Retrospective Study

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The association of blood lactate concentration with outcome in dogs with idiopathic immune-mediated hemolytic anemia: 173 cases (2003–2006)

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Abstract

Objective – To determine the association of blood lactate with outcome and response to transfusion therapy in dogs with idiopathic immune-mediated hemolytic anemia (IMHA).
Design – Retrospective study.
Setting – Urban veterinary small animal emergency hospital.
Animals – One hundred and seventy-three client-owned dogs with IMHA.
Interventions – None.
Measurements and Main Results – Serial blood lactate concentration, therapeutic interventions, and outcome were recorded. Nonsurvivors were defined as those that died or were euthanized. One hundred and thirty-three dogs (77%) survived, 35 (20%) were euthanized, and 5 (3%) died. One hundred forty-five dogs (84%; 145/173) had a lactate concentration above the laboratory reference interval [0.46–2.31 mmol/L] on presentation. Blood lactate at presentation was higher in the nonsurvivors (median 4.8 mmol/L; 0.5–13.6) compared with survivors (median 2.9 mmol/L; 0.3–13.2) ($P < 0.01$). All dogs presenting with hyperlactatemia that normalized (<2.0 mmol/L) within 6 hours of admission survived, whereas, 71% of dogs that had a persistent hyperlactatemia at 6 hours survived ($P = 0.034$). Lactate was positively correlated with age, BUN, and alkaline phosphatase, and inversely correlated with PCV. Receiver operating curve analysis for lactate concentration at admission as a test for outcome had an area under the curve of 0.69 with an optimal lactate cutoff concentration of 4.4 mmol/L correctly predicting outcome 73% of the time (sensitivity 60%, specificity 77%).
Conclusions – Lactate concentration at presentation was significantly higher in nonsurvivors than survivors. Lactate was significantly correlated with previously reported outcome variables but lactate concentration at admission, as a predictor for outcome was less than optimal. However, serial lactate concentration measurements may be more predictive as patients with persistent hyperlactatemia 6 hours after admission were less likely to survive. Prospective studies evaluating serial lactate concentration while controlling for other variables may provide further insight into lactate measurement as a prognostic indicator in animals with IMHA.

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Keywords: illness severity, lactate clearance, monitoring, prognostic indicators

Introduction

Canine immune-mediated hemolytic anemia (IMHA) can be a challenging clinical disease because of high complication rates, high cost of treatment, lack of an optimized treatment protocol, and often, an unidentified etiology. Reported survival rates for dogs with IMHA vary from 29% to 70%.[a,1–6] Several retrospective studies have suggested a variety of prognostic factors associated with mortality in IMHA including severity of anemia,[4] presence of autoagglutination,[1] thrombocytopenia,[1,6] leukocytosis,[1,3] absence of erythrocyte regeneration,[4] increased serum alkaline phosphatase,[2,6] increased total bilirubin,[a,1–4,6] and decreased serum concentration of albumin.[1,6] Many of these risk factors, however, require hematology and biochemical profiles, rather than a point-of-care test. An accurate and rapid method of survival prediction in dogs with IMHA would be advantageous.

The introduction of affordable and readily accessible hand-held analyzers has made lactate measurement a

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valuable technique in the assessment of a patient's hemodynamic status and response to therapy. Point-of-care lactate analyzers have been validated in dogs and cats using blood collected from direct venipuncture or peripheral IV catheters.[7,8] A reference interval for plasma lactate concentration of 0.3–2.5 mmol/L has been established for healthy, adult dogs depending on the site of blood collection and analyzer.[9] A plasma lactate concentration of <2 mmol/L has been recommended as a targeted end point of resuscitation in both veterinary and human patients.[10,11]

Plasma lactate concentration has been used clinically as a prognostic indicator for critically ill people, and hyperlactatemia has been associated with a higher mortality rate in patients with various diseases, including septic or hypovolemic shock,[11–18] cardiopulmonary arrest,[19] systemic inflammatory response syndrome,[20] and trauma.[21] Other studies have shown that the ability of a patient to clear lactate is a better predictor of the response to interventions (eg, fluid resuscitation) and patient outcome than single measurements.[21–25] Studies have also demonstrated that serial lactate measurement in people is a reliable assessment of response to initial and continued resuscitative therapy, thereby permitting appropriate modification of therapy in postoperative cardiovascular surgical patients.[22,23] Monitoring serial lactate concentrations has become standard of care in assessing response to therapy and guiding further intervention in critically ill humans.[13]

