Peripheral and Central Venous Blood Glucose Concentrations in Dogs and Cats with Acute Arterial Thromboembolism

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Background: Acute limb paralysis because of arterial thromboembolism (ATE) occurs in cats and less commonly in dogs. ATE is diagnosed based on physical examination findings and, occasionally, advanced imaging.

Hypothesis/Objectives: Peripheral, affected limb venous glucose concentration is decreased in ATE, whereas its systemic concentration is within or above reference interval.

Animals: Client-owned cats and dogs were divided into 3 respective groups: acute limb paralysis because of ATE (22 cats and 9 dogs); acute limb paralysis secondary to orthopedic or neurologic conditions (nonambulatory controls; 10 cats and 11 dogs); ambulatory animals presented because of various diseases (ambulatory controls; 10 cats and 9 dogs).

Methods: Prospective observational, clinical study. Systemic and local (affected limb) blood glucose concentrations were measured. Their absolute and relative differences (ΔGlu and %ΔGlu, respectively) were compared among groups.

Results: ΔGlu and %ΔGlu were significantly higher in the ATE cats and dogs groups, compared to both of their respective controls ($P < .0001$ and $P < .001$, respectively). No significant differences were observed between the control groups. Receiver operator characteristics analysis of ΔGlu and %ΔGlu as predictors of ATE had area under the curve of 0.96 and 0.99 in cats, respectively, and 1.00 and 1.00, in dogs, respectively. ΔGlu cutoffs of 30 mg/dL and 16 mg/dL, in cats and dogs, respectively, corresponded to sensitivity and specificity of 100% and 90% in cats, respectively, and 100% in dogs.

Conclusions and Clinical Importance: ΔGlu and %ΔGlu are accurate, readily available, diagnostic markers of acute ATE in paralyzed cats and dogs.

Key words: Canine; Feline; Glucometer; Thrombosis.

Arterial thromboembolism (ATE) is a devastating condition in cats, mostly associated with underlying cardiac diseases, less frequently with hyperthyroidism and neoplasia, and in a minority of cases, the etiology is undetermined.[1] In cats, ATE is clinically manifested mostly by an acute onset of unilateral or bilateral paraparesis or paraplegia, weak or absent arterial pulses, pain, pale or cyanotic footpads and nail-beds, and cold extremities of the affected limbs.[a,1] It most commonly affects both hind limbs, although a single hind limb, or the front limbs, might be affected.[a,1] ATE occurs less commonly in dogs than in cats and is associated with a wider range of predisposing conditions.[2–6]

The diagnosis of ATE is based primarily on the clinical presentation, which might be insufficient to allow confirmation. A history of predisposing conditions might support the diagnosis, but a definitive diagnosis requires advanced imaging tools. Selective iodine contrast medium angiography is the gold standard for diagnosing aortic thrombosis.[7] Conventional angiography has excellent spatial resolution, but is invasive and

Abbreviations:

95% CI	95% confidence interval
%ΔGlu	absolute blood glucose concentration difference between the central and peripheral samples divided by central blood glucose concentration
ΔGlu	absolute blood glucose concentration difference between the central and peripheral samples
ATE	arterial thromboembolism
AUC	area under the curve
MRA	magnetic resonance angiography
ROC	receiver operator characteristics

technically challenging.[8,9] Radionuclide angiography allows assessment of the location and severity of aortic thrombosis, but requires special facilities, whereas anatomic detail is poor.[10] Abdominal ultrasonography might be useful for identifying aortic thrombi, but might require technical expertise, and can be challenging, because the ultrasonographic characteristics of thrombi vary depending on their age.[8,9,11] In acute ATE, the size of the thrombus might be insufficient for a diagnosis.[7,12] Doppler-flow evaluation is usually necessary for the assessment of thrombi.[8,11] Contrast enhanced magnetic resonance angiography (MRA) was anatomically accurate for detection the full extent of aortic thrombi in 4 dogs with ATE[7]; however, MRA is often unavailable, is cost-prohibitive, and requires general anesthesia.

