

2 lymphoma, 2 infectious diseases, 1 discospondylitis). At onset of clinical illness, cats with AA-amyloidosis vs. those without AA-amyloidosis had significantly lower albumin [median 2.3 g/dL (interquartile range 2.1-2.4) vs. 2.6 g/dL (2.5-2.9), $P = 0.031$] and higher illness severity score [0.60 (0.50-0.69) vs. 0.40 (0.38-0.50), $P = 0.025$]. Before death, cats with AA-amyloidosis vs. those without AA-amyloidosis had significantly higher WBC [18400/ μ L (15900-23000) vs. 9550/ μ L (4800-12100), $P = 0.005$] and lower albumin [2.3 g/dL (2.1-2.4) vs. 2.9 g/dL (2.4-3.0), $P = 0.039$]. Differences between the 2 groups were not observed for duration of stay and of illness, hematocrit, MCV, creatinine, bilirubin, globulin and UPC, at either time point. Age and gender did not differ.

In conclusion, AA-amyloidosis in shelter cats is associated to lower serum albumin concentrations throughout clinical illness. Hypoalbuminemia does not seem caused by proteinuria. The higher illness score at onset of clinical signs might suggest that disease severity has a permissive role in the pathogenesis of AA-amyloidosis in shelter cats. Further studies are needed to confirm these preliminary findings.

Disclosures

No disclosures to report.

ESVIM-P-15

Alendronate treatment in cats with idiopathic hypercalcemia: A retrospective control study of 20 cases

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Alendronate has been advocated for long-term management of idiopathic hypercalcemia of cats (IHC). To date, only three case reports and one prospective uncontrolled study have documented the usefulness of alendronate in IHC.

The aims of this study were to investigate whether treatment with alendronate is associated with a decrease of ionized calcium (iCa) in comparison with other (or no) treatment.

Cats with IHC were recruited. IHC was defined by persistently elevated iCa and exclusion of other causes of hypercalcemia based on paraclinical including PTH dosage. Patients were divided into group 1 (cats treated with alendronate) and 2 (cats not treated). T_0 was defined as last control before treatment initiation, and iCa_{T0} as iCa at T_0 . Two endpoints were investigated: occurrence of normocalcemia ($iCa < 1.40$: $iCa_{<1.40}$) and occurrence of a 15%-decrease in iCa in comparison to iCa_{T0} ($iCa < iCa_{T0} - 15\% iCa_{T0}$: $iCa_{-15\%}$). Kaplan-Meier method with logrank testing was used to compare time to endpoints between groups. Variables were presented as medians [25th quartile ; 75th quartile] and compared with Mann-Whitney test. Differences were considered significant when $P < 0.05$.

Twenty cats were included. The two groups were comparable regarding epidemiologic and biological data. Three cats in group 2 received

other treatments: prednisolone (2) or furosemide (1). In 6/11 cats (55%), alendronate dose had to be increased from 10 to 15 (1) or 20 mg (5) weekly. Median iCa variation from iCa_{T0} at 6 months of follow up (+/- 60 days) was -18% [-21 ; 3] in group 1 and -1% [-6 ; 3] in group 2 ($P = 0.35$). Median percentage of days spent with normocalcemia over total duration of follow-up was 66% [6 ; 18] in group 1 and 17% [17 ; 40] in group 2 ($P = 0.106$). Median time to $iCa_{-15\%}$ was significantly longer in group 1 (119 days) than in group 2 (median not reached; $P = 0.02$). Median times to $iCa_{<1.40}$ were not significantly different between group 1 (80 days) and group 2 (150 days; $P = 0.81$). Severe hypophosphatemia was observed in one treated cat ; alendronate was stopped. No other sign of toxicity was observed.

These results suggest that treatment with alendronate in IHC seems to be associated with a shorter time to a 15%-decrease of iCa from baseline, as compared with other (or no) treatment. Alendronate might be more indicated than other or no treatments for IHC.

Disclosures

No disclosures to report.

ESVNU-P-1

A prospective evaluation of contrast-induced nephropathy (CIN) in dogs

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Administration of intravenous iodinated contrast (IVIC) in humans has been causally associated with the development of acute kidney injury, known as contrast-induced nephropathy (CIN). Serum creatinine has been shown to increase 3-5 days after IVIC and kidney injury could range from subclinical forms to severe kidney failure. Scattered information exists in dogs in vitro as well as in laboratory studies. In a recent retrospective study, an increase in serum creatinine after IVIC was observed in dogs, however, a causal association was not demonstrated. A population of dogs undergoing computed tomography examination was prospectively evaluated for evidence of CIN after IVIC administration. Biochemical parameters (serum creatinine, blood urea nitrogen, total protein, albumin, chloride, phosphorus, potassium, calcium, sodium and symmetric dimethylarginine) and urinalysis (specific gravity, dipstick, sediment, protein/creatinine ratio, alkaline phosphatase/creatinine ratio, γ -glutamyl transferase/creatinine ratio) were evaluated at the time of IVIC administration (T_0) and after 3-7 days (T_1). Twenty-three dogs of different age, breed and sex were enrolled. Three dogs showed increased symmetric dimethylarginine and hyperphosphatemia at T_1 , whereas 6 dogs showed isostenuria, cilindruria and proteinuria. An increased in serum creatinine >25% and γ -glutamyl transferase/creatinine ratio >50% from baseline was found in 2 and 4 dogs, respectively. None of these dogs had a pre-existing kidney disease. A significant difference between T_0 and T_1 for serum

albumin, total protein, chloride, calcium and phosphorus was found. Although no clinically relevant kidney injury was found, CIN developed in some dogs after IVIC administration. Further studies are need to confirm these preliminary results.