In veterinary medicine, measurement of plasma lactate concentration at admission has been a predictor of survival in gastric dilatation-volvulus,[26] babesiosis,[27,28] critical illness,[29] and injured dogs.[30] In addition to reflecting the severity of systemic hypoperfusion secondary to various disease states, measurement of plasma lactate has also been evaluated as a diagnostic test in septic abdominal effusions,[31,32] neoplastic abdominal effusions,[33,34] pericardial fluid,[35] and in arterial thromboembolism.[36] Serial measurement of blood lactate concentrations have been prospectively evaluated in dogs.[28,29] Nel et al[28] found that an increase in blood lactate or a $<50\%$ decrease in the blood lactate concentration at the 8- and 16-hour sampling interval was associated with increased mortality in canine babesiosis. Stevenson et al,[29] evaluated serial lactate concentrations in 80 dogs with various systemic illnesses. Dogs with lactate concentrations greater than the reference interval at 6 hours were 16 $\times$ more likely not to survive compared with dogs with lactate concentrations within the reference interval and hyperlactatemia that did not improve by $\geq 50\%$ within 6 hours was significantly associated with mortality.[29] These studies emphasize the importance of serial lactate concentrations in evaluating prognosis and response to therapy. Blood lactate concentrations on admission have not been evaluated as a predictor of survival in dogs with IMHA. Furthermore, serial lactate measurements as an indicator of outcome and response to therapy have not been evaluated in dogs with IMHA.

The primary goal of this study was to assess whether blood lactate concentration at admission was associated with outcome in dogs with IMHA. Our secondary aim was to determine if serial measurements of lactate could be used to assess patient outcome or response to transfusion therapy. In client-owned dogs with IMHA, we hypothesized that a higher blood lactate concentration on admission would be associated with a worse outcome and patients with a normal blood lactate concentration within 6 hours would be more likely to survive.

Materials and Methods

The computerized medical record database of the Matthew J. Ryan Veterinary Hospital at the University of Pennsylvania was searched to identify dogs that presented to the emergency service with a diagnosis of IMHA between January 2003 and October 2006. Criteria for inclusion in the study included a diagnosis of IMHA and a blood lactate measurement at time of presentation. Criteria for the diagnosis of IMHA were presence of anemia (PCV $<30\%$ at time of initial presentation) and at least 2 of the following: erythrocyte agglutination, positive direct antiglobulin test (Coombs test at 37 °C), presence of spherocytosis or hemoglobinemia.[37] Dogs with incomplete medical records or an identifiable underlying disease such as lymphoma, ehrlichiosis, or babesiosis were excluded from the study.

The following information was obtained from medical records (at presentation): duration of clinical signs, signalment, rectal temperature, heart rate, respiratory rate, presence of jaundice, mean arterial blood pressure, PCV, and total plasma proteins. Biochemical and hematological parameters included: RBC count, absolute reticulocyte count, WBC count, mature neutrophil count, band neutrophil count, platelet count, albumin, total bilirubin, potassium, and alkaline phosphatase. Serial venous blood gas measurements (including blood lactate, blood glucose, BUN, creatinine, ionized magnesium, and ionized calcium) were recorded for the first 24 hours after admission and total number of blood units transfused (packed RBC units) were recorded for each patient with respect to change in blood lactate concentration. 'Lactime,' defined as the time during which the blood lactate levels remained above 2.0 mmol/L, was calculated for each patient. A history of blood transfusion within 24-hours before admission, total number of blood units transfused, days of

hospitalization, and outcome (survival to discharge, died or euthanized) were also recorded.

All blood cell counts were determined by use of an automated analyzer[b] and blood smears were manually examined for verification of cell counts and detection of spherocytosis. Autoagglutination was differentiated from rouleaux by the saline dispersion test.[37] Blood lactate concentration was determined by a bench-top blood gas analyzer[c] using heparinzed blood samples obtained from the cephalic, saphenous, or jugular vein.

