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Research article

Pre- and post-surgical evaluation of plasma lactate concentration in 45 dogs with gastric dilatation-volvulus: A preliminary study

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ABSTRACT

This preliminary study was designed to contribute to the evaluation of reliability of plasma lactate concentration (PLC) and its clearance as predictive and prognostic factors of gastric necrosis and clinical outcome of dogs affected by gastric dilatation-volvulus (GDV). Main aims of the study were: 1) to evaluate the prognostic reliability of PLC at presentation (T0) in dogs with GDV, 2) to compare the obtained data and considerations with the veterinary literature, and 3) to introduce the possible validity of PLC values at 24 (T24) and 48 (T48) hours after surgery as a predictive factor.

Dogs with GDV were retrospectively evaluated. PLC at T0, T24 and T48 were recorded and correlated to the presence or absence of macroscopic necrosis of the stomach and to outcome.

Forty-five dogs met the inclusion criteria. Significant differences were not detected in the mean values between the initial PLC in dogs with and without necrosis of the gastric wall, as well as between surviving and non-surviving dogs; these values were not associated with higher risk of gastric necrosis or death.

At T24 and T48 no significant differences were recorded between necrosis and non-necrosis, and surviving and non-surviving categories.

A median plasma lactate concentration clearance from arrival to T24 $\geq$ 50% was identified in both groups (with and without necrosis), and this parameter failed in identifying dogs that survived to discharge.

In conclusion, the results presented here failed to detect PLC at T0 and its clearance at T24 as prognostic factors in this population of dogs with GDV.

1. Introduction

Gastric dilatation-volvulus (GDV) is one of the most frequent emergency conditions affecting large and giant breed dogs. The pathological mechanism entails acute dilatation of the stomach with cardiac and pyloric obstruction, with clockwise- or, rarely, counterclockwise volvulus on the mesenteric axis (Glickman et al., 2000). High mortality rates are reported for GDV, between 10% and 43% in most recent studies (Beck et al., 2006; Buber et al., 2007; Mackenzie et al., 2010; Zacher et al., 2010; O'Neill et al., 2017). Gastric necrosis significantly increases mortality by 25–59% (Buber et al., 2007; Zacher et al., 2010); Green et al. (2011) described it as the only aspect significantly affecting clinical outcome (survival or death) in its GDV dogs.

In veterinary literature, several factors (e.g. plasma lactate concentration, pepsinogen, procalcitonin, lipase) have been examined as prognostic factors to predict gastric necrosis onset and survival. However, high variability in results has been shown by many studies (De Papp et al., 1999; Shober et al., 2002; Zacher et al., 2010; Green et al., 2011; Israeli et al., 2012; Santoro Beer et al., 2013; Mooney et al., 2014; Spinella et al., 2018; Troia et al., 2018).

Lactates are negative ions deriving from a reduction of the redox potential from pyruvate, in low presence of oxygen (Mooney et al., 2014).

Hyperlactatemia can derive from several causes and it can be categorized as types A and B. Type A is described with clinical evidence of tissue low oxygen. Increased muscle activity (exercise, shivering,

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tremors) produces relative oxygen deficiency and physiological hyperlactatemia, which should resolve 30–60 min after stopping muscle activity. The reported range of exercise-induced hyperlactatemia in racing Greyhounds varies from 4.5 to 30 mmol/L (Pieschl et al., 1992; Robergs et al., 2004; Rovira et al., 2007; Mooney et al., 2014). Conversely, global hypoperfusion causes absolute tissue oxygen deficiency as occurs in case of shock (hypovolemic, cardiogenic, obstructive). Type *B hyperlactatemia* could occur even if clinical signs are not observable and three subtypes are described: B_1– related to an underlying disease (sepsis or systemic inflammatory response syndrome); B_2– associated with toxins or administration of drugs (adrenaline and noradrenaline); or B_3– arising from hereditary or congenital metabolic defects (Mooney et al., 2014).

Hyperlactatemia is frequently present in GDV dogs. Type A hyperlactatemia can be induced by global hypoperfusion linked to hypovolemia resulting from intragastric fluid sequestration, reduced venous return, maldistributive shock or their combination (Mooney et al., 2014). Type B_1 might be present, caused by sepsis, septic shock or SIRS, characterized by bacteremia or bacterial translocation from the gastric/enteric lumen to the blood, due to a damaged gastric wall in presence of gastric necrosis and intestinal ischemia (Mooney et al., 2014). Lactate produced by the spleen and gastric wall necrosis might enter the systemic circulation during the decompression of the stomach (Mooney et al., 2014).

