Journal of Equine Veterinary Science 36 (2016) 58–62

Contents lists available at ScienceDirect

Journal of Equine Veterinary Science

journal homepage: www.j-evs.com

Short Communication

Lactate Dehydrogenase Activity in Abdominal Fluid From Horses With Colic

Celia Smuts*, Jennifer Mills, René Myles, Tibor Gaál

Department of Pathology, Veterinary and Life Sciences, Murdoch University, Murdoch, Western Australia, Australia

ARTICLE INFO

Article history:
Received 24 July 2015
Received in revised form
13 October 2015
Accepted 15 October 2015
Available online 23 October 2015

Keywords:
Equine colic
Abdominal lactate dehydrogenase activity
LDH
Prognosis

ABSTRACT

The purpose of this study was to determine whether lactate dehydrogenase (LDH) activity in abdominal fluid could be used as a prognostic indicator in horses with colic. Lactate dehydrogenase activity was measured in 27 abdominal effusions from horses with colic presented to Murdoch University Veterinary Teaching Hospital using three different LDH test methods. Lactate in effusions was also measured in 11 of the horses. Lactate dehydrogenase activity was significantly different for each test method used—the ratio of Randox wet chemistry LDH lactate to pyruvate:Randox wet LDH pyruvate to lactate (P-L):IDEXX dry chemistry P-L was approximately 1:2:4. Lactate dehydrogenase activity in the abdominal effusions was significantly higher with all methods in the horses that died or were euthanized because of abdominal sepsis or advanced neoplasia than in those that survived after treatment for colic signs because of mechanical obstructions or nonseptic abdominal inflammation. Lactate dehydrogenase activity showed moderate-to-good correlation ($r = 0.73$ to 0.86) with lactate concentration of the fluid. In conclusion, LDH activity in abdominal fluid may be a useful prognostic test in horses with colic. The test method for LDH measurement must be known and remain constant for meaningful interpretation. Significantly higher levels of LDH activity may be present in horses with colic because of sepsis or advanced neoplasia than in those with colic because of nonseptic inflammation or mechanical obstructions that may respond to treatment.

1. Introduction

Although lactate dehydrogenase (LDH) activity measurement is used extensively in human medicine to differentiate mainly between transudative and exudative pleural effusions [1–4], few studies have been carried out on the body cavity effusions of veterinary patients [5–7].

Lactate dehydrogenase is an enzyme that is present in the cytoplasm of almost all cells, including leukocytes [8] and red blood cells (RBCs), and is an end enzyme in the glycolysis pathway. It acts as a hydrogen transfer enzyme, catalyzing the conversion of LDH-lactate to pyruvate (L-P), which is the final step in the metabolic chain of anaerobic glycolysis. The reaction is reversible and the reaction equilibrium favors the reduction of pyruvate to lactate (P-L) [9,10]. Lactate dehydrogenase can be measured using both the forward and reverse reactions, using both wet chemistry (conventional) and dry (solid phase) chemistry methods.

On average, human tissues have about 500 times the total LDH levels found in the serum, with very high levels in the liver (9,000 IU/g), heart (25,000 IU/g), skeletal muscle (9,000 IU/g), and lung (9,500 IU/g) [11]. Enzymes such as LDH appear to serve no function in body cavity effusions but can serve as indicators of disturbed cellular integrity induced by pathologic conditions [11]. Even a small amount of tissue damage can result in significant elevation of LDH activity and its extracellular appearance can, therefore, be used to detect cell injury or death [10]. Increased activities

* Corresponding author at: Celia Smuts, Department of Pathology, Veterinary and Life Sciences, Murdoch University, South Street, Murdoch, Western Australia 6150, Australia.
E-mail address: celiasmuts@gmail.com (C. Smuts).

0737-0806/$ – see front matter © 2015 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.jevs.2015.10.004

Table 1
Twenty-seven effusions from horses presenting with colic.

