The Association Between Selected Metabolic Parameters and Left Displaced Abomasum in Dairy Cows

T Geishauser, K Leslie, T Duffield and V Edge

Department of Population Medicine, Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1, Canada

Abstract

The objective of this study was to examine the association between selected metabolic parameters and subsequent left displaced abomasum (LDA) diagnosis in dairy cows. Forty-four LDA cows were sampled in the third week ante partum (a.p.), which was at a median of 34 days prior to LDA diagnosis; 36 LDA cows were sampled in the first week post partum (p.p.), which was at a median of 14 days prior; and 28 LDA cows were sampled in the second week p.p., which was at a medain of 9 days prior to LDA diagnosis were used. Each case was matched to 3 controls by herd and calving date. Data were available from a large field study. Aspartateaminotransferase (AST) activity, the concentrations of β-hydroxybutyrate (BHB), glucose, calcium and urea in blood, and the body condition score (BCS) were studied. Logistic regression was used to analyse the association between these parameters and subsequent LDA, adjusting for the effects of parity and pretreatment. A separate model was used for each sampling week and each parameter. In the third week a.p., none of the parameters were significantly associated with LDA. AST and BHB sampled in the first week p.p. and in the second week p.p. were significantly associated with LDA diagnosis. The higher the AST and BHB the higher the odds of being diagnosed subsequently with LDA. The lower glucose and Ca in the second week p.p. the higher the odds of subsequent LDA diagnosis. Urea and BCS were not significantly associated with LDA in any of the weeks examined. We conclude that AST and BHB in the first and second week p.p. might be used as tests for subsequent LDA.

Introduction

The lactational incidence risk of left displaced abomasum (LDA) in Ontario dairy herds is currently about 2% (1). This disease causes losses due to treatment costs, discarded milk, decreased milk yield, culling, and death. Although displaced abomasum (DA) has often been studied, methods of prevention and tools to monitor for DA are not well described. The objective of the present study was to examine the association between selected metabolic parameters sampled in the third week before calving, and in the first and in the second weeks after calving, and subsequent LDA diagnosis. Metabolic parameters were aspartate-aminotransferase activity, the concentrations of β -hydroxybutyrate, glucose, calcium, and urea in blood serum, and the body condition score.

Material and Methods

Data were taken from a field study that was conducted in 1995 with 1052 Holstein cows in 26 dairy herds in the area of Guelph, Ontario, Canada. One purpose of this study was to evaluate the efficacy of a monensin controlled release capsule (CRC) for the prevention of subclinical ketosis (2). Assignment of CRC and placebo were randomized within herd and were administered intraruminally approximately three weeks prior to the expected calving date. Every herd was visited weekly, on the same day of the week and at approximately the same time of the day. Blood was collected at the time of CRC administration and during the first and second weeks p.p. Cows were body condition scored (BCS) on a scale of 1 to 5 (3) at the time of each sample collection. Blood was collected from a coccygeal vein into 10 ml vacuum tubes and analyzed within five hours after collection. Blood was centrifuged at 1200 G for 10 minutes. Aspartate-aminotransferase (AST) activity (U/I) and \(\beta\)-hydroxybutyrate (BHB) (\(\mu\)mol/I), glucose, calcium (Ca) and urea concentrations (mmol/l) were determined in blood serum using an autoanalyzer (2). Data from samples showing hemolysis were excluded. All LDA were diagnosed by veterinarians and confirmed during surgery.

Data were analysed as a matched case control study. Matching criteria were herd and calving date, so as to limit effects of herd and season including feeding. All cases of LDA were selected and dates of calving and LDA diagnosis were identified. Data were included if samples were taken before LDA diagnosis. Data that were derived from samples that were taken on or after

Adapted from the Proceedings of the XX World Association for Buiatrics Congress, Sydney, Australia, July, 1998.

the day of LDA diagnosis were excluded. Each LDA case was matched with three controls not diagnosed with LDA. Cows having calved closest to the case were selected as controls. Each control was assigned only once to a case.

Descriptive statistics are given as means in normal data and as medians in non-normal data. Non-normal variables, such as AST and BHB, were transformed using a natural log (ln). Parity was a binary variable indicating lactation number (0 = first lactation, 1 =lactation number ≥ 2). Treatment was a binary variable indicating CRC administration (0 = no, 1 = yes). Statistical analysis was performed separately for samples taken in the third week ante partum (a.p.), or in the first or second week post partum (p.p.). The effect of each metabolic parameter on LDA was evaluated using logistic regression (4). The effect of AST, BHB, glucose, Ca, urea, and BCS on LDA was evaluated in separate models. Parity and treatment, known to be related to LDA, as well as to metabolic parameters (2, 5; 6) were controlled for, as possible confounders, by forcing them into each model. Because blood was collected prior to treatment, treatment was not controlled for in the third week a.p. The fit of the models was assessed by means of the Hosmer-Lemeshow goodness of fit test (4). Significance level was set at 5%. Calculations were performed using SAS (7).