Dogs were categorized as either survivors (survival to discharge) or nonsurvivors (died or euthanized). All continuous variables were visually inspected and evaluated for normality using the Shapiro-Wilks test. Nonparametric data were reported as the median (minimum value, maximum value). Parametric data were reported as mean (SD). Proportions are reported as percent. The Mann-Whitney test or Kruskal-Wallis test was used to compare continuous variables between groups, as the majority of continuous variables were not normally distributed. Categorical data were compared using the χ^2-test and Fisher's exact test. The Spearman rank-correlation method was used to assess the relationship between lactate concentration and other continuous variables. The trapezoid method was used to determine the area under the curve for the receiver operating characteristic analysis. A P-value of <0.05 was considered significant for all analyses. All data analyses were performed using commercially available statistical software.[d]

Results

One hundred and eighty-nine dogs were identified with the diagnosis of IMHA. Sixteen of these dogs were excluded for the following reasons: 6 were determined to have underlying diseases and 10 had incomplete records. Thus, 173 dogs were included in the study, of which 133 dogs (77%) survived and 40 (23%) did not survive (5 died and 35 were euthanized).

Signalment

For all 173 dogs the mean age was 6.9 (3.5) years. Ninety-four (54.4%) were spayed females, 17 (9.8%) were intact females, 54 (31.2%) were castrated males, and 8 (4.6%) were intact males. The most common breeds were mixed breed dog ($n = 38$), Labrador Retriever (10), Shih Tzu (10), Cocker Spaniel (10), Maltese (7), American Pit Bull Terrier (7), Miniature Schnauzer (5), English Springer Spaniel (5), Beagle (5), Boxer (5), Dachshund (5), and Jack Russell Terrier (5). All other breeds had <5 dogs in each breed. Sex ($P = 0.5749$), weight ($P = 0.4578$), and neuter status ($P = 0.203$) were not significantly associated with outcome.

Selected historical, physical, and clinicopathologic findings

History and physical exam findings, such as, duration of clinical signs ($P = 0.3451$), rectal temperature ($P = 0.8348$), heart rate ($P = 0.2852$), and respiratory rate ($P = 0.2100$) were not significantly different between outcome groups. There was no correlation between HCT and heart rate at admission ($P = 0.6484$). In those patients where the physical examination commented on the mucous membrane color ($n = 171$), the presence of jaundice was noted more frequently ($P = 0.007$) in nonsurvivors (21/40, 53%) compared with survivors (37/131, 28%). The median Doppler blood pressure of survivors (111 mm Hg, range 36–200 mm Hg) was not significantly different ($P = 0.5762$) than nonsurvivors (104 mm Hg, range 43–180 mm Hg). BUN, total bilirubin, and alkaline phosphatase were significantly higher in nonsurvivors than in survivors. HCO_3 concentration and base excess were significantly lower in nonsurvivors when compared with those of survivors. Table 1 summarizes the clinicopathologic variables that were assessed as individual prognostic indicators of outcome in 173 dogs with IMHA.

Lactate at presentation

Eighty-four percent (145/173) of the dogs had a blood lactate concentration above the bench-top analyzer[f] reference interval (0.46–2.31 mmol/L) on admission. Dogs with an increased blood lactate concentration were less likely to survive (OR 1.34, 95% CI 0.5, 5.0) compared with dogs with blood lactate concentration within the reference interval but this was not statistically significant ($P = 0.54$). Median blood lactate concentration was significantly lower ($P < 0.01$) in survivors (2.9 mmol/L, 0.5–13.6 mmol/L) compared with nonsurvivors (4.8 mmol/L, 0.3–13.2 mmol/L) (Table 2). Blood lactate was positively correlated with age ($P = 0.0024$), heart rate ($P = 0.0034$), and BUN ($P = 0.0003$), and inversely correlated with HCT ($P = 0.0004$) (Figure 1). Receiver operating curve analysis (Figure 2) shows that a blood lactate concentration of 4.4 mmol/L yielded the best accuracy for predicting outcome. This cutoff point yielded 60% sensitivity, 77% specificity, and 73% positive predictive values for nonsurvival (Figure 2).

Serial lactate measurements

Serial blood lactate measurements were available at 6 hours ($n = 129$), 12 hours ($n = 54$), and 24 hours ($n = 16$) postadmission. Median lactate was significantly different between survivors and nonsurvivors at admission (2.9 versus 4.8 mmol/L; $P = 0.0002$), 6 hours (1.8 versus 2.5 mmol/L; $P = 0.02$), and 12 hours (1.5 versus 2.1 mmol/L; $P = 0.03$) after admission (Figure 3).

