Effect of oral administration of pimobendan in cats with heart failure

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Objective—To determine the effect of PO administration of pimobendan on clinical and echocardiographic variables and survival time in cats with heart failure characterized by ventricular systolic dysfunction.

Design—Retrospective cohort study.

Animals—27 client-owned cats (16 male and 11 female) with heart failure, treated with pimobendan (mean ± SD dosage, 0.26 ± 0.08 mg/kg [0.118 ± 0.036 mg/lb], PO, q 12 h).

Procedures—Information on medical history, laboratory results, diagnostic imaging findings, treatments received, and survival time were obtained from medical records of cats that received pimobendan because of cardiac disease. When possible, additional follow-up information was obtained through telephone interviews with referring veterinarians and owners.

Results—The mean ± SD age of all 27 cats was 8.9 ± 5.2 years. All cats had received several cardiac medications. Types of heart disease represented included unclassified cardiomyopathy (CM; n = 11 [41%]), dilated CM (8 [30%]), arrhythmogenic right ventricular CM (4 [15%]), congenital heart disease (3 [11%]), and hypertrophic CM with regional hypokinesis (1 [4%]). All cats had ventricular systolic dysfunction. One cat with systolic anterior motion of the mitral valve became severely hypotensive after initial administration of pimobendan and was excluded from the survival analysis. Median survival time was 167 days (95% confidence interval, 32 to 339 days).

Conclusions and Clinical Relevance—Pimobendan appeared to be well tolerated in cats with heart failure characterized by ventricular systolic dysfunction of various etiologies. Cats with systolic anterior motion of the mitral valve may develop systemic hypotension when treated with pimobendan. Additional studies are needed to establish dosages for pimobendan and its effects before it can be recommended for treatment of cats with CHF. (*J Am Vet Med Assoc* 2012;241:89–94)

Pimobendan is predominantly an inodilator, with additional ancillary properties including endothelium-dependent vasodilatation, enhanced myocardial relaxation (positive lusiotropy), and antiplatelet effects. Additionally, pimobendan may have other salutary effects in patients with HF. Oral administration of pimobendan (0.25 to 0.30 mg/kg [0.114 to 0.140 mg/lb], q 12 h) prolongs survival time and reduces signs of illness in dogs with CHF secondary to myxomatous mitral valve disease and dilated CM and has been licensed in the United States for this indication since 2007.[1–3]

In cats, heart disease leading to CHF is characterized predominantly by diastolic dysfunction.[4] Chronic treatment with orally administered drugs has conventionally been used to control clinical signs associated with CHF (eg, furosemide with or without an angiotensin-converting enzyme inhibitor), arterial thromboembolism prophylaxis (eg, clopidogrel with or without acetylsalicylic acid), and medications that may directly or indirectly improve ventricular relaxation (eg, diltiazem or a β-adrenoreceptor antagonist). However, several heart diseases are characterized by echocardiographic evidence of systolic ventricular (myocardial) dysfunction. These diseases include many forms of unclassified CM, dilated CM, arrhythmogenic right ventricular CM, some forms of hypertrophic CM, and some severe forms of congenital and other acquired heart disease.[4] In addition, cats with CHF due to systolic (myocardial) failure have a poor long-term prognosis, with a report-

Abbreviations

ALT	Alanine aminotransferase
CHF	Congestive heart failure
CI	Confidence interval
CM	Cardiomyopathy
HF	Heart failure
HR	Hazard ratio
IQR	Interquartile range
SAM	Systolic anterior motion of the mitral valve
SAP	Systolic arterial blood pressure

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ed median survival time of 13 days with conventional treatment.[a] The objective of the study reported here was to determine the effect of PO administration of pimobendan on clinical and echocardiographic variables and survival time in cats with HF characterized by ventricular systolic dysfunction.

