Veterinary Integrative Sciences 2019; 17(3): 245-254

Vet Integr Sci

Veterinary Integrative Sciences

ISSN; 2629-9968 (online)

Website; www.vet.cmu.ac.th/cmvj

Research article

Testosterone deprivation increases tendency to obesity but does not affect cardiac function in dogs

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Abstract

Low testosterone levels in humans is associated with increased risk of cardiovascular diseases (CVD). A previous study reported that low testosterone can induce left ventricular dysfunction and cardiac mitochondrial impairment as well as increase the risk of ischemic heart disease. The effect of testosterone deprivation on cardiac function in dogs, however, has never been investigated. The present study tested the hypothesis that testosterone deprivation induces impairment of cardiac function in healthy castrated male dogs. In this study, twenty-three healthy male dogs were divided into two groups: an intact group (n = 15) and a castrated group which had been castrated at least 12 months (n = 8). Metabolic parameters, blood pressure, and cardiac function using electrocardiography and echocardiography were investigated. We found that although the testosterone level in the castrated group (0.48 ng/mL) was significantly lower than the intact group (5.88 ng/mL) ($P < 0.05$), metabolic parameters, blood pressure, and cardiac function were not different in the two groups. Castrated dogs did, however, have a higher body condition score than intact dogs. These findings suggest that testosterone deprivation in male dogs can induce obesity but that it does not induce impairment of cardiac function.

Keywords: Cardiac function, Castration, Testosterone deprivation

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Article history; *received manuscript: 26 August 2019, revised manuscript: 16 September 2019, accepted manuscript: 9 December 2019, published online: 16 December 2019*

Academic editor; *Korakot Nganvongpanit*

INTRODUCTION

The testosterone hormone is produced mainly by the testes with a smaller amount from the adrenal glands. There are two kinds of testosterone in the blood system: a free form and a bound form. This hormone can bond with androgen receptors and has an impact on the body anatomy and the growth of the testes resulting in secondary sexual characteristics, inducing bone and muscle growth, causing male dogs to increase in size as well as affecting the red blood cell production process (Kausihik et al., 2010; Kloner et al., 2016).

Male dog castration continues to be a popular method of controlling dog populations and reducing aggressiveness and some sexual behaviors (Heidenberger and Unshelm, 1990; Root, 2010). Castration has also been found to reduce the incidence of benign prostatic hyperplasia, prostatitis and testicular tumors (Moulton, 1990; Johnston, 1991). There are, however, reports about several health problems after castration, e.g., obesity, increased risk of bone diseases, thyroid hormone deficiency and diabetes (Panciera, 1994; Root, 2012).

Studies of testosterone in men found levels to be associated with a risk of hypogonadism which increases with age. Studies in males with a low testosterone level have reported symptoms of tiredness and pain, low sexual desire, low sexual ability, unstable emotions, reduced muscle mass, high cholesterol, low bone density and increased risk of cardiovascular diseases (CVD) (Jones, 2010). Low testosterone in men can also result in obesity, diabetes, hypertension and high blood pressure as well as high cholesterol levels (Muraleedharan and Jones, 2010; Kelly and Jones, 2015; Mohammed et al., 2018). The testosterone hormone plays a multifaceted role in metabolism; however, the effect of testosterone deprivation on cardiac function and metabolic balance in castrated male dogs has never been investigated. This study tested hypotheses regarding testosterone deprivation inducing impairment of cardiac function and metabolic balance in healthy castrated dogs.

MATERIALS and METHODS

This experiment has been approved by the Committee on Ethics on Animal Use for Scientific Experimentation, Faculty of Veterinary Medicine, Chiang Mai University (S10/2559).

Population and study designs

Twenty-three healthy male dogs age 2-8 years old and with a body weight of less than 20 kg were enrolled in the study. The dogs were divided into two groups. Group 1 was composed of 8 dogs which had been castrated for at least 12 months. Group 2 was composed of 15 intact dogs. All dogs were given physical and blood test examinations; standard physical examination and baseline data including body condition score (BSC) (Baldwin et al., 2010) were recorded. Dogs were excluded from the study if they were found

to have cardiac diseases as determined by echocardiography. Dogs were also excluded if they showed any abnormalities in their hematology and/or blood biochemistry profiles compatible with systemic diseases, inflammation/infection, or neoplastic diseases.