In ATE, the blood supply to the area distal to the thrombus is diminished, with decreased nutrient and oxygen delivery.[b,13] Our hypothesis was that ATE results in diminished local venous glucose concentration in the affected, paralyzed limbs, whereas systemic glucose concentration remains unaffected by the ATE,

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and will therefore remain within reference interval (RI), or might be increased because of stress, pain, and excitement. The difference between the systemic and the affected limb, peripheral blood glucose concentration in cases of ATE is therefore expected to be higher than that between the systemic glucose concentration and its peripheral concentration in an unaffected limb, and higher than this difference in ambulatory animals and those with paralysis of other causes. This difference may therefore serve as a useful, readily available indicator of acute ATE in cats and dogs.

Materials and Methods

Study Design, Selection of Animals, and Definitions

This prospective observational clinical study was approved by the Institutional Committee of Animal Care and Experimentation. The study groups included cats and dogs presented to the Hebrew University Veterinary Teaching Hospital (years 2005–2012) with paralysis of an acute onset (<24 hours from onset of clinical signs), which were diagnosed with acute ATE (ATE group). The clinical diagnosis of ATE was based on the history and clinical signs at presentation, including pain, pallor, decreased limb temperature by palpation, and absence of arterial pulse in the affected limb. In addition, in some animals, the diagnosis of ATE was further supported by Doppler ultrasonography or by post mortem examination findings. Animals with a history of chronic paresis or paralysis were excluded.

There were 2 negative control groups for each animal species, which included animals presented during the same time-period as the ATE cases, with their owners' consent. One consisted of animals presenting with paresis or paralysis because of orthopedic or neurological injuries (nonambulatory control group), and the second included nonparalyzed, nonparetic ambulatory animals (ambulatory control group). In the nonambulatory controls, ATE was definitively excluded based on their history, physical examination, presence of a palpable arterial femoral pulse in the affected limb, and advanced imaging. These animals were diagnosed with orthopedic and neurological diseases deemed unrelated to ATE, based on the history, orthopedic, and neurological examinations, appropriate survey radiography, cerebrospinal fluid analysis, myelography, or computed tomography, as deemed necessary for the diagnosis. The ambulatory control group animals did not present with limb paralysis or paresis, and were selected randomly, during the study period.

Blood Sampling and Glucose Measurement

In all animals with ATE and the nonambulatory controls, concurrent whole blood samples were collected for glucose concentration measurement from the jugular vein or a large vein in an unaffected limb and from peripheral vein in the affected (ie, paretic or paralytic) limb ("peripheral" sample). In all cases, samples either were collected into heparinized capillaries or into a syringe, and within 1 minute into such capillaries. Analysis was performed within 1 minute. In animals presenting bilateral hind limb paresis or paralysis, the affected limb vein was chosen at the attending clinicians' preference. In all cases of hind limb paralysis, systemic blood samples were obtained from the cephalic or jugular veins. In 1 ATE cat, with front limb paralysis and in 1 ATE dog, with paralysis of 3 limbs, systemic blood samples were obtained through jugular venipuncture. In the ambulatory control animals, the peripheral blood sample was obtained by hind limb venipuncture. Clinicians were blinded to the results of this study.

Blood glucose concentration was measured using commercially available glucometers,[c] which are based on the EN ISO 15197 standard and their performance is within the minimum acceptable performance criteria in 99.5% of cases tested, when blood glucose concentration is between 10 and 600 mg/dL.[d,14]

Statistical Analysis

To determine the minimum size of each of the study groups, a power analysis was performed a priori. We assumed a power of 80%, alpha of 5% and standard deviations of glucose concentration (for both systemic and local samples) of 20 mg/dL. To detect significant ΔGlu difference between groups, the minimum size of each group, of both dogs and cats, is 9 animals.