Results

Forty-four LDA cases and 132 controls from 18 herds were included in this study. Twenty-five percent of the cases and 30% of the controls were in first lactation. Forty-eight percent of the cases and 58% of the controls were CRC treated. Controls had calved at a mean of 1.5 days after cases. Samples were taken at a mean of 19 days a.p., 3, and 10 days p.p., showing no statistical differences between cases and controls. Eighty-two percent of the cases were diagnosed in the first month, 11% in the second month and 7% in the third month of lactation.

Third week ante partum - Forty-four LDA cases and 132 controls were available to study the association between metabolic parameters sampled in the third week a.p. and subsequent LDA diagnosis. LDA was diagnosed at a median of 16 days p.p.. Cases were sampled at a median of 34 days prior to LDA diagnosis. None of the metabolic parameters, adjusted for parity, had significant effects on the odds of LDA diagnosis (Table 1).

First week p.p. - Thirty-six LDA cases and 108 controls were available to study the association between metabolic parameters sampled in the first week p.p. and subsequent LDA diagnosis. LDA was diagnosed at a median of 16 days p.p. Cases were sampled at a median of 14 days prior to LDA diagnosis. AST, and BHB adjusted for parity and treatment, had significant effects on LDA diagnosis, whereas glucose, Ca, urea and BCS had not. The higher the AST and BHB, the higher were the odds of LDA diagnosis (Table 1).

Second week p.p. - Twenty-eight LDA cases and 84 controls were available to study the association between metabolic parameters sampled in the second week p.p. and subsequent LDA diagnosis. LDA was diagnosed at a median of 19 days p.p. Cases were sampled at a median of 9 days prior to LDA diagnosis. AST, BHB, glucose and Ca, adjusted for parity and treatment, had significant effects on LDA diagnosis, whereas urea and BCS had not. The higher the AST and BHB and the lower the glucose and Ca, the higher the odds of LDA diagnosis. In all models the Hosmer-Lemeshow test indicated that the number of predicted cases was not significantly different from the number of observed cases (Table 1).

Discussion

We conclude that AST and BHB in the first and second week p.p. and glucose and calcium in the second week p.p., might be used as tests for subsequent LDA diagnosis in dairy cows. Urea and BCS were not significantly associated with subsequent LDA diagnosis in any of the sampling weeks. Thus, urea and BCS might not be helpful as tests for prediction of LDA.

Blood BHB concentrations found in the first and second week p.p. in cows subsequently diagnosed with LDA were within a range (1000 to 3000 μ mol/l), that was attributed to subclinical ketosis (8). Increasing blood AST activity was associated with increasing degrees of hepatic lipidosis in other studies (9). Increased blood AST activity and hepatic lipidosis are often present during ketosis (10). Our findings suggest that subclinical ketosis is a significant risk factor of LDA. This is in accordance with clinical studies reporting ketosis prior to LDA diagnosis (11, 12, 13), and with epidemiological studies reporting clinical ketosis as a risk factor of LDA (14, 15, 16).

Low blood calcium concentration may reduce abomasal motility (17) and hypocalcemia (< 1.97 mmol total Ca/l blood) at calving was reported a risk factor of LDA(18). In our study, however, calcium concentrations in the third week a.p. and in first week p.p did not significantly affect the odds of subsequent LDA diagnosis, whereas decreasing calcium concentrations in the second week p.p. significantly increased the odds of subsequent LDA diagnosis. Given that blood Ca levels need to fall below 1.2 mmol total Ca/l blood to reduce abomasal motility (17) the effect of Ca on abomasal motility was low in our study. Lower 95% confidence limits were 2.27 mmol total calcium/l blood in the third

Table 1. Logistic regression of displaced abomasum on blood lnAST, lnBHB, glucose, calcium, urea, and BCS sampled in the third week a.p., in the first and in the second week p.p., adjusting for parity and treatment. Coefficients (β), standard errors (SE), P values (P) are given for variables; and Hosmer-Lemeshow Goodness of Fit P values (GOFP) are given for models.

