±0.080	0.27±0.060	0.23±0.021	0.47±0.039a
36 weeks	Ca-def-I	10	0.61±0.038a	0.51±0.043	0.33±0.017	0.26±0.027	0.53±0.033a
	Ca-def-II	10	0.71±0.047b	0.52±0.022	0.40±0.034	0.24±0.012	0.63±0.032b

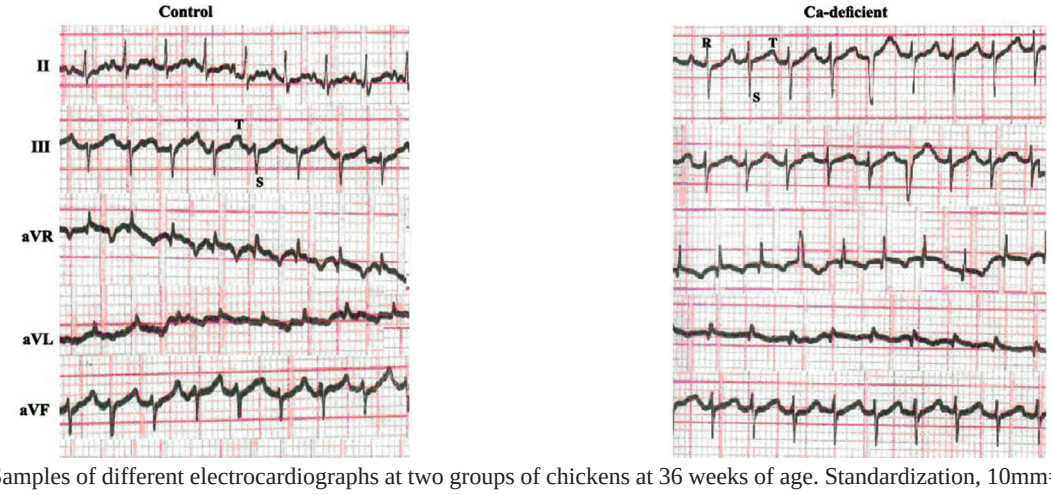

Figure1. Samples of different electrocardiographs at two groups of chickens at 36 weeks of age. Standardization, 10mm=1mV; chart speed, 50mm/s. QRS and S waves are increased on the ECG of Ca-deficient chicken.

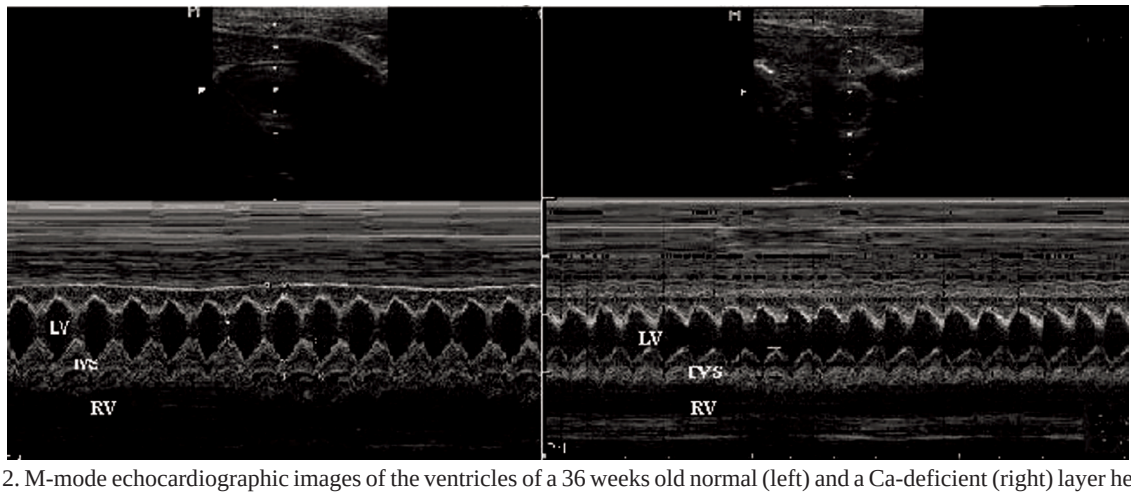

Figure 2. M-mode echocardiographic images of the ventricles of a 36 weeks old normal (left) and a Ca-deficient (right) layer hen. IVS, inter-ventricular septum.

septal diameters at end-diastole (IVSDD), left ventricular fractional shortening (LVFS) and LV/TV were insignificant in the Ca-deficient groups compared to the control.

Parameters of the post-mortem left ventricle are shown in the table 4. Left ventricular free wall diameter (LFWD) were significantly ($p<0.05$) increased in two Ca-deficient groups at 36 weeks of age compared to the control group. Left ventricular diameter (LVD) and inter-ventricular septal diameter (IVSD) did not significantly changed in the Ca-deficient groups compared to the control.