Materials and Methods

Case selection—The electronic medical records database of Texas A&M University Veterinary Medical Teaching Hospital was searched for all feline patients for which pimobendan[b] had been dispensed between October 1, 2004, and October 1, 2009. To be eligible for inclusion in the initial descriptive analysis, cats were required to have had at least 1 dose of pimobendan administered and not have been euthanized because of a poor prognosis despite clinical improvement within the first 24 hours following pimobendan treatment initiation. Cats that died spontaneously or were euthanized because of progressive clinical signs were included only when ≥ 1 dose of pimobendan was administered.

For inclusion in the follow-up portion of the study, cats were required to have received > 1 dose of pimobendan. Most data were collected from the medical records and, when possible, additional follow-up data were collected through telephone interviews with referring veterinarians and owners.

Medical records review—The following data were obtained from the medical history of each cat that had been recorded prior to initiating pimobendan treatment: signalment, historical information obtained from the owner or referring veterinarian, results of physical examination, results of hematologic and serum biochemical analyses (including whole blood taurine concentration in some cats), and results of diagnostic tests and imaging (indirect systemic arterial blood pressure measurement by use of Doppler sphygmomanometry, thoracic radiography, echocardiography, and ECG). Additional information obtained from the medical records included medications administered prior to pimobendan treatment and dose of pimobendan administered.

Variables reevaluated for the first time after pimobendan treatment began were recorded as long as that reevaluation occurred within 60 days after pimobendan treatment initiation. In particular, attempts were made to record results of repeated echocardiographic assessments that were obtained within 1 to 60 days after treatment started. Any adverse events and date and cause of death or reason for euthanasia were also recorded.

Ventricular systolic dysfunction was considered to exist when ≥ 1 of the following criteria were met: a low fractional shortening percentage (ie, < 30%) evaluated from 2-D or M-mode images, a high left ventricular internal dimension in systole (ie, > 1.12 mm) evaluated from 2-D or M-mode images, the presence of regional hypokinesis believed to impair left ventricular ejection fraction, or severe dilatation of the right ventricle and right atrium consistent with a diagnosis of arrhythmogenic right ventricular CM.

Statistical analysis—The Shapiro-Wilk test was used to evaluate all continuous variables for normal distribution of values. Normally distributed values are summarized as mean ± SD, and nonnormally distributed data are summarized as median (IQR). Comparisons were made between initial and follow-up data with the paired *t* test for means, Wilcoxon matched-pairs signed rank test for medians, or an exact McNemar test for proportions, as appropriate.

Parametric survival models with a Gompertz distribution were used for univariate and multivariate survival estimates. The potential effect on survival time of all variables before pimobendan treatment was evaluated initially in a univariate analysis. For each medication other than pimobendan, a dichotomous variable was created (administered vs not administered). For pimobendan, dosage (mg/kg/d) was used as the variable. More than 50 univariate models were fit: one for each of various examination findings, echocardiographic findings, the 5 drug treatments, and 5 others (age, body weight, sex, pure breed [yes vs no], and euthanasia [yes vs no]). Multivariate model selection was performed with a backward stepwise approach. All statistical analyses were performed with a commercially available software package.[c] Values of $P < 0.05$ were considered significant.

Results

Animals—Thirty cats were identified for the initial descriptive analysis. One of these cats was excluded because administration of pimobendan was not documented, the drug was not prescribed by veterinarians of the cardiology service, and a cardiac indication could not be identified. Two additional cats were excluded because, despite clinical improvement, they were euthanized because of a poor prognosis within 24 hours after pimobendan administration began. Therefore, 27 cats were included in the initial analysis.