For each animal, the absolute glucose concentration difference between the systemic and peripheral samples (ΔGlu) was calculated (ie, ΔGlu; calculated systemic sample glucose – peripheral sample glucose), as well as the absolute proportional glucose difference (%ΔGlu; calculated as ΔGlu/systemic sample glucose concentration). Cats and dogs of the 3 groups were compared separately. Systemic and peripheral blood glucose concentrations within each group were compared using the Wilcoxon signed-rank test. In each animal species, blood glucose concentrations among the groups were compared using the Kruskal–Wallis test, and if differences were significant, post hoc analysis for multiple pairwise comparisons were made using the Mann–Whitney U-test, with Bonferroni's correction of the significance level. In cats, in addition, the ATE group was also divided into 2 subgroups, namely, those in which the diagnosis of ATE was based only on clinical signs, and those in which it was based on clinical signs as well as ultrasonography or postmortem findings. These 2 groups and the 2 control groups were compared using the Kruskal–Wallis test, and if differences were significant, post hoc analysis for multiple pairwise comparisons were made using the Mann–Whitney U-test, with Bonferroni's correction of the significance level.

The receiver operator characteristics (ROC) procedure, with its area under the curve (AUC) and 95% confidence interval (95% CI), was used to assess the predictive performance of ΔGlu and %ΔGlu as predictors of ATE, and to identify optimal cutoff points, associated with the least misclassifications, with their corresponding sensitivity and specificity for prediction of ATE. All tests were two-tailed, and a P value ≤.05 (unless Bonferroni's correction was applied) was considered significant. All analyses were performed using a statistical software package.[e]

Results

The study groups included 22 cats and 9 dogs diagnosed with ATE, whereas the corresponding nonambulatory control groups included 10 cats and 11 dogs, and the ambulatory control groups included 10 cats and 9 dogs. There were significant age differences among the 3 study groups, of both cats and dogs ($P = .03$ and $P = .01$, respectively). The median age of the ATE cat group was 6 years (range 1.5–15.0) compared to 1 year (range 0.3–11.0) in the nonambulatory control group, and 1.7 years (range 0.3–15.0) in the ambulatory control group. The median age of the ATE canine group was 10 years (range 7–14), compared to 4.5 years (range 0.5–8.0) in the nonambulatory control group, and 7 years (range 1–14) in the ambulatory control group. There were significant age differences among groups, in both cats and dogs, and

post hoc analysis showed these resulted because of differences between the ATE groups and their respective nonambulatory control groups.

In the ATE groups, 15/22 cats (68%) and 4/9 dogs (44%) did not survive to discharge, of which 12/15 cats (80%) and 4/4 dogs (100%) were euthanized.

In the ATE feline group, 19 cats (86%) presented with bilateral hind limb paralysis and 3 with a single limb paralysis (2 with a right hind leg paralysis and 1 with right front limb paralysis). Doppler ultrasonography, confirming the ATE, was performed in 3 cats, whereas in 2 additional cats, ATE was confirmed post mortem. Therefore, in 17/22 cats ATE was diagnosed based only on their clinical presentation. Underlying cardiac diseases were diagnosed in 9/22 cats (41%), based on physical examination findings, thoracic radiography and echocardiography, performed by a board-certified cardiologist, including hypertrophic cardiomyopathy (7 cats), left ventricular enlargement (1), and restrictive cardiomyopathy (1). In 9 additional cats (41%) in which the owners declined a board-certified cardiologist echocardiogram because of financial constraints, underlying cardiac diseases were suspected based on physical examination findings (eg, presence of cardiac murmur or gallop rhythm), thoracic radiography and echocardiography, performed by an emergency clinician, revealing left atrial enlargement and a left atrium/aorta diameter ratio >1.5. An underlying cardiac disease was therefore diagnosed in 18/22 of the cats (82%). In the remaining 4 cats (18%), no underlying disease was diagnosed, despite an investigation that included CBC, serum chemistry, urinalysis, and abdominal ultrasonography and radiography. Moreover, in 1 of these 4 cats necropsy did not reveal the underlying cause of ATE.

