

**ESVC-P-19**

**USE OF TORASEMIDE IN CATS FOR CONGESTIVE HEART FAILURE.** R. McDonald. University of Glasgow, Glasgow, UK

Torsemide is a loop diuretic which is more potent and of greater persistency than furosemide. Torsemide is a potentially useful treatment for dogs and cats in congestive heart failure (CHF) but is only licensed for use in dogs in the UK. The aim of this study was to describe the use of oral torsemide in cats with naturally occurring CHF and to report tolerability and adverse effects.

Two hospital databases were searched for cats with cardiac disease receiving torsemide from March 2014-March 2016; 11 client-owned cats with suitable records were subsequently retrospectively evaluated.

Collected data included signalment, presenting signs, diagnosis, concurrent medications, maximum dose of furosemide before transition to torsemide, starting torsemide dose, maximum torsemide dose reached during CHF therapy, survival time after commencing torsemide and comparison of renal parameters and electrolytes prior to and after commencing torsemide. Adverse reactions were recorded. A board-certified cardiologist retrospectively reviewed the cases. All owners signed consent forms to permit the use of off-licensed drugs.

Cats included all had CHF and had acquired cardiomyopathy or congenital cardiac disease confirmed on echocardiography. All were initially treated with furosemide; 55% were on oral therapy, 45% on intravenous therapy.

Various concurrent therapies included ACE inhibitors, spironolactone, antithrombotics, pimobendan, diltiazem, atenolol, carbimazole, famotidine, mirtazapine. At initiation of torsemide, cats were either poorly responsive to oral furosemide ( $n = 9$ ) or management was deemed more appropriate with torsemide ( $n = 2$ ).

The median dose of furosemide was 6 mg/kg/24 hrs (range 1–10 mg/kg/24 hrs) prior to commencing torsemide. The median initial dose of torsemide was 0.5 mg/kg/24 hr (0.1–0.75 mg/kg/24 hrs) administered once or divided into twice daily dosing.

At the first recheck there was a trend towards an increase in creatinine and urea and a decrease in potassium. Azotaemia was observed to be more marked in patients receiving a higher dose of torsemide. During chronic therapy, torsemide was reduced in 3 cats due to increasing azotaemia.

At the time of writing, 5 cats were still alive. Mortality was due to euthanasia for refractory CHF ( $n = 4$ ), progressive azotemia after starting torsemide ( $n = 1$ ) and 1 cat was lost to follow up. Median survival time was 48 d (7–177 d) with a median final torsemide dose of 0.36 mg/kg/24 hrs (0.21–0.75 mg/kg/24 hrs).

Oral torsemide was tolerated by this population of cats with CHF. The main adverse clinical effect was worsening azotaemia. Prospective studies are needed to further evaluate the use of torsemide in cats.

**Disclosures:** No disclosures to report.

**ESVC-P-20**

**IRON STATUS IN DOGS WITH MITRAL VALVE DISEASE.** A. Savarese, M. Probo, C. Locatelli, G. Traini, P.G. Brambilla, S. Paltrinieri. University of Milan, Milano, Italy

In people, anemia and serum iron (SI) deficiency (SID) are frequent co-morbidities in chronic heart failure (CHF). Recent studies showed that SID alone reduces quality of life and survival.

Mitral valve disease (MVD) is the most common acquired heart disease in dogs, which can lead to CHF.

To the best of our knowledge, no studies have examined iron status in MVD dogs.

The aims were to determine prevalence and characteristics of SID (SI < 90 µg/dL) in dogs with MVD, to analyze differences in SI among ACVIM classes, symptomatic and asymptomatic patients, and to study the association between SID and survival.

Fifty privately owned MVD dogs admitted to the Cardiology Service of DIMEVET (January 2015–April 2016) with complete physical evaluation, chest x-ray, echocardiographic examination and serum biochemical panel were included. Patients with other heart or systemic diseases were excluded.

Blood samples were collected during routine clinical evaluation; complete CBC and routine biochemistry were performed using an automated laser hematology analyzer and automated spectrophotometer, while excess serum was frozen until analysis. Iron status was evaluated measuring SI and total iron-binding capacity (TIBC); percentage transferrin saturation (% SAT) was calculated.