Blood sample collection

All dogs were fasted for at least 12 hours prior to blood sample collection which was done by venipuncture of the cephalic or saphenous vein during the period 09.00-12.00 AM. Five milliliters of blood were collected from each dog and divided into an EDTA tube (1 mL) for hematology testing and a heparin tube (2 mL) for biochemistry profiles testing. The remaining 2 ml. of blood were centrifuged at 1,500 rpm for 10 minutes to obtain serum which were kept at -80^{o}C until examined for cholesterol and hormone levels.

Electrocardiographic measurement

The dogs were placed in a right lateral recumbent position and the 6 ECG leads (I, II, III, aVR, aVL and aVF) were measured (EDAN VE-300, China) at a paper speed of 25 mm/second and a sensitivity of 1 cm=1 mV. The MEA determined by examining the QRS complexes in lead I and lead aVF (Edwards, 1978).

Systolic blood pressure measurement

Systolic blood pressure (SBP) measurement was done using the Doppler flow technique with a model BF2 (Parks Medical Electrics Company, USA). The dogs were placed in a right lateral recumbent position. A cuff was placed on the mid-antebrachium; the cuff with a width equal to 40% of the circumference of the limb was attached. Blood pressure was measured five times, and the average of those values was recorded.

Echocardiography measurements

Echocardiography (ALOKA® ProSound SSD-3500SX, USA) was performed with a frequency of 3.5-7.5 Hz by measuring (1) the left ventricular free wall (LVPW), the interventricular septum (IVS), the left ventricular internal dimension (LVID) on the systole and diastole, (2) the left atrial (LA) size and the size of the aorta (AO) to find the LA/AO ratio, and (3) the percentage of fractional shortening (%FS). The percentage of ejection fraction (%EF) was automatically calculated by the program. Standard transthoracic right parasternal views were obtained for echocardiographic evaluation and measurement (Thomas et al., 1993). The LV wall thickness, dimension and systolic were evaluated by 2D-guided M-mode at the level just above the head of papillary muscle. The FS and EF values were calculated automatically using Teicholz formula and ultrasound equipment software. The left atrial to aortic root ratio (LA:AO ratio) was measured using LA and AO from the right parasternal short axis view, as previously described (Rishniw & Erb, 2000).

Testosterone level measurement

This study used a double-antibody enzyme immunoassay (EIA). Anti-rabbit IgG (10 µg/mL; catalogue no. A009; Arbor Assays, Ann Arbor, MI, USA) was used as a secondary antibody. Serum testosterone level was compared with a testosterone standard (T3006; Sigma-Aldrich, Poole, UK) using a microplate reader at an optical density of 405 nM (TECAN Sunrise™). All serum samples were re-analyzed if the coefficient of variation was greater than 10 percent, i.e., the intra-assay coefficient of variation was less than 10 percent. The concentration level of testosterone was expressed as ng/mL.

Lipoprotein-cholesterol measurements

Cholesterol and triglycerides were measured by enzymatic methods (Giesse Diagnostics S.N.C.). High-density lipoprotein-cholesterol (HDL-c) and low-density lipoprotein-cholesterol (LDL-c) was calculated using the Friedewald formula LDL = TC - HDL - TG/5.0 (mg/dL), whereas very low-density lipoprotein-cholesterol (VLDL-c) was determined by the formula VLDL-c = TG/5.0 (Friedewald et al., 1972).

Statistical analysis

Blood levels of testosterone and cholesterol as well as cardiac parameters measured by echocardiography are displayed as mean and standard deviation (mean ± SD). The Student's t-test was used to calculate the difference of means of various parameters between intact dogs and castrated dogs using R statistical software. The level of statistical significance was set at $\alpha = 0.05$.

