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Effect of coenzyme q10 supplementation on total antioxidant status and lipid peroxides levels in dogs with chronic valvular heart disease*

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Abstract

Oxidative stress management has been found to have beneficial effects in the management of several disease conditions. A study was conducted to evaluate the efficacy of Coenzyme Q10 supplementation on the total antioxidant status and lipid peroxides levels in dogs with chronic valvular heart disease. Total antioxidant status was measured by Ferric Reducing Antioxidant Power assay and level of lipid peroxides in serum was determined by estimating malonaldialdehyde level. Ten dogs with chronic chronic valvular heart disease were administered with coenzyme Q10 @ 45mg q12h orally for 60 days along with the treatment for management of valvular heart disease. A significant decline in Ferric Reducing Antioxidant Power value was recorded at the end of the study. The malonaldialdehyde levels declined in animals with chronic valvular heart disease by day 60, though reduction was not statistically significant. The administration of Coenzyme Q10 in chronic valvular heart disease may be beneficial in improving the quality of life of the patient.

Keywords: *Dog, Coenzyme Q10, Chronic valvular heart disease, Oxidative stress*

Chronic valvular disease is the most common acquired heart disease in dogs. This disorder is considered to be a frequent cause of cardiac mortality in dogs (Haggstrom *et al*., 2004). Valve leaflets will undergo consequent before degeneration, deformation and thickening along with other senile changes of the body. Valvular affections reduce normal cardiac functions which in turn results in cardiac failure. Age related oxidative stress directly affects the cardiovascular system and it is assessed by measuring different biomarkers of oxidative stress. Coenzyme Q10, an antioxidant, is a cellular membrane stabilizer and free radical scavenger that suppresses the formation of reactive oxygen species during lipid peroxidation. Investigations on the role of antioxidant molecules in

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the treatment of heart diseases in dogs could provide new therapeutic options for veterinary clinicians.

Materials and methods

Dogs brought to the University Veterinary Hospital, Kokkalai and Teaching Veterinary Clinical Complex, Mannuthy with cardiac ailments were screened for the study. Based on a detailed clinical examination along with electrocardiographic, radiographic and echocardiographic studies, chronic valvular heart disease was confirmed in 10 dogs. The animals were treated with furosemide @ 2mg/kg bodyweight q12h, enalapril @ 0.5mg/kg bodyweight q12h, pimobendan @ 0.25mg/kg body weight q12h orally and coenzyme Q10 @ 45mg q12h orally. Six apparently healthy adult dogs were taken as the control group. Oxidative stress was evaluated with the assessment of total antioxidant status and lipid peroxidation level on 0th, 30th and 60th day of treatment.

Total antioxidant status (TAS) was measured by Ferric Reducing Antioxidant Power (FRAP) assay (Benzie and Strain, 1996). Level of lipid peroxides in serum was determined by the method of Yagi (1984) by estimating the malonaldialdehyde (MDA) level. Statistical analysis was done by one way ANOVA and paired't' test using SPSS version 24.0.

Results and discussion

Total antioxidant status of diseased animals was significantly high ($p \leq 0.01$) when compared to healthy animals on 0th and 30th day of treatment. Hetyey *et al.* (2007) made similar observations and reported a significantly higher FRAP value in dogs with dilated cardiomyopathy and mitral endocardiosis when compared to healthy animals. Increased FRAP value may be due to a higher level of uric acid as it contributes to 60 per cent of the fresh human blood plasma (Benzie and Strain, 1996). High uric acid concentration was observed in heart failure patients (Khan *et al*., 2017). In heart failure, increased production, reduced excretion or both will result in elevated uric acid levels. Increased abundance and activity of xanthine oxidase (XO) which is a contributor of reactive oxygen species in the failing heart coupled with elevated XO substrates and an increased conversion of xanthine dehydrogenase to XO are possible contributors of an elevated uric acid production (Hare *et al.,* 2003; Doehner and Anker, 2005).

From 0th to 60th day of treatment with coenzyme Q10, a significant decline ($p \leq 0.01$) in FRAP value from 1599.43±132.28 to 910.17±118.17µM /L was recorded. Maheshwari *et al.* (2014) and Sharma *et al.* (2016) reported on the ability of coenzyme Q10 to reduce uric acid levels. So the reduction in FRAP value after treatment with coenzyme Q10 may be attributed to the decrease in uric acid level or to reduced oxidative stress. Therefore, it can be concluded that the supplementation of coenzyme Q10 could reduce the oxidative stress associated with chronic valvular heart disease in dogs.

Serum MDA levels of diseased and healthy animals did not reveal any significant variations on the 0th, 30th and 60th day of treatment. Freeman *et al.* (2005) and Reimann *et al*. (2017) reported similar findings that there was no association between serum MDA levels in clinical stages of mitral valve disease in dogs. The mean value of lipid peroxides was high in dogs with valvular heart disease (4.93±0.64nM/mL) when compared to healthy animals (2.85±0.12nM/mL) on the initial day of treatment. According to Prasad *et al*. (1996), high level of MDA in cardiac tissues may exist during heart diseases and that need not be reflected in serum.

The MDA levels decreased to 2.93±0.74 from 4.93±0.64 nM / mL on 60th day of treatment with coenzyme Q10 even though no statistical significance was observed In conditions like coronary artery disease, diabetes and hepatocellular carcinoma in humans, a reduction in the MDA levels have been recorded after coenzyme Q10 supplementation (Lee *et al.*, 2011; Moazen*et al.*, 2015; Liu *et al*., 2016). Litarru and Tiano (2007) reported that coenzyme Q10 had the ability to prevent the peroxidation of lipids present in cell membrane and circulation. A significant reduction in MDA levels in the coenzyme Q10 supplemented animals could be expected if administered for a longer period of time.

The effect of coenzyme Q10 supplementation on total antioxidant status and lipid peroxides are presented in the table below.

	Total antioxidant status (µM/L)			Lipid peroxides (nM/mL)		
	0th day	30th day	60th day	0th day	30th day	0th day
Diseased animals	1599.43±132.28^{aA}	1408.71±110.37^{aA}	910.17±118.17^{bB}	4.93±0.64	3.45±1.02	2.93±0.74
Healthy animals	824.78±75.29^{b}	824.78±75.29^{b}	824.78±75.29^{b}	2.85±0.12	2.85±0.12	2.85±0.12

Mean±S.E. bearing different small letter as superscripts differ significantly within column (between diseased and healthy animals) at 1% level (p≤0.01)

Mean±S.E. bearing different capital letter as superscripts differ significantly within rows for each parameter at 1% level (p≤0.01)

Based on the results of the present study, supplementation of coenzyme Q10 can be considered in the management of oxidative stress associated with chronic valvular heart disease in dogs. The quality of life of the patient could be improved with the administration of coenzyme Q10.

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