The median age of dogs was 11 years (IQR 10–14) and median body weight 11 kg (IQR 6–22). Most were intact males (42%). The most represented breed was mongrel (46%). Twenty dogs were ACVIM class B1 (40%), 12 B2 (24%), 15 C (30%) and 3 D (6%). Non-symptomatic and symptomatic dogs were respectively 64% ( $n = 32$ ) and 36% ( $n = 18$ ).

The prevalence of SID in MVD dogs was 16% (8/50: 6 symptomatic and 2 non-symptomatic). Only 3 patients (6%) presented anemia (Hct ≤ 37%). TIBC was within or above the reference range (NV: 270–496 µg/dL) in all dogs with SID, except one (reduced TIBC), while % SAT was below the minimum level (NV: >23%) in 5/8 dogs (62%).

No differences in SI were found between ACVIM classes and symptomatic/non-symptomatic patients.

Log-rank analysis showed shorter survival in MVD dogs with SID ( $P$  value: 0.020), nevertheless multivariate Cox analysis showed that only symptoms presence affect survival.

Our results show that SID, although does not appear to influence survival, is more common in symptomatic dogs and can be occasionally present without anemia in dogs with MVD; TIBC and % SAT values suggest that SID is most frequently true (primary SID) than functional (secondary to inflammatory conditions).

**Disclosures:** No disclosures to report.

**ESVC-P-21**

**RELATIONSHIP BETWEEN ONE-DIMENSIONAL LEFT ATRIAL PHASIC FUNCTION AND POST-CAPILLARY PULMONARY HYPERTENSION IN DOGS WITH DEGENERATIVE MITRAL VALVE DISEASE.** A.C. Merville, E. Roels, A. Gautier, C. Clercx, K. Mc Entee. Faculty of Veterinary Medicine, University of Liège, Liège, Belgium

Post-capillary pulmonary hypertension is common in dogs with degenerative mitral valve disease (DMVD). Its prevalence increases with severity of DMVD and its presence is a predictor of worse outcome. Left atrial (LA) function and size are prognostic indicators in DMVD. In dogs, LA contractile function has been shown to decrease with severity of DMVD. In human with chronic mitral regurgitation, LA function is an important correlate of right ventricular systolic pressure. The aim of this study was to assess if LA dysfunction was associated with PH in dogs with DMVD. Dogs with DMVD and a measurable tricuspid regurgitation (TR) were retrospectively recruited. Maximal LA diameter, LA diameter at the onset of the P wave and minimal LA diameter were measured on anatomic M-mode from 2D cine-loops on aortic short axis view. Left atrial reservoir (LA expansion index; Total LA shortening fraction), conduit (Passive LA shortening fraction) and contractile function (Active LA shortening fraction) indices were derived from above measures. Ninety three dogs including ACVIM stage B1 (22), B2 (28), C (39) and D (4), were included. Dogs were assigned to pulmonary hypertensive group (PH) (TR pressure gradient >40 mmHg) ( $n = 29$ , median: 51 mmHg; range: 40–114) or pulmonary normotensive group (PN) ( $n = 64$ , 29 mmHg; 5–40). LA reservoir and contractile function indices were reduced in ACVIM stage C and D compared to asymptomatic stages ( $P < 0.001$ ) and TR pressure gradient was higher in symptomatic dogs ( $P < 0.05$ ) compared to asymptomatic dogs. TR gradient was positively correlated with LA size measured at different time intervals ( $P < 0.001$ ) and negatively correlated with LA reservoir ( $P = 0.02$ ) and contractile ( $P = 0.009$ ) variables LA reservoir variables ( $P = 0.008$ ), and active LA shortening fraction ( $P = 0.006$ ) were lower in PH group compared to PN ANCOVA was used to test the categorical effect of ACVIM stages along with the effects of LA function indices on TR pressure gradient. ACVIM stage had a strong effect on TR gradient ( $P < 0.001$ ) but LA function parameters did not persist after correction for ACVIM stages. This study confirmed that, in dogs with DMVD, PH is strongly associated with the stage of heart failure but failed to show an independent relationship between LA dysfunction and development of PH. The