An additional cat had pimobendan treatment discontinued after 1 dose because of an adverse event and was thus eliminated from the follow-up group, leaving 26 cats for the follow-up portion of the study. The excluded cat was a 4.7-year-old spayed female domestic variety (body weight, 4.5 kg [9.9 lb]) with underlying congenital mitral valve dysplasia, ventricular septal defect, and SAM, leading to severe left atrial and ventricular enlargement and systolic dysfunction. Results of clinicopathologic testing indicated serum concentrations of creatinine and urea nitrogen were high at 47 mg/dL (reference limits, 19 to 33 mg/dL) and 3.2 mg/dL (reference limits, 0.8 to 1.8 mg/dL), respectively. Blood total solids concentration, PCV, and serum ALT activity were within reference limits (6.0 to 8.0 g/dL, 29% to 45%, and 26 to 84 U/L, respectively). The ratio of the left atrial dimension to the aortic diameter calculated from a short axis M-mode view was 2.3 (reference limit, < 1.8), and the left ventricular internal dimensions from M-mode short axis view were high in diastole at 2.2 cm (reference limit, < 2.1 cm) and in systole at 1.6 cm (reference limit, < 1.1 cm). Fractional shortening was low at 29% (reference limits, 35% to 67%).[5] Although SAM was identified, the transaortic velocities were within reference limits.

The cat had been initially evaluated because of tachypnea, dyspnea, and signs of lethargy consistent with decompensation of previously diagnosed CHF, de-

spite treatment with furosemide (4.4 mg/kg [2 mg/lb], PO, q 12 h), enalapril (0.4 mg/kg [0.18 mg/lb], PO, q 24 h), clopidogrel (18.75 mg, PO, q 24 h), and spironolactone (1.3 mg/kg [0.59 mg/lb], PO, q 24 h). A grade 3/6 left parasternal systolic heart murmur and gallop rhythm were detected, with a heart rate of 168 beats/min and a respiratory rate of 90 breaths/min. The cat's SAP was low at 97 mm Hg. Contemporaneous thoracic radiography revealed pulmonary edema and pleural effusion. One dose of pimobendan had been administered PO in the hospital (0.14 mg/kg [0.064 mg/lb]), and the cat's heart rate and SAP were reevaluated approximately 4 hours later. At that point, the SAP had decreased to 60 mm Hg and the heart rate had increased to 240 beats/min. Pimobendan was subsequently discontinued. The cat was euthanized because of CHF that was refractory to treatment 169 days after its last visit.

The mean ± SD body weight of the 27 cats in the initial analysis was 4.1 ± 1.3 kg (9.0 ± 2.9 lb). Sixteen (59%) were males, and the mean age of all 27 cats was 8.9 ± 5.2 years. Most cats (18/27 [67%]) were of domestic breeds. Purebred cats included 2 (7%) each of Burmese, Sphinx, and Maine Coon and 1 (4%) each of Siamese, Birman, and Persian.

Initial characteristics—Clinical signs in the 27 cats before treatment began included dyspnea or tachypnea (n = 21 [78%]), anorexia (16 [59%]), lethargy (14 [52%]), physical collapse or syncope (5 [19%]), and ascites (2 [7%]). Twenty-six (96%) cats had at least 1 clinical sign reported, with most having > 1. Cardiac medications prior to pimobendan treatment included enalapril or benazepril (mean dosage, 0.39 ± 0.16 mg/kg [0.177 ± 0.072 mg/lb], q 24 h; n = 20), furosemide (1.9 mg/kg/d [0.86 mg/lb/d]; 20), and clopidogrel (18.75 mg, q 24 h; 14). Other cardiac medications were used for specific indications, including diltiazem, atenolol, spironolactone, sotalol, and digoxin.

In cats for which relevant data were recorded, the mean initial heart rate before pimobendan treatment was 197 ± 29 beats/min (n = 27 cats) and median respiratory rate was 60 breaths/min (IQR, 44 to 76 breaths/min; n = 25). Median SAP was 110.5 mm Hg (IQR, 88 to 135 mm Hg; n = 22). Systolic arterial blood pressure was low (< 110 mm Hg) in 9 of 22 (41%) cats, and no cat was hypertensive. The PCV and blood total solids concentration were less than the lower reference limit of 29% and 6.0 g/dL in 4 of 26 (15%) and 1 of 26 (4%) cats, respectively. Blood total solids concentrations were high (> 8.0 g/dL) in 6 of 26 (23%) cats, and a concurrently elevated PCV (> 45%) was detected in 2 of these 6 cats. A high PCV was also detected in 1 other cat. Serum urea nitrogen and creatinine concentrations were higher than the upper reference limits in 8 of 27 (30%) and 15 of 27 (55.6%) cats, respectively. Serum ALT activity was high in 13 of 25 (52%) cats.