Discussion

For the maintenance of cardiac function, there may be no more important molecule than calcium. Ca^{2+} plays a central role in the regulation of myocardial contraction and relaxation. Hypocalcemia can cause heart failure that is responsible to the infusion of calcium. Elevation of serum ionized calcium has been shown to augment contractility in patient with heart failure. In myocytes obtained from patient with end-stage heart failure, the action potential is prolonged. The intracellular Ca^{2+} transient, as assessed by the fluorescent indicator fura-2, demonstrates a blunted rise with depolarization reflecting a slower deliry of Ca^{2+} to the contractile apparatus and a slowed rate of fall during repolarization. These two abnormalities could explain both systolic and diastolic dysfunction (Braunwald et al., 2001). At the present study we found that in long dietary calcium deficiency, some electrocardiographic parameters such as S and QRS waves were significantly increased that could be evidence of abnormality in ventricular action potential which

Table 3. Echocardiographic parameters in the different groups. [a,b,c] Means with the different indices between groups (within the same times) are significantly different for p<0.05. n=numbers; LVDD = left ventricular diameter at end-diastole; LVDS = left ventricular diameter at end-systole; LFWDD = left ventricular free wall diameter at end-diastole; LFWDS = left ventricular left free wall diameter at end-systole; IVSDD = inter-ventricular septal diameters at end-diastole; IVSDS = inter-ventricular septal diameters at end-systole; LVFS = left ventricular fractional shortening.

Age	Group	n	LVDD (cm)	LVDS (cm)	LFWDD (cm)	LFWDS (cm)	IVSDD (cm)	IVSDS (cm)	LVFS (%)
	Control	**10**	**0.89±0.01**	**0.34±0.01**	**0.22±0.01**	**0.46±0.01**	**0.28±0.01**	**0.57±0.01**	**62.12±0.82**
20 weeks	**Ca-def-I**	**10**	**0.86±0.03**	**0.34±0.03**	**0.22±0.01**	**0.46±0.02**	**0.28±0.01**	**0.53±0.01**	**60.44±3.21**
	Ca-def-II	**10**	**0.86±0.02**	**0.34±0.03**	**0.23±0.01**	**0.46±0.02**	**0.25±0.01**	**0.51±0.03**	**61.07±3.02**
	Control	**10**	**0.99±0.02**	**0.43±0.01**	**0.20±0.01**	**0.42±0.01a**	**0.25±0.01**	**0.51±0.03**	**56.56±1.21**
28 weeks	**Ca-def-I**	**10**	**0.98±0.01**	**0.45±0.02**	**0.20±0.01**	**0.42±0.01a**	**0.24±0.01**	**0.52±0.01**	**54.57±1.39**
	Ca-def-II	**10**	**0.96±0.02**	**0.40±0.02**	**0.19±0.01**	**0.46±0.01b**	**0.25±0.01**	**0.54±0.01**	**58.95±1.53**
	Control	**10**	**0.90±0.01**	**0.41±0.02**	**0.20±0.01**	**0.42±0.01a**	**0.24±0.01**	**0.54±0.02a**	**54.24±1.46**
36 weeks	**Ca-def-I**	**10**	**0.88±0.02**	**0.39±0.02**	**0.19±0.01**	**0.41±0.01a**	**0.21±0.01**	**0.45±0.01b**	**55.87±1.63**
	Ca-def-II	**10**	**0.90±0.02**	**0.37±0.01**	**0.20±0.01**	**0.46±0.01b**	**0.22±0.01**	**0.46±0.02b**	**58.40±0.97**

Table 4. Post mortem left ventricle parameters in the different groups. [a,b,c] Means with the different indices between groups (within the same times) are significantly. different for p<0.05. Ca-def-I = Ca-deficient group I; Ca-def-II = Ca-deficient group II; n= numbers; LVD = left ventricular diameter; LFWD= left ventricular free wall diameter; IVSD= inter-ventricular. septal diameters.

Ages	Group	n	LFWD(cm)	LVD(cm)	IVSD
	Control	**10**	**0.53±0.069**	**0.50±0.024ab**	**0.49±0.035**
20 weeks	**Ca-def-I**	**10**	**0.53±0.015**	**0.44±0.020a**	**0.46±0.016**
	Ca-def-II	**10**	**0.54±0.029**	**0.54±0.017b**	**0.42±0.013**
	Control	**10**	**0.50±0.008**	**0.63±0.017ab**	**0.42±0.016**
28 weeks	**Ca-def-I**	**10**	**0.50±0.005**	**0.59±0.018a**	**0.41±0.008**
	Ca-def-II	**10**	**0.51±0.016**	**0.68±0.014b**	**0.38±0.021**
	Control	**10**	**0.42±0.004a**	**0.64±0.005**	**0.40±0.06**
36 weeks	**Ca-def-I**	**10**	**0.46±0.009b**	**0.61±0.010**	**0.42±0.06**
	Ca-def-II	**10**	**0.44±0.004c**	**0.63±0.020**	**0.41±0.08**