RESULTS

Levels of testosterone hormone

The level of testosterone hormone in the two groups of dogs were different significantly ($P<0.05$). The mean testosterone hormone level in the intact dogs was 5.88 ng/mL, and in the castrated group was 0.48 ng/mL (Table 1)

Table 1 Mean testosterone levels in intact and castrated dogs.

	Intact (n = 15)	Castrated (n = 8)	P - value
Testosterone level (ng/mL)	5.88 ± 5.46	0.48 ± 0.37	P < 0.05

Reduced testosterone in the castrated dogs resulted in increased body condition scores but not affects in systolic blood pressure

The average ages of the intact group and the castrated group were 51.14 months and 66.00 months, respectively. The average weight of the intact group was 7.64 ± 5.38 kg., and that of the castrated group was 7.51 ± 1.97 kg. Neither the age nor the weight of the two groups differed significantly. However, the body condition score of the castrated group was significantly higher than that of the intact group: 3.75 ± 0.46 and 3.10 ± 0.21, respectively ($P<0.05$) (Table 2). The mean systolic blood pressure in intact group was 158.14 ± 35.85 mmHg and castrated group was 164.00 ± 43.52 mmHg. Blood pressure differences did not reached the level of statistical significance.

Table 2 Mean age, bodyweight, body condition score (BCS) and systolic blood pressure in intact and castrated dogs.

	Intact (n = 15)	Castrated (n = 8)	P - value
Age (months)	51.14 ± 28.72	66.00 ± 37.40	P > 0.05
Body weight (kg)	7.64 ± 5.38	7.51 ± 1.97	P > 0.05
BCS	3.10 ± 0.21	3.75 ± 0.46	P < 0.05
Systolic blood pressure (mmHg)	158.14 ± 35.85	164.00 ± 43.52	P > 0.05

Reduced testosterone levels in castrated dogs did not cause changes in electrocardiograms

The electrocardiograms of both groups, including P wave, PR interval, RII amplitude, QRS duration, T wave, QT interval, mean electrical axis and ST segment, are shown in Table 3. The ECG parameters were no significant difference between the two groups.

Table 3 Electrocardiographic values of intact and castrated dogs.

	Intact (n = 15)	Castrated (n = 8)	P – value
Heart rate (bpm)	111 ± 10.77	115 ± 10.95	P > 0.05
P wave (mV)	0.23 ± 0.07	0.28 ± 0.08	P > 0.05
P duration (sec)	0.04 ± 0.005	0.04 ± 0.00	P > 0.05
PR interval (sec)	0.10 ± 0.03	0.08 ± 0.007	P > 0.05
RII (mV)	1.63 ± 0.74	2.02 ± 0.34	P > 0.05
QRS duration (sec)	0.04 ± 0.01	0.04 ± 0.007	P > 0.05
T wave (mV)	0.27± 0.14	0.30 ± 0.10	P > 0.05
QT interval (sec)	0.18 ± 0.02	0.19 ± 0.01	P > 0.05
Mean electrical axis (degree)	68.71 ± 41.10	71.50 ± 14.32	P > 0.05

Reduced testosterone in castrated dogs did not reduce cardiac function

Echocardiography of both groups measuring thickness and width of the left ventricle and the size of the left atrium, ejection fraction and fractional shortening were not significantly different (Table 4).

Table 4 Echocardiographic values of intact and castrated dogs.