Seven of 27 (26%) cats had systolic murmurs ranging in grade from 2 to 5/6, and 13 (48%) had a gallop rhythm. The cat with a grade 5/6 murmur had a congenital endocardial cushion defect. Arrhythmias as confirmed through physical examination, echocardiography, and ECG were recorded for 9 (33%) cats and were categorized as ventricular in 5 of 7. Atrial fibrillation was identified in 2 (7%) cats, and unclassified arrhythmias were detected in 2 (7%) other cats. Arrhythmias identified through physical examination or echocardiography only were not classified.

Radiography revealed cardiomegaly as judged on the basis of a vertebral heart size in the right lateral projection > 8.1 in 21 of 22 (95%) cats.[6] There was radiographic evidence supportive of CHF in 20 of 26 (77%), with 11 of 26 (42%) having evidence of pulmonary edema and 13 of 26 (50%) having pleural effusion or venous congestion. The underlying etiology of HF as judged on the basis of echocardiography was unclassified CM in 11 of 27 (41%) cats, dilated CM in 8 (30%), arrhythmogenic right ventricular CM in 4 (15%), congenital in 3 (11%), and hypertrophic CM in 1 (4%). The 1 cat with hypertrophic CM had regional areas of both hypokinesis and hyperkinesis but overall was considered to have a low left ventricular ejection fraction despite a fractional shortening (determined in short-axis by M-mode) of 85%.

The 3 cats with congenital disease had an endocardial cushion defect (10.9 years of age at the time of diagnosis), ventricular septal defect and mitral valve dysplasia with SAM and resultant outflow tract obstruction (4.7 years), or tricuspid valve dysplasia with pulmonary hypertension and right-to-left shunting patent ductus arteriosus (1.0 years). The congenital heart disease was severe, and each affected cat was considered to have ventricular systolic dysfunction.

Whole blood taurine concentration was evaluated in 4 of the 8 cats with dilated CM. Three of the 4 cats had a high concentration (278, 408, and 1,403 nmol/mL; reference interval, 80 to 120 nmol/mL), and 1 cat had a low concentration (30 nmol/mL). Three cats with dilated CM were receiving a taurine supplement; 2 cats had had taurine prescribed empirically (no whole blood evaluation) and the third was the cat with the low whole blood taurine concentration. This third cat had no myocardial recovery as determined on the basis of echocardiography that was repeated 30 days after supplement initiation.

Spontaneous echocardiographic contrast (smoke), which is a swirling pattern of increased blood flow echogenicity, was detected in the left atrium of 4 of 27 (15%) cats. A thrombus was seen or suspected in 6 (22%), pericardial effusion without tamponade in 10 (37%), subjectively assessed right atrial enlargement in 10 (37%), and right ventricular enlargement in 7 (27%).

Follow-up and survival analysis—Twenty-six cats were included in the follow-up and survival analysis. The administered pimobendan dosage was 0.26 ± 0.08 mg/kg every 12 hours. Examination findings before and after pimobendan treatment began were summarized (**Table 1**). Results of the paired tests to compare initial with follow-up findings suggested that most variables evaluated during follow-up examinations were not significantly different. There was a significant decrease in the percentage of cats with dyspnea ($P = 0.016$) and anorexia ($P = 0.004$) at follow-up, compared with before pimobendan treatment.