The final diagnoses in the nonambulatory control cats group included pelvic fracture (3 cats), vertebral fracture and brachial plexus avulsion (2 each), spinal neoplasia, infectious meningomyelitis, and intervertebral disc herniation (1 each). The ambulatory control cats group included healthy, staff-owned cats (5), a cat presented postovariohysterectomy because of dehiscence of the surgical incision, as well as cats with feline panleukopenia virus infection, acute kidney injury, chronic gastritis, and an infected skin laceration (1 each).

In the ATE dog group, 6/9 dogs presented with bilateral hind limb paralysis, of which 1 had left front limb paralysis as well, whereas 3 dogs had single hind limb paralysis. ATE was confirmed using Doppler ultrasonography in all dogs. In 3 dogs, compatible post mortem findings of ATE were recorded as well. The primary concurrent diagnoses included adrenal mass with mitral and tricuspid regurgitation, severely hypertensive acute kidney injury with pancreatitis, normal pregnancy, ruptured splenic hemangiosarcoma with disseminated intravascular coagulation, and granulomatous meningoencephalitis (1 each). The latter was definitely diagnosed with ATE at necropsy. In 4 dogs, no underlying disease was diagnosed despite an extensive workup, similar as described in cats. In 1 of

these dogs, a necropsy was performed and revealed GME, as well as the saddle ATE; however, the cause of the ATE was undetermined.

The nonambulatory control dogs group included traumatic vertebral fracture (6 dogs), intervertebral disc herniation (4) and traumatic pelvic fractures (1). The ambulatory control dog group included healthy blood donors (2 dogs), and dogs with hemoperitoneum, idiopathic epilepsy, esophageal spirocercosis, cervical vertebral disc herniation, granulomatous meningencephalitis, post-operative gastric dilatation, and volvulus and traumatic mandibular fracture (1 each).

In the animals with ATE, peripheral glucose concentration, obtained from the affected limb, was significantly lower compared to systemic glucose concentration in both cats (median 45 mg/dL; range 17–182, versus 181 mg/dL; range 98–394, respectively; $P < .0001$) and dogs (median 46 mg/dL; range 10–161, versus median 94 mg/dL; range 58–298 mg/dL, respectively; $P = .008$). There were no significant differences between systemic and peripheral blood glucose concentrations in all animals in both control groups (Table 1).

There were significant ΔGlu and %ΔGlu differences among the cat groups and among the dog groups ($P < .0001$ and $P < .001$, respectively, for both variables) (Table 2; Fig 1). Post hoc analysis showed that both ΔGlu and %ΔGlu, in both cats and dogs with ATE, were significantly higher compared to their respective 2 negative control groups ($P < .0001$ and $P < .001$, respectively, for both variables), whereas there were no differences in these parameters among the 2 respective negative control groups in each animal species ($P > .05$ for both variables, in both species) (Table 2).

The ATE group of cats was divided further into 2 subgroups: confirmed (5 cats) and clinically diagnosed (17 cats) subgroups. These subgroups and the 2 control groups were then compared. There was no significant difference in ΔGlu between the 5 definitely diagnosed cats with ATE and the 17 diagnosed clinically (median 72 mg/dL [range 53–147] and median 110 mg/dL [range 51–354], respectively; $P = .22$). There were significant ΔGlu and %ΔGlu differences when these 2 subgroups and the 2 control groups were compared ($P < .001$, for both). Post hoc analysis, with Bonferroni's correction for α, showed no significant ΔGlu and %ΔGlu differences between the 2 ATE subgroups ($P > .0125$ for both). There were significant ΔGlu and %ΔGlu differences between both ATE subgroups and each of the control groups ($P < .007$ for all comparisons). The median ΔGlu for the definitely diagnosed ATE feline subgroup and the clinically diagnosed ATE feline subgroup were 72 mg/dL (range 53–147) and 110 mg/dL (range 51–354), respectively, compared to 8 mg/dL (range 10–101) in the nonambulatory control group and median 0 mg/dL (range −13 to 7) in the ambulatory control group.