Gastric necrosis was correlated with higher risk of complications, hospitalization costs (De Papp et al., 1999) and lower survival rate: in studies by De Papp et al. (1999), Zacher et al. (2010), Green et al. (2011) and Santoro Beer et al. (2013), survival rate ranged from 38% to 75%, compared to an overall survival rate, ranging from 77% to 89%.

During the past twenty years, several studies were conducted to determine whether PLC and lactate clearance can be predictors of gastric necrosis and survival.

De Papp et al. (1999) detected an initial PLC of 6.0 mmol/L as a valuable predictor of stomach necrosis and death. This cut-off value was subsequently increased to 7.4 mmol/L by Santoro Beer et al. (2013) and to 9.0 mmol/L by Zacher et al. (2010). Green et al. (2011) detected two different, lower cut-off values: 2.9 mmol/L as a predictor of gastric necrosis and 4.1 mmol/L for survival. Considering the studies which indicate the initial PLC cut-off points that are likely to predict the presence of gastric necrosis (De Papp et al., 1999; Santoro Beer et al., 2013), they are compatible with the cut-off values predicting survival. As gastric wall necrosis is the factor that mostly affects outcome, this finding was expected.

Mooney et al. (2014) summarized the findings of these studies, indicating a compromise among their data, concluding that an initial PLC>6 mmol/L should be considered as a good indicator of possible gastric necrosis and expected higher complications and hospitalization expense, while a PLC<4 mmol/L is an indicator of more likely survival. However, authors strongly suggested performing an exploratory laparotomy to verify gastric necrosis, because there was no full methodological consensus among the studies. Mooney et al. (2014) also advises communicating to the dogs' owners that when the initial PLC is within the reference range or moderately heightened, complications are less probable but still possible.

Zacher et al. (2010) observed that, following initial resuscitation fluid treatment, changes in PLC may assist in determining the prognosis; in particular, it was underlined that PLC clearance of ≤42.5% and absolute decrease of ≤4 mmol/L, were associated with significantly lower survival (Zacher et al., 2010). Green et al. (2011) were in line with this study, concluding that a decrease of ≥50% of the median initial PLC within 12 h after surgery was a good indicator for survival. At date, to the authors' knowledge, these studies investigated the importance of PLC variation in some periods of time, but there are no data about a possible prognostic validity of its modification at 24 and 48 h after surgery, intervals proposed in this study.

Aim of our study was to retrospectively evaluate PLC in dogs with GDV at presentation, at 24 and 48 h after surgery, as a survival indicator, in relation to the presence of gastric wall necrosis and outcome. Data were compared with literature about the validity of using PLC as a predictive factor.

2. Materials and methods

Dogs with GDV presented to the University Veterinary Hospital between 1st April 2012 and 1st July 2017 were collected examining surgical case logs and medical records. Only cases supported by radiographs or abdominal exploratory surgery were included. Dogs with GDV were excluded when admission PLC was not available or if they received any medical treatments before arrival.

Computerized case logs were examined to record administered treatments and pre-surgical procedures, description of surgical procedure (focusing on the presence of gastric wall necrosis), complications, animal's condition during hospitalization, registered PLC values and outcome (death, euthanasia, survival).

PLC was retrospectively recorded at arrival (T0), at 24 (T24) and 48 h (T48) after surgery. All animals were classified by presence or absence of surgically assessed gastric necrosis and outcome.

Data were statistically studied to evaluate if there was a cut-off T0 PLC value which could provide a prognostic indication for stomach necrosis and for outcome.

Moreover, PLC values recorded at T24 and T48 were statistically analysed to assess their potential importance as prognostic factors.

The preoperative treatment was similar for all dogs: intravenous fluid resuscitation, antibiotic therapy and supporting care. All dogs underwent gastric decompression performed via orogastric tubing and/or percutaneous 18-G needle, followed by an explorative laparotomy to assess the state of gastric wall, reposition the stomach and perform right gastropexy. All dogs presenting massive gastric necrosis were intraoperatively euthanised with owner's consensus, while dogs with smaller, surgically approachable necrotic areas were treated by gastrectomy.