	Final Diagnosis	TCC ($\times 10^9$/L)	TP (g/L)	PCV	Lactate (mmol/L)	Wet LDH L-P (IU/L)	Wet LDH P-L (IU/L)	Dry LDH P-L (IU/L)	Survival >1 mo
1	Colic, nonsurgical	1.2	28	<0.01	1.15	124	220	660	Survived without surgery
2	Colic, nonsurgical	2.1	<25	<0.03	ND	147	252	532	Survived without surgery
3	Colic, nonsurgical	2.7	<25	<0.01	ND	71	129	528	Survived without surgery
4	Diarrhea, hepatic fibrosis	0.9	32	<0.01	ND	132	267	571	Survived without surgery
5	Mild abdominal inflammation	1.4	<25	<0.01	ND	35	66	ND	Survived without surgery
6	Peritonitis (no bacterial growth)	4.2	31	<0.01	ND	188	377	619	Survived without surgery
7	Peritonitis (no bacterial growth)	4.9	31	0.01	ND	197	392	601	Survived without surgery
8	Peritonitis (no bacterial growth)	3.2	30	0.01	ND	231	452	701	Survived without surgery
9	Septic peritonitis	8.5	35	0.02	3.6	281	497	771	Survived without surgery
10	Uterine hematoma/inflammation	39.1	39	0.01	ND	279	542	877	Survived without surgery
11	Colic, nonsurgical, abdominal inflammation	60.2	41	<0.01	ND	1,080	2,815	6,490	Survived without surgery
12	Cecal impaction, nonstrangulating 180° cecal torsion	0.61	<25	<0.01	3	118	227	179	Survived with surgery
13	Colic (torsion, viable)	0.4	<25	<0.01	3.3	178	338	664	Survived with surgery
14	Colic strangulated colon, viable	2.6	<25	<0.01	5.1	264	476	899	Survived with surgery
15	Colon volvulus (black, viable)	0.2	<25	<0.01	2.1	153	281	ND	Survived with surgery
16	Small colon feed impaction and a subacute grade 1 rectal tear	1.1	<25	<0.01	3.3	315	557	962	Survived with surgery
17	Splenic entrapment colic, no necrosis	1.1	26	<0.01	2.7	349	674	1,092	Survived with surgery
18	Thromboembolic colic, ischemia of the large colon, resection	0.9	<25	<0.01	1.6	117	215	653	Survived with surgery
19	Uroperitoneum	0.1	<25	<0.01	ND	14	35	281	Survived with surgery
20	Duodenal rupture and sepsis	0.3	ND	0.03	13.6	416	773	2,000	Died
21	Gastric squamous cell carcinoma	41.6	65	0.01	ND	2,800	5,380	15,340	Died
22	Gastric squamous cell carcinoma	26.9	41	0.01	ND	376	658	995	Died
23	GIT necrosis and sepsis	0.65	54	0.04	ND	573	1,092	1,553	Died
24	GIT rupture and sepsis	31	35	0.02	ND	258	479	815	Died
25	Hepatic squamous cell carcinoma	32.3	33	0.01	ND	287	539	910	Died
26	Septic peritonitis	65.8	46	0.01	ND	1,920	3,380	11,060	Died
27	Septic peritonitis (*Actinobacillus* sp.)	500	42	<0.01	ND	4,920	8,360	12,300	Died

Abbreviations: GIT, gastrointestinal tract; LDH, lactate dehydrogenase; L-P, lactate to pyruvate; ND, not done; PCV, packed cell volume; P-L, pyruvate to lactate; TCC, total nucleated cell count, TP, total protein measured by refractometry.

of LDH in peritoneal fluid may indicate inflammation and tissue damage including ischemia [12]. Lactate can also be measured in abdominal fluid on laboratory-based and point-of-care monitors and is frequently used for evaluating abdominal fluid in horses with colic [13].

This study explores the use of LDH activity of abdominal effusions as an aid in determining prognosis in horses with colic.

2. Materials and Methods

Abdominal fluid was collected from 27 horses presented to the Murdoch University Veterinary Hospital and submitted to the MUVH clinical pathology laboratory for evaluation. Samples submitted to the laboratory were often from referral cases and in cases wherein the clinicians required more information than their in-house analysis provided, for example, prolonged colic, complicated medical or surgical cases, or insurance cases. Samples were excluded if they were analyzed later than 24 hours after collection. Use of samples complied with the Murdoch University animal ethics protocols for excess samples obtained for diagnostic purposes.

Lactate was also measured in the abdominal samples from 11 of the horses on a radiometer ABL 700 blood gas analyzer (Radiometer Medical ApS, Denmark) using samples collected into EDTA anticoagulant and analyzed within 15 to 30 minutes after collection.

Routine analysis was performed on all submitted abdominal effusions. This included obtaining a total nucleated cell count and red cell count by flow cytometry (Advia 120 hematology analyzer; Siemens Health Care Diagnostics) on the effusion sample. Packed red cell volume (PCV) was measured in hematocrit capillary tubes after centrifugation. Total protein was measured on the supernatant of the effusion using a temperature-compensated refractometer (Reichert Vet 360, NY).

A direct smear and a cytocentrifuged preparation (50 and 200 μL of abdominal fluid, depending on visual density of the sample) were air dried and stained with Wright–Giemsa (HEMA-TEK–modified Wright-Giemsa stain pack, Bayer, Germany) and evaluated microscopically by a clinical pathologist. Culture and sensitivity were performed by an independent laboratory (Vetpath, Perth, Western Australia) on samples with a high neutrophil percentage (>70%), presence of microorganisms observed visually on microscopy, and/or high clinical suspicion of infection.

An aliquot of each abdominal effusion sample was spun at 3,000 rpm (1,500 G) in a Jouan CR3i multifunction centrifuge (Thermo Fisher Scientific) at 4°C for 10 minutes, the supernatant placed in a 1-mL plastic tube, and the sediment discarded. The supernatant was kept at 4°C and analyzed for LDH activity within 24 hours of sample