Peripheral glucose concentration was significantly lower ($P = .006$) in the affected limb in the ATE cat

Table 1. Systemic and peripheral, limb blood glucose concentrations in dogs and cats with acute arterial thromboembolism and their respective negative control groups.

| Group | Source of Blood Sample | N | Blood Glucose Concentration (mg/dL) | | P Value |
			Median	Range	
Cats					
ATE	Systemic	22	181	98–394	<.0001
	Affected limb	22	45	17–182	
Nonambulatory controls	Systemic	10	175	77–292	.14
	Affected limb	10	176	87–218	
Ambulatory controls	Systemic	10	129	23–183	.67
	Hind limb	10	127	23–196	
Dogs					
ATE	Systemic	9	94	58–298	.008
	Affected limb	9	46	10–161	
Nonambulatory controls	Systemic	11	84	42–149	.23
	Affected limb	11	90	46–136	
Ambulatory controls	Systemic	9	84	75–140	.48
	Hind limb	9	90	73–134	

ATE, arterial thromboembolism; N, number of animals.

Table 2. Absolute and relative differences between systemic and peripheral, affected limb blood glucose concentration in dogs and cats with acute arterial thromboembolism and in their respective negative control groups.

Group	N	ΔGlu (mg/dL), Median (Range)	%ΔGlu (%), Median (Range)	P Value*
Cats				
ATE	22	107[a] (51–354)	74[a] (35–95)	<.0001
Nonambulatory controls	10	8[b] (−10 to 101)	5[b] (−13 to 35)	
Ambulatory controls	10	0[b] (−13 to 7)	0[b] (−7 to 6)	
Dogs				
ATE	9	46[a] (18–187)	46[a] (31–95)	<.001
Nonambulatory controls	11	−2[b] (−23 to 13)	−3[b] (−32 to 9)	
Ambulatory controls	9	1[b] (−10 to 8)	1[b] (−13 to 6)	

Different superscript letters designate significant post hoc difference between group medians in pairwise comparisons (Mann–Whitney tests).

ATE, arterial thromboembolism; N, number of animals; ΔGlu, absolute difference between systemic and local, limb glucose concentrations; %ΔGlu, difference between systemic and local, limb blood glucose concentrations divided by systemic blood glucose concentration.

*P values refer to Kruskal–Wallis test, compering all 3 groups in each species. Different superscript letters designate significant difference between group medians in pairwise comparisons.

group (22 cats combined; median 45 mg/dL, range 17–182) compared to both nonambulatory (median 176 mg/dL, range 87–218) and ambulatory control (median 127 mg/dL, range 23–196) groups. In dogs, peripheral glucose concentration was significantly lower ($P = 0.006$) in the affected limb in the ATE group (median 46 mg/dL, range 10–161) compared to its concentration in both the nonambulatory (median 90 mg/dL, range 46–136) and ambulatory control (median 90 mg/dL, range 73–134) groups. There were no statistical systemic glucose concentration differences among the cat or dog groups.

Receiver operator characteristics analyses for ΔGlu and %ΔGlu as predictors of ATE in cats showed AUCs of 0.98 (95% confidence interval [$CI_{95\%}$] 0.90–1.00), and 1.00 ($CI_{95\%}$ 0.94–1.00), respectively. In dogs, ROC analyses for the ΔGlu and the %ΔGlu as predictors of ATE showed AUCs of 1.00 ($CI_{95\%}$ 0.89–1.00), and 1.00 ($CI_{95\%}$ 0.89–1.00), respectively. A ΔGlu cutoff of 30 mg/dL corresponded to sensitivity and specificity of 100% and 90%, respectively, for predicting ATE in cats. A %ΔGlu cutoff of 23% corresponded to a sensitivity and specificity of 100% and 90%, respectively in cats. In dogs, corresponding cutoffs of 16 mg/dL for ΔGlu, and 20% for %ΔGlu, corresponded to sensitivity and specificity of 100%, for both.