	Intact (n = 15)	Castrated (n = 8)	P – value
IVSd (cm)	0.68 ± 0.15	0.72± 0.06	P > 0.05
LVIDd (cm)	2.52± 0.63	2.27 ± 0.36	P > 0.05
LVPWd (cm)	0.73± 0.16	0.73 ± 0.08	P > 0.05
IVSs (cm)	0.97 ± 0.24	1.01± 0.06	P > 0.05
LVIDs (cm)	1.53± 0.45	1.28 ± 0.26	P > 0.05
LVPWs (cm)	1.09 ± 0.25	1.13 ± 0.15	P > 0.05
EDV (mL)	24.83 ± 14.30	18.13 ± 7.23	P > 0.05
ESV (mL)	7.50 ± 5.04	4.35 ± 2.32	P > 0.05
SV (mL)	17.42 ± 10.07	13.88 ± 5.17	P > 0.05
EF (%)	71.43 ± 9.78	76.46 ± 5.42	P > 0.05
FS (%)	39.55± 7.61	43.20 ± 4.86	P > 0.05
LA (cm)	1.80 ± 0.45	1.73± 0.21	P > 0.05
AO (cm)	1.58 ± 0.40	1.45± 0.16	P > 0.05
LA:AO	1.15 ± 0.10	1.21 ± 0.09	P > 0.05

(IVSd = Interventricular septal end diastole, LVIDd = Left ventricular internal diameter end diastole, LVPWd = Left ventricular posterior wall end diastole, IVSs = Interventricular septal end systole, LVIDs = Left ventricular internal diameter end systole, LVPWs = Left ventricular posterior wall end systole, EDV = End-diastolic volume, ESV = End-systolic volume, SV = Stroke volume, EF = Ejection fraction, FS = Fractional shortening, LA = Left atrial diameter, AO = Aortic root diameter , LA : AO = Left atrial diameter to aortic root diameter ratio)

Reduced testosterone in castrated dogs did not increase blood cholesterol levels

Measurements of cholesterol levels in both groups, including total cholesterol, triglyceride, high-density lipoprotein-cholesterol, low-density lipoprotein-cholesterol and very low-density lipoprotein-cholesterol, showed no significant difference between the two groups. The mean values of lipid profiles are shown in Table 5.

Table 4. Echocardiographic values of intact and castrated dogs.

	Intact (n = 15)	Castrated (n = 8)	P – value
TC	174.28 ± 50.76	180.12 ± 39.22	P > 0.05
TG	64.21 ± 32.75	72.00 ± 41.79	P > 0.05
HDL	132.21 ± 35.77	157.25 ± 23.47	P > 0.05
LDL	31.94 ± 32.94	30.72 ± 26.67	P > 0.05
VLDL	12.84 ± 6.55	14.40 ± 8.35	P > 0.05

(TC = Total cholesterol, TG = Triglyceride, HDL = High-density lipoprotein-cholesterol, LDL = Low-density lipoprotein-cholesterol, VLDL = Very low-density lipoprotein- cholesterol)

DISCUSSION

Testosterone levels in castrated dogs decreased significantly compared to that of intact dogs ($P < 0.05$) because removing the testicles, or castration, destroys a dog's major source of testosterone production (Kaushik et al., 2010; Kloner et al., 2016). This study found that decreased testosterone levels in castrated dogs caused an increase in body condition score (BCS). However, the lower level of testosterone did not result in a change in blood pressure, size of the heart, electrocardiogram, cardiac function or level of cholesterol.

The statistically significant of higher BCS in the castrated group showed that castration induces a tendency toward obesity as BCS is an effective indicator of obesity in dogs (White et al., 2011; Bjornvad et al., 2019). The castrated dogs accumulated additional body fat causing the BCS increase (Kobayashi et al., 2014). Moreover, the castrated dogs also ate more and spent longer periods resting and relaxing and were less active (Heidenberger and Unshelm, 1990). These factors induced a propensity to become fat after castration which could potentially result in cardiac and circulatory system problems. Although this study found that testosterone deprivation of at least 12 months after castration caused the BCS to increase, it did not affect either the cardiac system or the circulatory system in any way.