Table 1: Clinicopathologic variables in 173 dogs with IMHA at initial presentation

Variable	Nonsurvivors (NS) median (range)	Survivors (S) median (range)	*P*-value	*n* (NS/S)
PCV (%)	17 (6–30)	19 (5–30)	0.0915	40/133
Total plasma proteins (g/dL)	7 (4.3–9)	7 (4.3–10.4)	0.9768	40/133
Total plasma proteins (g/L)	70 (43-90)	70 (43-104)		
pH	7.4 (7.2–7.5)	7.4 (7.2–7.6)	0.1461	40/133
Sodium (mmol/L)	147 (138–159)	146 (130–155)	0.8188	40/128
Potassium (mmol/L)	3.7 (2.6–8.7)	3.8 (2.6–6.7)	0.0892	40/130
Chloride (mmol/L)	116 (106–125)	115 (97–133)	0.5188	40/127
Ionized calcium (mmol/L)	1.2 (0.9–1.5)	1.2 (1.0–1.5)	0.1149	40/133
Ionized calcium (mg/dL)	4.8 (3.6–6)	4.8 (4–6)		
Ionized magnesium (mg/dL)	0.4 (0.2–0.7)	0.4 (0.2–0.7)	0.6045	40/131
Ionized magnesium (mmol/L)	0.2 (0.1–0.3)	0.2 (0.1–0.3)		
Glucose (mg/dL)	114 (34–337)	112 (42–320)	0.8909	39/125
Glucose (mmol/L)	6.3 (1.9–18.7)	6.2 (2.3–17.8)		
Urea (mg/dL)	28 (11–111)	22 (9–85)	0.0002*	37/124
Urea (mmol/L)	10 (3.9–39.6)	7.9 (3.2–30.4)		
Creatinine (mg/dL)	0.8 (0.4–6.5)	0.7 (0.5 -2.5)	0.2167	16/63
Creatinine (μmol/L)	70.4 (35.2–572)	61.6 (44–220)		
HCO_3 (mmol/L)	18.5 (8.4–31.4)	19.8 (7.7–29)	0.0283*	38/131
Base excess (mmol/L)	− 6.3 (− 18.5–8.3)	− 4.8 (− 19.7–5.2)	0.0213*	38/131
White cell count (×10e3/μL)	23.7 (5.6–85.2)	24.8 (6.3–98.8)	0.3560	37/126
Total bilirubin (mg/dL)	3.9 (0.1–80.3)	1.3 (0.1–63.2)	0.0007*	36/119
Total bilirubin (μmol/L)	66.7 (1.7–1373)	22.2 (1.7–1080)		
Alkaline phosphatase (U/L)	377 (94–1580)	231 (45–13920)	0.0004*	35/119
Albumin (g/dL)	3.1 (1.5–7.2)	2.9 (1.2–7.3)	0.2904	36/120
Albumin (g/L)	31 (15–72)	29 (12–73)		

*$P < 0.05$ considered significant.
IMHA, immune-mediated hemolytic anemia.

Lactime

The time during which blood lactate exceeded 2.0 mmol/L (lactime) was calculated for 85 patients and was increased for a mean of 7.7 (3.7) hours. The lactime was incalculable in 88 patients because the last lactate measurement recorded was still above 2.0 mmol/L ($n = 39$) or paired lactate measurements were not available ($n = 49$). Patients presenting with hyperlactatemia that was reduced to <2.0 mmol/L within 6 hours of admission had a 100% survival rate, whereas those that had persistent hyperlactatemia at 6 hours had a significantly lower survival rate of 71%; $P = 0.034$.

Table 2: Blood lactate concentration on admission in dogs with IMHA

Group	Median lactate (mmol/L)	Lactate range (mmol/L)	*n*
Survivors	2.9*	(0.3–13.3)	133
Nonsurvivors died	5.1*	(2.4–12.1)	5
Nonsurvivors euthanized	4.7*	(0.5–13.6)	35

*P-valve < 0.001 for all groups.
IMHA, immune-mediated hemolytic anemia.