Discussion

The results of this study show that in both cats and dogs with ATE, peripheral (affected limb) blood glucose concentration is significantly lower compared to systemic glucose concentration. These results are in agreement with previous findings, showing that decreased local venous glucose in samples obtained
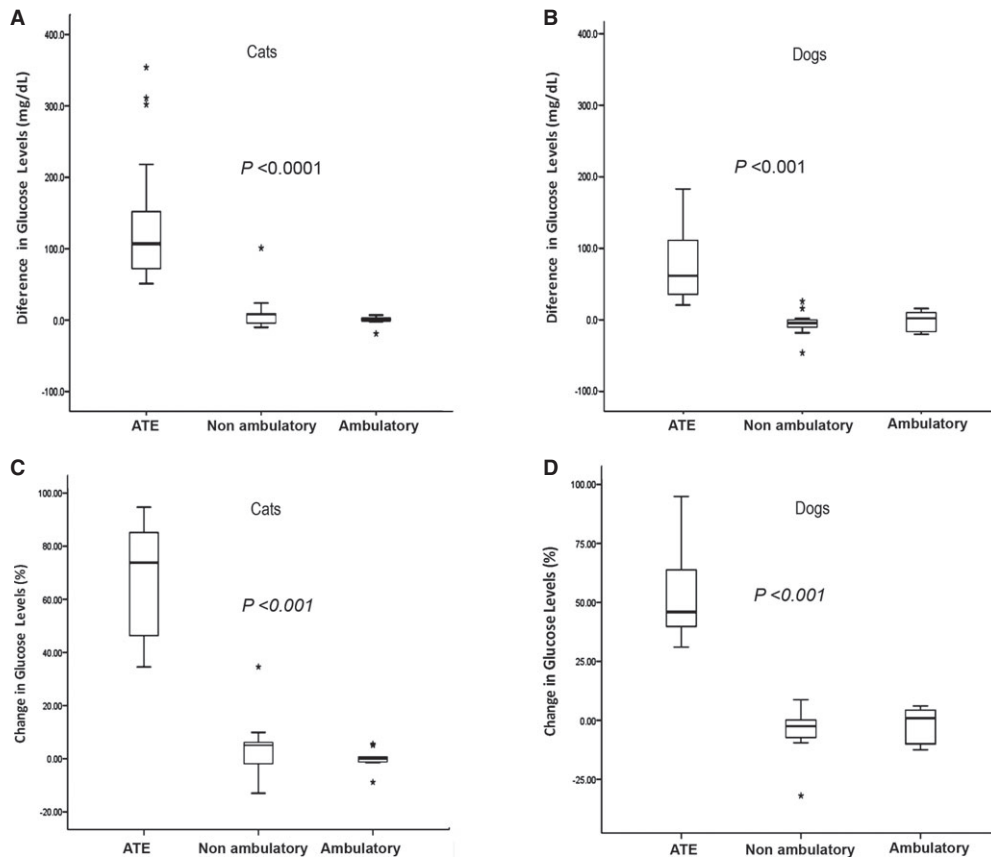
Fig 1. Box and whiskers plots of the absolute difference (calculated as systemic blood glucose – peripheral blood glucose) between systemic and peripheral glucose concentrations, and the relative difference (calculated as the absolute difference/systemic blood glucose concentration) in the study groups (**A**, **C**: cats, **B**, **D**: dogs). The line within the boxes represents the median, whereas the boxes represent the interquartile range, the whiskers represent the range, extending to a maximum of 1.5 times the interquartile range, and outliers are marked as dots.

from affected limbs of 11 cats and 2 dogs with acute ATE is a sensitive marker of ATE.[b]