The anaesthetic protocol consisted of premedication with methadone (0.2–0.3 mg/kg IM; Synthadon, Le Vet Pharma), induction with propofol (1–2 mg/kg IV; Proposure, Merial), tracheal intubation and maintenance with isoflurane (Vetflurano, Virbac) mixed with pure oxygen.

After surgery, all animals were monitored at the critical care unit for 3–5 days. A focused diet and restricted activity were suggested to the owners for 7–10 days after discharge together with the administration of antibiotic and gastroprotective drugs.

Standard descriptive statistics were used to express data: they were presented as mean ± Standard Deviation (SD) or median and ranges, based on their distribution. All data were submitted to statistical analysis using Fisher's exact test with a significance value of $P \leq 0.05$. A probability model for predicting necrosis and survival was carried out using a logistic regression, detecting the cut-off value (sensitivity and specificity) using the receptor operating characteristics (ROC) curve. The risk of gastric necrosis presence was performed using the Cox proportional hazards regression model for survival analysis. $P < 0.05$ was considered significant. All statistical analyses were performed using Stata v.14 software (StataCorp LP, College Station).

3. Results

Forty-five dogs were included: 30 males (six neutered) and 15 females (five spayed). German Shepherd (15) was the most common breed: other breeds included mixed large breed dogs (nine), Doberman Pinschers (six), Great Danes (five), Basset Hounds (one), Belgian Shepherd (one), Corso Dog (one), Irish Setter (one), German Shorthaired Pointer (one), Leonberger (one), Maremma Sheepdog (one), Neapolitan Mastiff (one), Saint Bernard (one) and Siberian Husky (one). Mean body weight was 39 kg (range 23–65 kg) and mean age was 8.4 years (range 1–14 years).

Thirty-one of the 45 dogs (69%) survived to discharge, while 14 (31%) dogs did not survive. Ten dogs were intraoperatively euthanised because of wide gastric wall necrosis, one dog died spontaneously during

Table 1. PLC mean ± SD values observed in the study. The PLC values registered at T0, T24 and T48 in dogs with and without gastric wall necrosis, as well as in surviving and non-surviving dogs, are reported as mean ± SD. In square brackets the number of patients (P) with available data.

		[P] T0 (mmol/L)	[P] T24 (mmol/L)	[P] T48 (mmol/L)
Gastric necrosis	Yes	[13] 5.0 ± 3.2[a]	[4]1.1 ± 0.2	[2] 3.0 ± 1.6
	No	[32] 4.1 ± 3.8[a,b]	[28] 1.7 ± 1.6	[17] 1.0 ± 0.5
Survival	Yes	[31] 4.2 ± 3.2[a,b]	[29] 1.4 ± 0.7	[17] 1.1 ± 0.5
	No	[14] 4.9 ± 3.8	[3] 3.6 ± 4.7	[2] 2.4 ± 2.5

[a] significantly different from T24.
[b] significantly different from T48.

Table 2. PLC Median values observed in the study. The PLC values registered at T0, T24 and T48 in dogs with and without gastric wall necrosis, as well as in surviving and non-surviving dogs, are reported as Median (range). In square brackets the number of ptients (P) with available data. NA = Not Applicable, because only 2 dogs were included in these groups. The PLCs of the two patients are reported in brackets and presented as absolute values.

		[P] T0 (mmol/L)	[P] T24 (mmol/L)	[P] T48 (mmol/L)
Gastric necrosis	Yes	[13] 3.2 (0.9–12.7)	[4] 1.15 (0.8–1.4)	[2] NA (1.9–4.2)
	No	[32] 3.2 (0.9–16.4)	[28] 1.2 (0.6–9.1)	[17] 0.9 (0.6–2.5)
Survival	Yes	[31] 3.2 (0.9–16.4)	[29] 1.2 (0.6–4.2)	[17] 0.9 (0.6–2.5)
	No	[14] 3.25 (2.1–12.7)	[3] 1.1 (0.7–9.1)	[2] NA (0.6–4.2)

the surgical procedure and one a few hours later. One dog was euthanised on owner request in the immediate postoperative period because of the recurrence of severe gastric dilatations and one for severe systemic consequences of the GDV. The PLC values of 32 patients were available at T24 (29 surviving and three non-surviving) and of 19 at T48 (17 surviving and two non-surviving).