Lactate and transfusion therapy

Twenty-six dogs received a blood transfusion within the 24-hour period before admission. There was no significant difference in the median presenting lactate concentrations between dogs that received a packed RBC transfusion before admission (3.0 mmol/L;

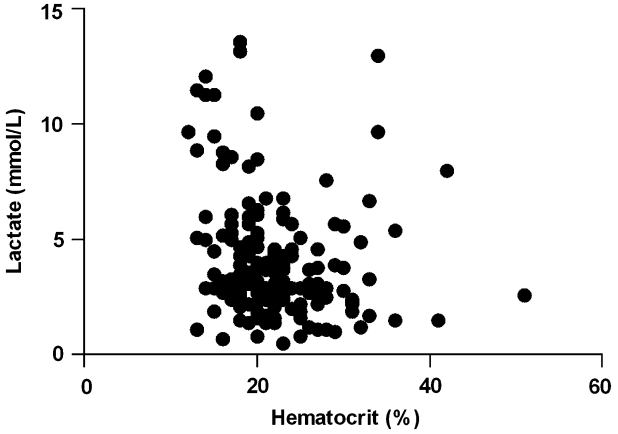


Figure 1: Correlation between lactate concentration and HCT on admission in dogs with immune-mediated hemolytic anemia. Lactate shows a significant correlation with HCT ($P = 0.0014$, $r = 0.2452$).

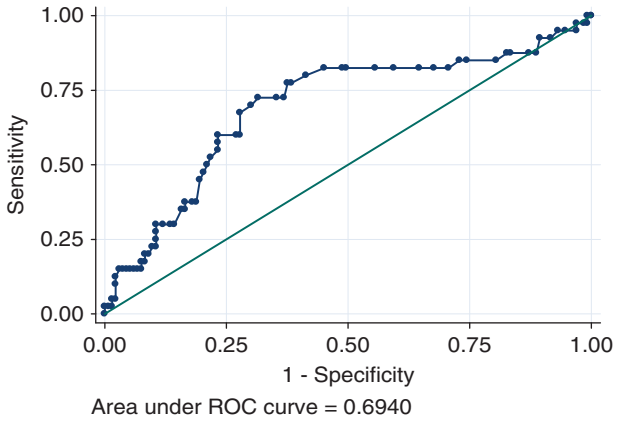
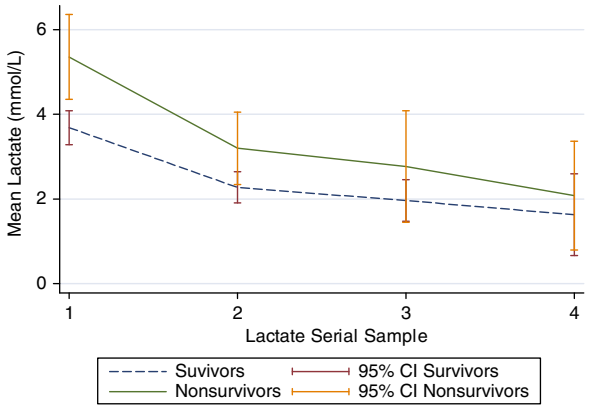


Figure 2: Lactate receiver operating curve (ROC) assessing non-survival in dogs with immune-mediated hemolytic anemia. ROC analysis shows that a lactate concentration of 4.4 mmol/L yielded the best accuracy for predicting outcome (60% sensitivity, 77% specificity).

1.1–10.5 mmol/L) and those that did not (3.4 mmol/L; 0.3–13.6 mmol/L, $P = 0.4232$). There were 129 dogs in which paired lactate measurements were available, of which 79 dogs received a packed RBC transfusion at admission and 49 that did not. Dogs that were transfused within the first 6 hours after admission (before the paired lactate measurement) had a significantly greater change in their median lactate concentration (-1.9 mmol/L; -0.8 to -2.6 mmol/L) than those dogs that were not (-1.2 mmol/L; -0.3 to -2.0 mmol/L, $P = 0.0478$) (Figure 4). There was no correlation between presenting lactate concentration and number of blood transfusions in hospital ($r = 0.113$; $P = 0.17$).

Discussion

Increased blood lactate concentration develops when the rate of lactate production in hypoxic tissue exceeds the rate of lactate metabolism in the body. Hyperlactatemia in dogs with IMHA may be the result of severe anemia or systemic hypoperfusion leading to decreased delivery of oxygen to the tissues. Dogs with the greatest degree of anemia and tissue hypoxia, and therefore the highest lactate concentration, would be expected to have the greatest risk of multiorgan failure and subsequent death. In the present study, the median lactate concentration on admission was significantly higher in nonsurvivors (4.8 mmol/L) compared with survivors (2.9 mmol/L). Using receiver operating curve analysis, a lactate concentration of 4.4 mmol/L provided an overall accuracy of 73% in predicting outcome but only yielded 60% sensitivity and 77% specificity. These findings indicate a substantial overlap in admission lactate concentrations between survivors and