Results of the paired tests to compare initial echocardiographic findings with follow-up findings were summarized (**Table 2**). There was no significant change in any variable evaluated. Although the median fraction-

Table 1—Physical and clinicopathologic findings in 26 cats with CHF before and after initiation of pimobendan treatment (0.25 to 0.30 mg/kg [0.114 to 0.140 mg/lb], PO, q 12 h).

Variable	No. of cats	Before treatment	After treatment	*P* value
SAP (mm Hg)	20	110 (88–135)	120 (104–125)	0.140
Heart rate (beats/min)	19	199 ± 25.0	207 ± 24.3	0.302
Respiratory rate (breaths/min)	16	60 (39–80)	44 (38–65)	0.147
SUN (mg/dL)	22	30.0 (26–42)	35.0 (27–43)	0.879
Creatinine (mg/dL)	22	1.9 (1.7–2.6)	2.1 (1.5–2.7)	0.721
ALT (U/L)	19	97.0 (48–157)	74.0 (33–230)	0.573
PCV (%)	17	35.2 ± 8.1	35.0 ± 7.8	0.875
Total solids (g/dL)	18	7.5 ± 0.7	7.6 ± 0.8	0.604
Any clinical sign	18	18 (100)	15 (83)	0.250
Dyspnea	18	13 (72)	6 (33)	0.016
Lethargy	18	10 (56)	7 (39)	0.508
Collapse	18	1 (11)	1 (6)	1.00
Anorexia	18	11 (61)	2 (11)	0.004
Ascites	18	2 (11)	1 (6)	1.00
Murmur	18	4 (22)	3 (17)	1.00
Gallop heart rhythm	18	10 (56)	8 (44)	0.625
Arrhythmia	17	8 (47)	4 (24)	0.219

Data are reported as median (IQR) for nonnormally distributed values, mean ± SD for normally distributed values, or No. (%) of affected cats.

Values of $P < 0.05$ were considered significant.

Table 2—Echocardiographic findings in 26 cats with CHF before and after initiation of pimobendan treatment (0.25 to 0.30 mg/kg, PO, q 12 h).

Variable	No. of cats	Before treatment	After treatment	*P* value
LVIDd (cm)	9	1.69 ± 0.40	1.73 ± 0.43	0.464
LVIDs (cm)	9	1.32 ± 0.65	1.24 ± 0.65	0.165
Fractional shortening (%)	9	22.0 (8–39)	22.0 (16–54)	0.095
LVPWd (cm)	8	0.38 (0.29–0.58)	0.46 (0.34–0.56)	0.673
IVSd (cm)	7	0.37 ± 0.07	0.39 ± 0.13	0.527
Left atrial diameter (cm)	9	1.81 ± 0.46	1.67 ± 0.37	0.162
Aortic diameter (cm)	9	0.77 ± 0.09	0.77 ± 0.08)	0.907
Left atrial-to-aortic diameter ratio	9	2.49 (1.9–2.9)	2.04 (1.8–2.1)	0.515
Aortic velocity (m/s)	2	0.54 ± 0.12	0.65 ± 0.13	0.058
Pulmonary velocity (m/s)	2	0.38 (0.29–0.46)	0.62 (0.39–0.85)	0.180
LVOTO	9	0 (0)	0 (0)	1.00
RVOTO	9	1 (11)	0 (0)	1.00
Mitral regurgitation				
None	9	4 (44)	4 (44)	—
Mild	9	2 (22)	5 (56)	—
Moderate	9	2 (22)	0 (0)	—
Severe	9	1 (11)	0 (0)	—
Any (vs none)	9	5 (56)	5 (56)	1.00
Moderate to severe	9	3 (33)	0 (0)	0.25
Tricuspid regurgitation				
None	9	4 (44)	6 (67)	—
Mild	9	3 (33)	3 (33)	—
Moderate	9	0 (0)	0 (0)	—
Severe	9	2 (22)	0 (0)	—
Any (vs none)	9	5 (56)	3 (33)	0.50
Moderate to severe (vs none or mild)	9	2 (22)	0 (0)	0.50

— = Not calculated. IVSd = Interventricular septum dimension in diastole. LVIDd = Left ventricular internal dimension in diastole. LVIDs = Left ventricular internal dimension in systole. LVOTO = Left ventricular outflow tract obstruction. LVPWd = Left ventricular posterior wall dimension in diastole. RVOTO = Right ventricular outflow tract obstruction.