The finding that testosterone deprivation did not affect the blood pressure of dogs castrated for a period of 12 months or longer differs from the human's study which found that men with naturally lower testosterone had higher blood pressure than men with normal testosterone levels (Fogari et al., 2002; Fogari et al., 2005). Similarly, an experiment with rats showed increased blood pressure 12 weeks after castration due to increased cholesterol and diastolic dysfunction (Pongkan et al., 2016a; Pongkan et al., 2016b). The difference in the blood pressure findings in this study and the human study might be due to the short duration of testosterone deprivation which might not yet have affected cardiac function or other related parameters. Measurement of cardiac function using M-mode echocardiography in this study showed normal cardiac wall condition and cardiac ventricular dimensions. The strain and strain rate might be superior than M-mode to detect systolic function. Moreover, we measured

only the systolic function parameters but was not diastolic function parameters which might be affected by the hormone deficiency. Additionally, no difference was found in the level of cholesterol between the two groups of dogs. Differences in duration of testosterone deficiency in the dog and rat models perhaps also due to the shorter life expectancy of rats may indicate that changes in cardiac function in rats cannot be used to predict abnormal functions in dogs.

Electrocardiogram results showed no difference between groups, which contradicts results of Fulop et al. in 2006 which found the QT interval in castrated dogs to be significantly longer than in intact dogs. However, HR is the main factor which have inversely relationship with the QT interval so the measured QT interval would be corrected to a less heart rate dependent value (QTc interval). A long QT interval could relate to an arrhythmia (Fulop et al., 2006). In addition, an experiment on ischemic and reperfusion injury rats with testosterone deprivation found an increased risk of arrhythmia (Pongkan et al., 2015; Pongkan et al., 2016a). and the possible mechanism could be due to the decreasing of gap junction connexin-43 (Cx43) protein in testosterone deprived group, which demonstrated that the phosphorylation Cx43 per total Cx43 ratio (P-Cx43/T-Cx43 ratio) was decreased in testosterone deprived group (Pongkan et al., 2015, Pongkan et al., 2016a). Therefore, these finding could be resulting in increased the repolarization time and lead to prolong QT interval. However, we did not find the differences of cardiac arrhythmia among groups in this clinical study, whereas the differences in this study could be due to differences in study designs and models. For example, the present study compared electrocardiograms of castrated and non-castrated dogs, whereas the previous studies compared ECG indicators before and after castration in the same dogs. Therefore, the further large clinical study populations about the effects of testosterone deprivation on the cardiac performance of dogs is still needed. A long QT interval could relate to an arrhythmia (Fulop et al., 2006). Additionally, an experiment on ischemic and reperfusion injury rats with testosterone deprivation found an increased risk of arrhythmia (Pongkan et al., 2015; Pongkan et al., 2016a). Differences in study results could be due to differences in study designs and models. For example, the present study compared electrocardiograms of castrated and non-castrated dogs, whereas the previous studies compared ECG indicators before and after castration in the same dogs. Further clinical study of the effects of testosterone deprivation on the cardiac condition of dogs is needed.

The limitation of this study is that although there was no difference in the values obtained from the electrocardiograms and the echocardiogram between the two groups of dogs, this could be because the period of testosterone deprivation was too short to cause cardiac dysfunction to appear in the measured parameters. Further clinical study should be investigated in the large population and study in the time course and/or might be design in the prospective study to provide the high clinical impact. The age-match control design might be used for the further investigation to minimize the heterogeneous nature of the study population. Another limitation was that this was a clinical study, making it possible to control other post-castration factors that could potentially affect cardiac function, e.g., food and care. Finally, we did not have breed control which might affect the heart function and also the obesity in dogs.

CONCLUSION

Testosterone deprivation in male dogs for 12 months or longer after castration increases tendency to obesity, but does not impair cardiac function or cholesterol level.

CONFLICT of INTEREST

The authors have no conflicts of interest to disclose.

ACKNOWLEDGEMENTS

The authors would like to thank the staff of the Hormone Laboratory, FVM-CMU for their assistance with the laboratory work. We would also like to thank Dr. Therdsak Yano for statistical consultation.

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How to cite this article;

Chavalit Boonyapakorn, Tirayuth Pinsuwan, Thanakorn Chumpuchai and Wanpitak Pongkan. Testosterone deprivation increases tendency to obesity but does not affect cardiac function in dogs. Veterinary Integrative Sciences. 2019; 17(3): 245-254