	Sample 1 (Admission)	Sample 2 (6 hr)	Sample 3 (12 hr)	Sample 4 (24 hr)
Non-survivors	4.8 (0.5-13.6)	2.5 (0.4-11.8) *	2.1 (1.5-9.2) *	1.7(1-4.4) *
Survivors	2.9 (0.3-13.2) Π	1.8 (0-12.5) * Π	1.5 (0.2-7.7) * Π	1.2(0.4-4.7) *
n	173	129	54	16

Lactate (mmol/L), expressed median (range).
* Significantly different from admission (P<0.05).
Π Significantly different between survivors and non-survivors at admission (P=0.0002), 6 hours (P=0.02), and 12 hours (P=0.03) after admission.

Figure 3: Blood lactate concentrations in immune-mediated hemolytic anemia dogs over 4 time points in the first 24 h after admission.

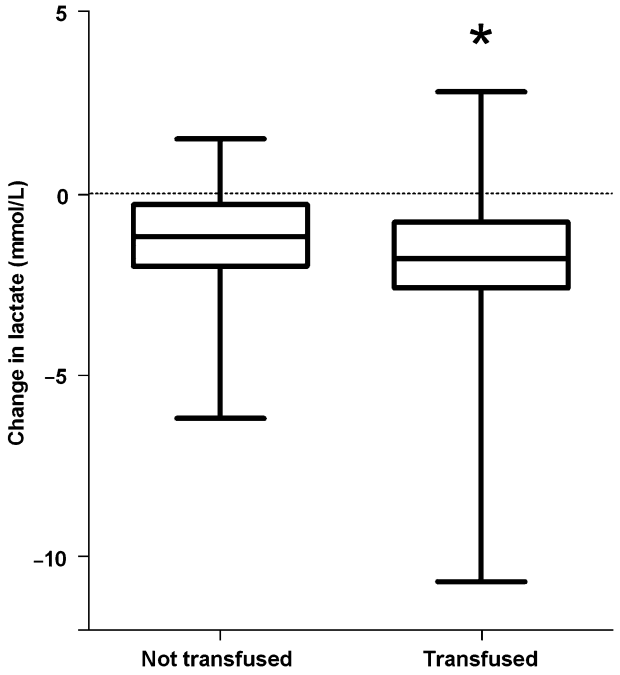


Figure 4: Blood lactate change with respect to transfusion therapy on admission in dogs with immune-mediated hemolytic anemia. In this boxplot the box represents the 25th–75th percentile and the line within the box is the median value. The lines extending vertically from the box represent the range of values. Note the significant change in lactate in those dogs that were transfused on admission compared with those that were not transfused. *P-value <0.05.

nonsurvivors indicating that measurement of blood lactate at admission is a poor discriminator of outcome.

Similar to previous studies we found that nonsurvivors had a more severe hyperbilrubinemia and a higher alkaline phosphatase when compared with survivors, however, we could not establish a statistically significant association between severity of anemia, hypoalbuminemia, leukocytosis, and survival in dogs with IMHA.

Serial blood lactate measurements have been used in early goal directed therapy in both human and veterinary patients and have been shown to be a more accurate predictor of mortality and response to therapy compared with a single measurement.[11,20] Vincent[14] evaluated serial arterial lactate concentrations during the resuscitation of human patients with noncardiogenic shock. Patients with a decrease in blood lactate concentration following resuscitation with fluid therapy survived, while those with persistently increased blood lactate concentrations despite fluid therapy did not survive.[25] Dogs in the survivor group had a median lactate of 1.8 mmol/L (reference interval 0.46–2.31 mmol/L), following 6 hours of therapy. This is contrasted with the nonsurvivor group, which had a median lactate of 2.5 mmol/L (reference interval 0.46–2.31 mmol/L) ($P = 0.02$) that remained above the reference interval despite therapy (Figure 2). Median blood lactate was also significantly different between survivors and nonsurvivors at 12 hours (1.5 versus 2.1 mmol/L; reference interval 0.46–2.31 mmol/L) after admission.