See Table 1 for remainder of key.

al shortening was unchanged, 6 cats had an increase, 1 cat had no change, and only 2 cats had a decrease in fractional shortening after pimobendan treatment.

All 26 cats used in the survival analysis died. Eleven were euthanized, 7 died naturally at home, and 8 had an undetermined cause of death. The median survival time was 167 days (95% CI, 32 to 339 days). The presence of so-called smoke had a significant (P = 0.021) negative effect on survival time (HR, 3.1; 95% CI, 1.2 to 8.0). Increases in PCV by 5% also had a small but significant (P = 0.042) negative effect (HR, 1.3; 95% CI, 1.0 to 1.8). For SAP, every 10 mm Hg increase from

the value before pimobendan treatment had a small but significant ($P = 0.037$) protective effect on survival time (ie, cats lived longer [HR, 0.9; 95% CI, 0.8 to 1.0]). Treatment with an angiotensin-converting-enzyme inhibitor also had a significant ($P = 0.04$) protective effect (HR, 0.4; 95% CI, 0.15 to 0.96).

Data from 24 cats were used for the multivariate survival analyses. Five significant variables were identified: sex, purebred, signs of lethargy, presence of smoke, and fractional shortening. Being male rather than female was a significant ($P = 0.001$) risk factor for death (HR, 3.7 [95% CI, 1.4 to 10.2]). Cats that were lethargic (vs not lethargic) at initial evaluation had an HR of 6.8 (95% CI, 2.3 to 20.1; $P = 0.001$), and those in which smoke was detected during echocardiography (vs not detected) had an HR of 6.1 (95% CI, 1.7 to 21.3; $P = 0.005$). Being a purebred versus nonpurebred cat and having a fractional shortening percentage that was 5% higher before versus after pimobendan treatment were protective factors for survival time, with HRs of 0.4 (95% CI, 0.1 to 1.0; $P = 0.049$) and 0.8 (95% CI, 0.7 to 0.9; $P = 0.001$), respectively. Pimobendan dosage was nonsignificant in the univariate (HR, 1.4; 95% CI, 0.8 to 2.7; $P = 0.252$) and multivariate (HR, 2.0; 95% CI, 0.9 to 4.4; $P = 0.100$) analyses.

Discussion

A dramatically short survival time in cats with CHF due to ventricular (myocardial) failure justifies exploration of novel treatments, including those not licensed for use in cats (ie, pimobendan). In the present study involving 27 cats with CHF, ventricular systolic dysfunction was considered to be present when at least 1 of the following criteria were met: low fractional shortening percentage, high left ventricular internal dimension in systole, presence of a regional area of hypokinesis subjectively believed to impair left ventricular ejection fraction, and severe dilatation of the right ventricle and right atrium consistent with a diagnosis of arrhythmogenic right ventricular CM.

To date, 4 research abstracts[d–g] have reported the use of pimobendan in cats with HF secondary to various underlying cardiac diseases. One abstract[e] describes a study in which pimobendan was used in a cohort similar to that of the present study, with a comparable survival time range of 9 to 585 days (median not reported). The median survival time in the present study was 167 days (95% CI, 32 to 339 days).