		Third week a.p.		First w			ek p.p.		Second week p.p.			
	ß	SE	Р	GOFP	ß	SE	Р	GOFP	ß	SE	Р	GOFF
InAST	-0.33	0.70	0.66	0.26	1.59	0.53	0.00	0.62	2.86	0.72	0.00	0.16
Parity	0.24	0.40	0.55		0.91	0.46	0.68		0.21	0.58	0.71	
Treatment					-0.80	0.42	0.06		-0.65	0.52	0.21	
InBHB	0.19	0.50	0.71	0.52	0.74	0.34	0.05	0.14	1.79	0.46	0.00	0.11
Parity	0.26	0.40	0.55	-0.01	0.48	0.98		-0.26	0.59	0.65		
Treatment					-0.47	0.40	0.24		-0.57	0.51	0.26	
Glucose	-0.14	0.31	0.63	0.12	-0.38	0.27	0.17	0.34	-1.15	0.41	0.01	0.40
Parity	0.24	0.39	0.54		0.19	0.46	0.58		-0.12	0.57	0.83	
Treatment					-0.55	0.40	0.17		-0.67	0.48	0.16	
Calcium	-0.96	1.35	0.48	0.57	-0.29	0.78	0.71	0.37	-3.89	1.79	0.03	0.40
Parity0.27	0.40	0.50		0.28	0.45	0.53		0.49	0.52	0.38		
Treatment					-0.58	0.40	0.14		-0.81	0.47	0.08	
Urea	0.03	0.12	0.81	0.80	0.00	0.12	0.98	0.30	-0.14	0.16	0.37	0.67
Parity	0.27	0.40	0.49		0.30	0.44	0.50		0.65	0.52	0.21	
Treatment					-0.59	0.33	0.13		-0.84	0.46	0.07	
BCS	0.38	0.43	0.38	0.44	-0.23	0.46	0.61	0.26	0.56	0.58	0.34	0.91
Parity	0.20	0.41	0.63		0.32	0.45	0.47		0.55	0.51	0.28	
Treatment					-0.50	0.40	0.15	N	-0.93	0.46	0.04	

week a.p., 2.0 in the first, and 2.15 in the second week p.p. In cows diagnosed with displaced abomasum blood calcium decreased with increasing base excess (19). Metabolic alkalosis was diagnosed prior to (13) and together with displaced abomasum (20). Whether hypocalcemia follows metabolic alkalosis in cows developing displaced abomasum needs further research. Lowering blood pH, however, is thought to prevent hypocalcemia (21).

Low body condition scores in dry cows were reported from a herd experiencing high LDA incidence after calving (22). However, others reported high body condition scores as a risk factor of LDA (23, 24). Both low and high body condition at calving might predispose dairy cows to ketosis (10), and thus to LDA. In our study, body condition did not affect the odds of being diagnosed with LDA. Blood urea concentration was associated with protein intake (25), protein/energy ratio (26), and ruminal and abomasal ammonia concentrations in dairy cows (27) by others. High ruminal ammonia concentrations decreased ruminal motility (28). No significant association was found between blood urea concentrations and subsequent LDA diagnosis in our study.

References

1. Kelton DF (1995) Monitoring, and investigating the relationships among, health, management, productivity and profitability on Ontario dairy farms *Guelph*, *Univ.*, *PhD-Thesis*.

2. Duffield T (1997) Effects of a monensin controled release capsule on energy metabolism, health and production in lactating dairy cattle *Guelph, University, DVSc Diss.*

3. Edmonson AJ, Lean IJ, Weaver LD, Farver T and Webster G (1989) A body condition scoring chart for Holstein dairy cows *J Dairy Sci* 72:68-78.

4. Hosmer DW and Lemeshow SW (1989) Applied logistic regression Wiley, New York / USA.

5. Constable PD, Miller GY, Hoffsis GF, Hull BL and Rings DM (1992) Risk factors for abomasal volvulus and left abomasal displacement in cattle *Am J Vet Res 53:1184-1192*.

6. Kauppinen K (1983) Prevalence of bovine ketosis in relation to number and stage of lactation *Acta Vet Scand* 24:349-361.

7. SAS Institute Inc. SAS/STAT users guide. SAS Institute, Cary/USA, Version 6.11, 1995.

8. Työpponen J and Kauppinen K (1980) The stability and automatic determination of ketone bodies in blood samples taken in field conditions *Acta Vet Scand* 21:55-61.

9. Rehage J Mertens M, Stockhofe-Zurwieden N, Kaske M and Scholz H (1996) Post surgical convalescence of dairy cows with left abomasal displacement in relation to fatty liver *Schweiz Arch Tierheilk* 138:361-368.