Serial blood lactate measurements can be used to calculate a lactime[11] (defined as the time during which the blood lactate levels exceeded 2.0 mmol/L). In human medicine, a greater lactime[11] or failure to normalize lactate within 24 hours[38] has been shown to be more sensitive indicators of survival than a single measurement. In a study evaluating blood lactate clearance in trauma patients, all patients whose arterial lactate concentration normalized within 24 hours survived, whereas the survival rate decreased to 75% if blood lactate concentrations did not normalize until 48 hours.[21] Nguyen et al[24] evaluated the prognostic importance of early blood lactate clearance in people with severe sepsis or septic shock, and found that a higher arterial blood lactate *clearance* within 6 hours of initial resuscitation was associated with improved survival compared with a more prolonged blood lactate clearance time.[24] Lactime, or duration (h) of hyperlactatemia was calculated in 85 dogs in the current study. Seventy-one percent of dogs that had a persistent hyperlactatemia 6 hours after admission survived, whereas dogs that presented with hyperlactatemia that normalized (<2.0 mmol/L) within 6 hours of admission had a 100% survival rate. The time for blood lactate clearance may be a better prognostic indicator than blood lactate on admission in dogs with IMHA. However, definitive conclusions cannot be made from these findings as confounding factors such as variations in transfusion and fluid therapy timing and the small number of subjects at each time point. A prospective study evaluating the utility of resuscitating dogs to a goal lactate of <2.0 mmol/L within 6 hours of admission would provide more standardized information to make conclusions.

This study evaluated blood lactate concentration with a bench-top analyzer,[f] but several hand-held point-of-care analyzers have been shown to be in agreement with laboratory blood analyzers.[7,8] With the wide availability of automatic point-of-care analyzers, blood lactate measurement could easily become part of the initial database performed on IMHA dogs presented to an emergency service.

Experimental studies in dogs have shown that hyperlactatemia occurs with decreased HCT.[39,40] If lactate accumulation is proportional to the severity of anemia and secondary decreased oxygen carrying capacity,

then with appropriate resuscitation efforts lactate should decrease or fall into normal range. In the current study, heart rate did not correlate with HCT on admission whereas there was a significant positive correlation between blood lactate concentration and HCT suggesting that heart rate is not a sensitive indicator of occult tissue hypoxia in dogs with IMHA. Because blood lactate is significantly correlated with HCT we evaluated the effect of transfusion therapy on lactate concentration. There was no difference in presenting blood lactate concentration between dogs that received or did not receive a transfusion within 24 hours before referral. This finding may be due to an inadequate transfusion volume before referral or ongoing transfusion requirements of the patient. Dogs that were transfused on admission had a significantly greater change in their blood lactate concentration (-1.9 mmol/L) than dogs that were not transfused on admission (-1.2 mmol/L) ($P = 0.0478$). This suggests that a quantitative resuscitation strategy that includes targeting blood lactate concentration of <2.0 mmol/L in the first 6 hours may have a beneficial effect on patient outcome.

Although blood lactate concentration at admission was significantly increased in nonsurvivors and significantly correlated with other variables associated with outcome, this evidence is insufficient to advocate using admission blood lactate concentrations as an unequivocal prognostic indicator for outcome in dogs with IMHA. However, this study suggests that using serial lactate measurements and lactate concentration to guide transfusion therapy in conjunction with conventional hemodynamic markers may be of benefit. Limitations to this study include inconsistent record keeping and large variations in the time and number of lactate measurements performed in relation to treatment. The small number of patients in the nonsurvivor group that died ($n = 5$) and the large number of patients that were euthanized ($n = 35$) was also a limitation. It was inferred through medical record data that patients were euthanized due to severity of clinical signs or perceived poor prognosis but other previously determined prognostic indicators combined with financial considerations may have influenced the decision for euthanasia. Additionally, there was no standardization of therapies, which may have influenced the rate of lactate clearance.

From this study we can conclude that a single measurement of blood lactate at admission is not a reliable predictor of outcome; however, monitoring serial blood lactate concentrations is a more accurate predictor of mortality and is useful in guiding transfusion therapy. Prospective studies are warranted to provide further insight into the value of measuring blood lactate concentration as a predictor of outcome in dogs with IMHA. In addition, studies evaluating serial measurements of blood lactate concentration may provide both prognostic information and help guide transfusion therapy in these patients.

Footnotes

[a] Davidow EB, Oncken A. Clinical and laboratory factors associated with prognosis in IMHA cases – a prospective study (abstr). J Vet Emerg Crit Care. 2004;14:S1–S17.
[b] Cell-Dyn 3700, Santa Clara, CA.
[c] NOVA Statlabs Profile 16, Biomedical, Waltham, MA.
[d] Stata 8.0 for Windows, College Station, TX.

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