Addition of pimobendan to the conventional treatment protocol for cats with HF secondary to spontaneous heart disease characterized by underlying ventricular systolic dysfunction and no SAM led to significant improvement in the proportion of cats with dyspnea and anorexia. There were no significant differences in echocardiographic findings at follow-up, compared with before pimobendan treatment began. One cat with complex congenital heart disease and SAM had worsening systemic hypotension after 1 dose of pimobendan, and the drug was subsequently discontinued. Multivariate survival analysis identified lethargy, smoke, and male sex as predicting shorter survival times, whereas higher fractional shortening and being purebred predicted longer survival times.

The present study had several limitations. No contemporaneous group that did not receive pimobendan was used; therefore, one is unable to conclude whether pimobendan treatment had any effect on survival time. Additionally, the small cohort of cats used all had echocardiographic evidence of a decrease in ventricular systolic function, which is not typical of most heart disease in cats. In a recent study,[f] outcomes were reported for 161 cats with HF that received pimobendan in addition to conventional treatments. At least some of this group of cats did not have reduced ventricular systolic function, and some of the cats had SAM detected via echocardiography. Systolic anterior motion of the mitral valve typically occurs in hypertrophic CM but can occur in other heart diseases, as was the situation in 1 cat in the present study with complex congenital heart disease including mitral valve dysplasia. The clinical concern with SAM is that it can lead to dynamic left ventricular outflow tract obstruction. Treatments resulting in afterload reduction, enhanced systolic function, or both are generally contraindicated in conditions associated with left ventricular outflow tract obstruction such as SAM because they could lead to worsening of the obstruction, resulting in systemic hypotension, tachycardia, worsening CHF, and arrhythmias. Pimobendan is an inodilator and thus could lead to undesirable consequences in some cats with SAM, as it appeared to do in the 1 cat with SAM in the present study. However, MacGregor et al[f] did not report any adverse effects in cats with SAM. Additional prospective studies are necessary to determine whether SAM is a relevant contraindication for the use of pimobendan in cats.

The dosage of pimobendan used in the present study (0.25 to 0.30 mg/kg, PO, q 12 h) was chosen on the basis of dosages established for dogs (0.23 to 0.30 mg/kg [0.105 to 0.140 mg/lb], PO, q 12 h); the authors are unaware of any published reports on the pharmacokinetics of pimobendan in cats. Although the increased median survival time in cats in the present study relative to that in cats not treated with pimobendan in another study[a] (13 days) is encouraging, pharmacokinetic studies as well as prospective clinical trials of this drug are needed in cats before the drug can be recommended for use in cats with CHF.

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From this month's *AJVR*

Prevalence and types of tooth resorption in dogs with oral tumors

Ana Nemec et al

Objective—To determine the prevalence and types of tooth resorption in dogs with oral tumors and to compare findings with those for control dogs.

Animals—101 dogs with oral tumors and 128 control dogs that did not have oral tumors and for which dental radiographs were available.

Procedures—Exclusion criteria for dogs included systemic disease, long-term administration of anti-inflammatory drugs, traumatic occlusion, severe semigeneralized or generalized periodontitis, and endodontic disease. For each dog with an oral tumor, histologic sections of biopsy specimens of tumors were examined. Dental radiographic images of dogs were examined, and the presence and type of tooth resorption were determined for each tooth. Statistical analyses were performed to compare data regarding prevalence of tooth resorption.

Results—Teeth at tumor sites in dogs with nonodontogenic tumors were significantly more frequently affected with external inflammatory resorption, compared with teeth at tumor sites in dogs with odontogenic tumors. Teeth at sites distant from tumors in dogs with oral tumors were 3.2 times as likely to have external surface resorption (OR, 3.2; 95% confidence interval, 1.3 to 7.9) and 83.4 times as likely to have external inflammatory resorption (OR, 83.4; 95% confidence interval, 9.7 to 719.6) as teeth in control dogs.

Conclusions and Clinical Relevance—Resorption of teeth at tumor sites and at sites distant from tumors was common in dogs with oral tumors. Results of the present study will contribute to an understanding of the complex effects of oral tumors on local and distant hard tissues. (*Am J Vet Res* 2012;73:1057–1066)

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