resulted in the hypertrophy of the left ventricle. On the other hand, among measured echocardiographic and post-mortem parameters, left ventricular free wall diameter at end-systole and left ventricular free wall diameter (post-mortem) were higher in calcium-deficient hens than normal groups. These parameters confirmed the hypertrophy of left ventricle in calcium-deficient layer hens (Schamroth, 1985). In the our previous study, we found that dietary calcium deficiency for 6 weeks in broiler chickens caused decreasing of R, S, T waves (as electrocardiographic parameters) and left ventricular fractional shortening and also increasing of the left ventricular diameter at end-systole (as echocardiographic parameters) in the Ca-deficient group that could be evidence of abnormality in ventricular action potential which resulted in the reduction of ventricular contractility (Zamani Moghaddam et al., 2010). It is probably that these controversial results are due to different types of bird with different ages and different requirements to calcium (broiler chicken vs. layer hen) and also the time of calcium deficiency (6 weeks for broilers vs. 21 weeks for layer hens). These factors could differ the influence of calcium deficiency on the heart of the broiler chickens and layer hens.

It has been confirmed that calcium deficiency induced systemic hypertension (Hatton and Mc-Carron, 1994; McCarron and Morris, 1987; Tuan and Nguyen, 1987) and increased plasma parathyroid hormone (López-Miranda et al., 1998). On the other

hand, It has been reported that pressure overload to the heart, such as hypertension, gradually results in pathological cardiac hypertrophy (Richey and Brown, 1998). Long-term of high plasma parathyroid hormone also adversely affect the myocardial function, induce cardiac hypertrophy and cause high arterial blood pressure (Hara et al., 1995; Liu et al., 2008; Saleh et al., 2003). Therefore, the previous studies suggest that cardiac hypertrophy (resulted from electro-echocardiography and post-mortem assessments) in the calcium-deficient hens of the present study is probably due to indirect factors such as long-term hypertension and high plasma parathyroid hormone.

It is concluded that long-term dietary calcium deficiency altered electro- and echocardiographic parameters, which might reflect pathological left ventricular hypertrophy in the laying hens.

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ارزیابی اثرات کمبود کلسیم طولانی مدت بر شاخص‌های الکتروکاردیوگرافی و اکوکاردیوگرافی قلب مرغان تخم گذار

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چکیده

زمینه مطالعه: کلسیم یکی از مهمترین و فراوانترین املاح بدن است که بسیاری از فعالیتهای فیزیولوژیک از قبیل واکنش‌های آنزیمی، فعالیت رسپتورها، انقباض عضلانی، قدرت انقباضی قلب و... به این عنصر وابسته است. کاهش میزان یون کلسیم باعث کاهش قدرت انقباض قلب و نارسایی قلب می‌شود. **هدف:** هدف از این مطالعه تعیین اثرات کمبود کلسیم جیره به مدت طولانی روی عملکرد قلبی مرغان تخم گذار بر پایه‌ی الکترو و اکوکاردیوگرافی می‌باشد. **روش کار:** نود قطعه پولت مرغ تخم گذار سویه Hyline W36 به مدت ۲۱ هفته نگهداری شدند و با جیره‌های حاوی مقادیر متفاوت کلسیم تغذیه شدند. در سنین ۲۰، ۲۸ و ۳۶ هفته پارامترهای الکتروکاردیوگرافی، اکوکاردیوگرافی و کالبدگشایی بطن چپ تعیین شد. **نتایج:** در هفته‌ی ۳۶ دامنه‌ی موج (S در lead II) به طور معنی داری ($p<0/05$) در گروه II کمبود کلسیم در مقایسه با گروه کنترل افزایش یافت. همچنین بلندی دامنه موج‌های QRS در سنین ۲۸ و ۳۶ هفتگی در دو گروه کمبود کلسیم (aVL, leads II, III, aVR و aVF) وجود داشت ولی تنها در گروه II کمبود کلسیم (leads II و aVF) در مقایسه با گروه کنترل معنی دار ($p<0/05$) بود. قطر دیواره‌ی آزاد بطن چپ در زمان سیستول در سنین ۲۸ و ۳۶ هفتگی در گروه II کمبود کلسیم نسبت به گروه کنترل به طور معنی داری ($p<0/05$) افزایش یافت. قطر دیواره‌ی بین دو بطن در زمان سیستول در سن ۳۶ هفتگی در گروه‌های مواجه شده با کمبود کلسیم نسبت به گروه کنترل به طور معنی داری ($p<0/05$) کاهش یافت. در مطالعات کالبدگشائی نیز در ۳۶ هفتگی، قطر دیواره آزاد بطن چپ در گروه‌های تیمار افزایش معنی داری ($p<0/05$) نسبت به گروه کنترل نشان داد. **نتیجه‌گیری نهایی:** نتایج حاصل از مطالعات الکتروکاردیوگرافی، اکوکاردیوگرافی و کالبدگشائی نشان داد که تغذیه با جیره حاوی کلسیم پایین می‌تواند باعث اختلال در عملکرد قلب مرغان تخم گذار به علت هیپرتروفی قلب شود.

واژه‌های کلیدی: اکوکاردیوگرافی، الکتروکاردیوگرافی، مرغان تخم گذار، کمبود کلسیم.

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