The peripheral blood glucose concentration in samples obtained from a paralyzed limb in cases of ATE is expected to be low in face of systemic normoglycemia or hyperglycemia.[15] The latter has been attributed to stress, anxiety, pain, and hyperlactatemia.[15] Anxiety, pain, and stress lead to increased concentrations of diabetogenic hormones, including catecholamines, glucagon, and cortisol, which inhibit insulin secretion and increase insulin resistance, gluconeogenesis, and glycogenolysis.[1,3] Myocyte anaerobic glycolysis in the affected limbs results in lactate production, a major gluconeogenic precursor, which is delivered through the unaffected venous blood supply in the affected limb to the systemic circulation, and to the liver, thereby contributing to increased systemic glucose concentration.[15] Since the underlying conditions may affect blood glucose concentration, analyzing single samples from affected limbs may not be sufficient for a diagnosis of ATE. However, when systemic and peripheral blood glucose samples are analyzed concurrently, and compared, glucose concentration difference becomes apparent, and is therefore diagnostic of ATE.

This study showed that ΔGlu and %ΔGlu are significantly higher in both cats and dogs of the ATE groups compared to their corresponding ambulatory as well as nonambulatory control groups, in which ATE was absent. Cutoffs of ΔGlu ≥30 mg/dL and % ΔGlu ≥23%, respectively, in cats, and of ΔGlu ≥16 mg/dL and %ΔGlu ≥20%, respectively, in dogs, showed very high sensitivity and specificity for prediction of ATE. Therefore, this diagnostic procedure seems to be extremely accurate. It has additional advantages, including high applicability and availability, minimal blood volume requirement, and high cost-efficiency, especially when compared to other more technically challenging, often cost-prohibiting (ie, conventional and Doppler ultrasonography[7,10]) or invasive diagnostic procedures (ie, nuclear scintigraphy,[9] contrast angiography,[7,8] and MRA[7]). Increased plasma D-dimer concentration was positively associated with ATE in dogs and cats,[16–18] however, although the test is simple, it is often unavailable, it requires a higher blood volume, and is performed in citrated plasma, thereby consuming more time. In addition, it most often cannot be performed bedside, since whole blood centrifugation is required. Finally, the test is much

more expensive compared to blood glucose measurement.

Routine serum biochemistry testing was also investigated in dogs and cats with ATE, and the common abnormalities include hyperglycemia, azotemia, hyperkalemia, and increased serum muscle enzyme activity.[1,4,15] All these abnormalities are nonspecific of ATE, and cannot be solely used to discriminate between damage to a limb because of ATE and other, nonvascular lesions (eg, trauma, hematoma, rhabdomyolysis, infection, or neoplasia). In addition, some of such abnormalities may result from the primary underlying condition, or from secondary complications (eg, azotemia and hyperkalemia in acute kidney injury,[19–21] or increased creatine kinase activity in sepsis and bacterial infections[21]).

Consistent with the pathogenesis of ATE, and similar to the behavior of systemic and peripheral glucose concentrations herein, differences in systemic and peripheral concentrations of other laboratory analytes may occur as well, including lactate, potassium, pH, and blood gases, which may also be considered as diagnostic indicators of ATE and were not investigated herein. This is because the arterial blood supply to the affected limb is diminished, with resultant increased local anaerobic metabolism. Lactate concentration in blood samples obtained from affected limbs in 11 cats and 2 dogs with acute ATE was shown to be a sensitive indicator of ATE.[b] Future studies of ATE may include these analytes, as well as physical parameters (eg, toe temperature) as predictors of acute ATE.[22] Herein, investigation of glucose concentration differences was selected because of the very high availability, low cost, and the simplicity of blood glucose testing. Results are quickly obtained, and the use of a glucometer requires as little as 0.6 μL of fresh whole blood.

Obtaining blood samples from affected limbs, in which arterial blood supply was diminished or absent, raised concerns regarding a risk of infection, and infliction of pain. We therefore elected to sample only a single paralyzed limb in each animal. However, infections were not noted in the sampled affected limb in any animal. As for infliction of pain, in all of the animals with ATE in this study, deep pain sensation was either absent, or markedly diminished, and no indication of pain was observed during blood sampling from the affected limbs in any of these animals. Since glucose concentration measurement using the glucometer requires only a single blood drop, we strongly believe that the procedure was safe, causing no discomfort to the animals with ATE and very little discomfort to the negative control animals.