Thirteen of 45 dogs (29%) presented macroscopic gastric necrosis: 10 dogs out of this group of 13 (77%) did not survive to discharge.

No PLC significant differences were detected at T0 between dogs with and without necrosis, and between surviving and non-surviving dogs. The same statistical results were detected at T24 and T48: no significant differences were noted between dogs presenting and non-presenting gastric necrosis and between surviving and non-surviving patients. Nevertheless, dogs which died recorded mean values higher than 1.5 mmol/L (normal range), both at T24 and T48. The same consideration was found for dogs with gastric necrosis at T48. Mean ± SD at T0, T24 and T48 are indicated in Table 1.

In our population, the logistic regression model used to predict necrosis and survival showed a T0 PLC of >2.55 mmol/L as the value presenting the best compromise between sensitivity (85%) and specificity (44%) for determining necrosis, and a T0 PLC of <4.45 mmol/L (sensitivity 33% and specificity 77%) as a good parameter for predicting survival.

Thirteen of 45 dogs (29%) recorded a T0 PLC≤2.55 mmol/L, while 30 of 45 (67%) had a T0 PLC<4.45 mmol/L. Eleven of the 31 surviving dogs (35%) registered an initial PLC≥4.45 mmol/L, while among the 14 dogs which did not survive, only four (28%) had a T0 PLC≥4.45 mmol/L. Furthermore, 11 dogs out of the 13 (85%) with macroscopic gastric wall necrosis presented a T0 PLC>2.55 mmol/L.

These values seem to demonstrate a low but not significant ($P > 0.05$) increase of the risk of necrosis and death in the population.

The results are not far from Green et al. (2011) results, which proposed a PLC cut-off of 2.9 mmol/L and of 4.1 mmol/L as the best cut-offs for predicting survival and absence of gastric necrosis in its population of dogs.

Considering Green's cut-offs and applying them to our dogs' population, 19 of 45 dogs (42%) had a T0 PLC≤2.9 mmol/L and 28 of 45 (62%) presented a T0 PLC<4.1 mmol/L. Among the 31 dogs which survived to discharge, 11 (35%) registered an initial PLC≥4.1 mmol/L; on the other hand, among the 14 non-surviving dogs, only six (43%) had a T0 PLC≥4.1 mmol/L. Moreover, eight dogs out of the 13 (62%) which presented macroscopic gastric necrosis had a T0 PLC>2.9 mmol/L.

Statistical analysis studied on the application of Green et al. (2011) cut-offs to this population of dogs, showed there was no significantly higher risk of gastric necrosis presence when T0 PLC was >2.9 mmol/L, and there was no significantly higher risk of a bad outcome in dogs presenting T0 PLC≥4.1 mmol/L.

Mean ± SD PLCs recorded at T24 and T48 were significantly different from T0 PLCs for dogs without necrosis and for surviving dogs; differently, a significant difference was only observed between T0 and T24 for dogs with gastric necrosis. Non-surviving dogs did not present any differences among T0, T24 and T48. No significant differences were found between PLCs at T24 and T48 for each category (Table 1).

Relatively to median values, they revealed a decrease of ≥50% at T24 in all the considered categories. Median PLC decrease of ≥50% was confirmed also at T48 in patients without gastric necrosis and surviving animals (Table 2).

4. Discussion

The role of PLC as predictor and prognostic factor in dogs with GDV is highly debated in veterinary literature, with contradictory indications and results (De Papp et al., 1999; Zacher et al., 2010; Green et al., 2011; Israeli et al., 2012; Mooney et al., 2014). Aim of this retrospective study was 1) to evaluate the prognostic reliability of PLC at presentation in dogs with GDV, 2) to compare the obtained data and considerations with veterinary literature, and 3) to introduce the possible validity of PLC at 24 and 48 h after surgery as predictive factors.