10. Baird GD (1982) Primary ketosis in the high-producing dairy cow:

Clinical and subclinical disorders, treatment, prevention and outlook J Dairy Sci 65:1-10.

11. Varden SA (1979) Displacement of the abomasum in the cow, incidence, etiological factors and results of treatment *Nord Med Vet* 31:106-113.

12. Grauerholz H, Godehart F and Grottker S (1982) Beitrag zur linksseitigen Labmagenverlagerung beim Rind *Prakt Tierarzt* 63:38-46.

13 Vörös K and Karsai F (1987) Blut- und Pansensaftveränderungen vor dem Auftreten der linksseitigen Labmagenverlagerung bei Milchkühen *Tierärztl Umsch* 42:617-624.

14. Curtis CR, Erb HN, Sniffen CJ, Smith RD and Kronfeld DS (1985) Path analysis of dry period nutrition, postpartum metabolic and reproductive disorders, and mastitis in Holstein cows J Dairy Sci 68:2347-2360.

15. Correa MT, Erb HN and Scarlett M (1993) Path analysis for seven postpartum disorders of holstein cows J Dairy Sci 76:1305-1312.

16. Gröhn YT, Eicker SW and Hertl JA (1995) The association between previous 305-day milk yield and disease in New York state dairy cows *J Dairy Sci* 78:1693-1702.

17. Madison JB and Troutt HF (1988) Effects of hypocalcaemia on abomasal motility Res Vet Sci 44:264-266.

18. Massey CD, Wand C, Donovan GA and Beede DK (1993) Hypocalcemia at parturition as a risk factor for left displacement of the abomasum in dairy cows J Am Vet Med Assoc 203:852-853.

19. Geishauser T and Oekentorp N (1997) The association between blood calcium and selected parameters in dairy cows diagnosed with left displaced abomasum J Vet Med A 44:493-500.

20. Kuiper R (1980) Reflux van Lebmaginhoud bij het rund *Utrecht, University, PhD-Thesis.*

21. Block E. (1994) Manipulation of dietary cation-anion difference on nutritionally related production diseases, productivity, and metabolic responses of dairy cows *J Dairy Sci* 77:1437-1450.

22. Jacobsen KL (1995) Displaced abomasa and thin cows in a component-fed dairy herd Food Animal Medicine and Management 17, 8:21-27.

23. Baudet HM (1992) Conduit alimentaire des vaches taries et incidence des maladies métaboliques en début de lactation Rec Med Vet 168:437-441.

24. Cameron REB, Dyk PB, Herdt TH, Kaneene JB, Miller R, Bucholtz HF et al. (1998) Dry cow diet, management, and enery balance as risk factors for displaced abomasum in high producing dairy herds *J Dairy Sci* 81:132-139

25. Manston R, Russel AM, Dew SM and Payne JM (1975) The influence of dietary protein upon blood composition in dairy cows Vet Rec 96:497-502

26. Lebeda M and Prikrylova J (1978) Der Einfluß des Energie- und Proteinangebotes auf die Harnstoffkonzentration im Blutserum und im Harn von Milchkühen *Monatsh Vet Med* 33:944-949.

27. Weiser M (1997) Untersuchungen zum Harnstoffgehalt im Blut und zum Gehalt von Ammoniak im Pansensaft, im Labmagensaft und im Blut von gesunden und an Labmagenverlagerung kranken Kühen Gießen, Universität, FB 18, Dissertionsschrift.

28. Bueno L., Doulou V and Candau M (1977) Ammoniogenèse et motricité, du rumen chez le mouton Ann Biol Anim Bioch Biophys 17:509-514.

Abstract

Biliary elimination of endogenous nortestosterone by pregnant cows J.D.G. McEvoy, C.E. McVeigh, W.J. McCaughey, S.A. Hewitt *Veterinary Record* (1998) 143, 296-299

A temporal study of the biliary elimination of endogenous 19-nortestosterone during two successive pregnancies was made in three cows with cannulated gall bladders. Bile samples were analysed for 17 β -19nortestosterone (β -NT) and the 17 α -epimer (α -NT) by using high resolution gas chromatography and mass spectroscopy. No β -NT was detected in any of the samples analysed. However, α -NT was detected from around 120 days of gestation in each of the cows. Peak concentrations were observed in the last week before calving and ranged from 9.5 to 36.7 ng/ml. After parturition, the concentrations of α -NT declined rapidly and were undetectable by seven days after calving, and it was not detected again until after 120 days of gestation. The biliary concentrations of α -NT detected subsequently were similar to those observed in cattle several weeks after an exogenous injection of the synthetic ester β -NT phenylpropionate.