This study has several limitations. First, the size of study groups was rather limited, although it met the numbers determined by the a priori power analysis. Larger groups might have allowed further substantiation of these results. Second, this study was not designed to investigate the extent of the damage to the affected limbs, the size of the thrombus, or the prognosis of acute ATE. It would be extremely interesting to perform such future studies, with further laboratory testing (eg, muscle enzyme activity and D-dimer concentration), and to investigate the correlation of such tests with systemic and peripheral blood glucose concentration differences, to further characterize the severity of the lesions and the prognosis of acute ATE. Third, in most of the cats with ATE herein, the diagnosis of ATE was based only on clinical signs, and unconfirmed by advanced imaging or necropsy. This subset of cats is not an ideal population to be used for confirmation of a diagnostic test. Nevertheless, in our clinical setting, cat owners most often decline further testing when given a diagnosis of ATE and its grave prognosis. Nonetheless, the results show that ΔGlu and %ΔGlu in this subset of cats did not differ statistically from those of the definitely confirmed ATE cases in cats. Fourth, this study has investigated only cases of acute ATE with paralysis. Dogs with ATE of chronic onset, and cats with long-standing (days) ATE were excluded. Therefore, these results should not be applied to this set of animals, or to paretic animals with ATE, and future investigation of blood glucose in such cases is warranted. In such cases, collateral arterial circulation does occur,[2] which will likely positively affect glucose concentration in peripheral, affected limb blood samples. Fifth, there were significant age differences between the study groups, in both dogs and cats. In both animal species, both the ATE and the nonambulatory control groups animals were older compared to the ambulatory negative controls. Although age may potentially have affected blood glucose concentration, no blood glucose concentration differences were observed between the control groups in both dogs and cats. We therefore believe that these results were not affected by the age differences among groups. Sixth, the study utilized particular glucometer models, and therefore the ΔGlu and %ΔGlu cutoff values recommended herein may be somewhat different when other hand-held glucometers or autoanalyzers are utilized. Our results should be therefore confirmed using other analytical methods. In addition, specific RIs should preferably be set. Seventh, during the study period, 2 different glucometers were utilized. Although theoretically differences in their reading might exist, both are based on the same ISO standard and measurement method, and are produced by the same manufacturer. We therefore believe that such potential differences did not affect the validity of our results. Lastly, this study included glucose samples obtained from a single affected limb. In animals in which more than a single limb was affected, it would have been interesting to test samples from all of the affected limbs, and possibly, compare blood glucose concentrations among samples from all 4 limbs, and compare these to a jugular vein blood sample. This was not done in this study to minimize the potential risks (ie, infection) and animal discomfort; however, it should be considered in future studies, in light of the lack of complications and pain inflicted by the procedure in this study.

In conclusion, there is a significant difference between systemic and peripheral, affected-limb glucose

concentrations in cats and dogs presented with paralysis because of acute ATE, providing an accurate diagnostic tool for ATE in such cats and dogs. With clear ΔGlu and the %ΔGlu cutoff points, the study demonstrates this test to be highly sensitive (100%) and specific (90% in cats and 100% in dogs). The test it is cost-efficient, minimally invasive, highly available in every clinical setting, and issues instant, clear-cut results.

Footnotes

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[c] Accu-Chek sensor glucometer, Roche, Mannheim, Germany (years 2005–2010) and Performa's Accu-Chek glucometer (years 2010–2012), Roche, Mannheim, Germany

[d] Accu-Chek sensor glucometer and Performa's Accu-Chek glucometer manuals, Roche, Mannheim, Germany

[e] SPSS 18.0; SPSS Inc, Chicago, IL

Acknowledgement

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