Our data recorded a ≥50% decrease of the median PLC from T0 to T24 in both groups of dogs without gastric necrosis and with good outcome; however, the same data were registered also for dogs with gastric necrosis and non-surviving dogs. As a decrease of ≥50% of the median values was detected for all the considered categories, this parameter cannot be probably used as a reliable indicator of good or bad prognoses. Different considerations were done by Zacher et al. (2010) and by Green et al. (2011), which detected that a PLC clearance of ≤42.5% after fluid resuscitation was associated with significantly lower survival, and of ≥50% of the median initial PLC within 12 h after the first measurement was a good indicator for survival, respectively. The retrospective nature of our study did not allow us to evaluate PLC clearance after fluid resuscitation or at T12, but only at T24. We hypothized that a correct and timely resuscitation, probably associated with the severe torsion of the stomach, has promoted a decrease in blood lactate concentration as a result of lactates entrapment within the gastric lumen, simulating an improvement in the PLC.

Statistical analysis did not underline a significantly higher risk of gastric necrosis presence and death considering the T0 PLC cut-offs of 2.9 mmol/L and 4.1 mmol/L respectively, conversely to Green et al. (2011). The same results were detected considering a T0 PLC cut-off of 2.55 mmol/L and 4.45 mmol/L, respectively, identified as the best compromise values between sensitivity and specificity in our population.

With reference to mean values, our study did not show a significant difference between the initial PLC in dogs with and without macroscopic gastric wall necrosis, as well as between surviving and non-surviving dogs, in contrast with the study of Oron et al. (2018), where the median values of PLC were significantly higher in non-surviving than in surviving dogs. This unexpected result could be conditioned by the limited number of dogs with gastric necrosis, that did not show extremely severe clinical conditions in the post-operative period, simulating a similar status in both survivors and dogs without necrosis.

The same results were recorded at T24 and T48. However, non-surviving dogs registered PLC mean values higher than 2 mmol/L both at T24 and T48. The same consideration was found for dogs with gastric necrosis at T48. Despite these differences were not significant, the limited number of non surviving dogs at T48 must be considered and statistical analyses on wider populations could lead to different considerations.

Non-significant differences were detected between T24 and T48 PLCs for all the dog categories considered: it is likely that longer than 24 h is needed to cause significant PLC variation after T24, or perhaps the normal PLC is already achieved at T24 and later evaluations are not needed, as they will not demonstrate further variations.

Macroscopic gastric wall necrosis was found in 29% of our animals, and 33% of those survived to discharge; 87.5% of dogs without macroscopic gastric necrosis survived. These data suggest that gastric wall necrosis importantly affects the outcome, as indicated in literature (De Papp et al., 1999; Zacher et al., 2010; Green et al., 2011; Santoro Beer et al., 2013). Overall mortality rate registered in this study is 31%, which is in line with the range indicated in more recent studies (Beck et al., 2006; Buber et al., 2007; Mackenzie et al., 2010; Zacher et al., 2010).

Main limit of this study is the restricted number of cases included, in particular for groups related to dogs with gastric necrosis and dead dogs analysed at T48, as the majority of dogs appertaining to these categories died before T48. In our T48 groups related to dogs with gastric necrosis and non-surviving ones, only two patients were included reporting PLC values with conflicting trends. Moreover, due to the retrospective nature of the study, histological diagnosis of gastric wall necrosis was not always available. Furthermore, due to the same limit, data regarding pre-hospitalisation factors, which may impact PLC, such as severity of the symptomps and elapsed time from GDV onset and the presentation of the patients to the clinic, are not available for most the dogs. Prospective and larger studies are suggested to complete and confirm these discussions.

5. Conclusions

Our results did not detect a prognostic reliability of PLC at presentation, nor of a $\geq$50% decrease of median PLC from T0 to T24 in our GDV dogs. Despite these conclusions are probably valid only for this relatively small population, it is authors' believe that it can be a starting point for further and wider studies about PLC, and that probably other prognostic and predictive elements should be investigated during GDV as well; furthermore, an exploratory laparotomy should always be suggested to assess the predicted presence or absence of gastric necrosis, according to Mooney et al. (2014).

Declarations

Author contribution statement

Lisa Grassato, Giuseppe Spinella, Simona Valentini: Conceived and designed the experiments; Performed the experiments; Wrote the paper.

Vincenzo Musella, José Manuel Vilar: Analyzed and interpreted the data.

Massimo Giunti: Performed the experiments; Contributed reagents, materials, analysis tools or data.

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Competing interest statement

The authors declare no conflict of interest.

Additional information

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