Disclosures

No disclosures to report.

ESVNU-P-2

Examination of serum hepcidin concentration in dogs with kidney disease

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Hepcidin is the key regulator hormone of the iron homeostasis. According to human studies, the serum hepcidin concentration in patients with kidney disease is frequently elevated, and the consequently evolved iron sequestration contributes the non-regenerative anemia and even may lead to erythropoietin resistance.

Our study aimed to measure serum hepcidin concentration in dogs with kidney disease; the hypothesis was that serum hepcidin in these sick dogs is elevated compared to healthy ones.

The study population included 21 dogs (7 with acute kidney injury [AKI] and 14 with chronic kidney disease [CKD]) from patients presented in the Small Animal Hospital Nephrology Service or Intensive Care Unit of the University of Veterinary Medicine - Budapest. Routine hematology, biochemistry (including C-reactive protein, iron, total iron-binding capacity) and urinalysis were performed by all patients. Left-over serum samples were used to measure hepcidin with liquid-chromatography tandem mass spectrometry method (LC/MS-MS). Results from our previous study evaluating serum hepcidin in 86 healthy dogs were used as control.

All dogs with AKI (7/7) and 50% of the dogs with CKD (7/14) had hepcidin concentration above the reference range, with mean hepcidin of 63,45 ng/mL (40,1-110,1) in AKI group and 38,45 ng/mL (17,7-66,9) in CKD group compared to the healthy population 16,6 ng/mL (2,3-41,1). The difference was significant in all dogs vs healthy ($P < 0,001$), in AKI vs healthy ($P = 0,015$), AKI vs CKD ($P = 0,031$), but not between the CKD and healthy groups ($P = 0,067$).

Serum hepcidin significantly correlated with C-reactive protein levels in the kidney disease population ($P = 0,037$, $\rho = 0,6142$), but not with hematocrit, serum iron and iron-binding capacity.

This study showed that correspondingly to human studies, elevated serum hepcidin concentrations were frequently detected in dogs with kidney disease.

This research was funded by National Distinction Program No NKB KEDH106320 and European Social Fund (grant agreement no. EFOP-3.6.3-VEKOP-16-2017-00005).

Disclosures

This research was funded by National Distinction Program No NKB KEDH106320 and European Social Fund (grant agreement no. EFOP-3.6.3-VEKOP-16-2017-00005).

ESVNU-P-3

3D bladder ultrasound for estimation of urine volume in dogs vs. traditional 2D ultrasound methods

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Urinary bladder volume (UBV) and residual volume can provide important clinical information for hospitalized dogs and dogs with micturition disorders. UBV can be measured directly via urethral catheterization or indirectly via 2D ultrasound formulations. However, these techniques impose risk such as those associated with sedation, moderate restraint needed, catheter-associated urinary tract infections and/or need for appropriate operator skill and equipment. 3D ultrasound for point-of-care volumetric assessments of the urinary bladder is the method of choice for monitoring UBV in people but is not routinely performed in dogs.

The aim of this study was to validate the application of 3D ultrasound at small, medium, and large urinary volumes in dogs, compare measurements of 3D bladder estimation obtained by a novice to traditional 2D measurements by a board-certified veterinary radiologist, and compare time required for examination by 3D ultrasound to traditional 2D, B-mode ultrasound calculations.

In this prospective, experimental study, 10 laboratory-bred Beagle dogs were utilized for estimation of UBV. Bladders were infused with a calculated amount of sterile saline to represent small, medium, and large volumes. Each UBV was estimated and calculated by a boarded radiologist using 2D ultrasound followed by a 3D ultrasound device by a novice. Measured UBVs were compared to the instilled UBV for each method, and the two methods were compared to each other. Time from start to end of examination was recorded for both methods.

Use of 2D ultrasound overestimated infused UBV with a mean [SD] difference of 4.2 ml +/- 13.1 ml. The 3D ultrasound underestimated infused UBV with a mean difference [SD] of -9.8 ml +/- 9.8 ml. 3D ultrasound took less time to measure UBV with a mean of 80 seconds per measurement compared to 165 seconds per measurement for 2D.

The tested 3D ultrasound device is a safe, efficient, and clinically effective tool for measuring UBV in dogs. The device decreases need for operator skill or board certification, reduces time for bladder estimation and provides a quick estimate of bladder volume in real time.