Case Report Rapport de cas

Ruminal acidosis in a 21-month-old Holstein heifer

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Abstract — Rumen and blood biochemical profiles were monitored in 8 Holstein heifers exposed to a carbohydrate feeding challenge. One of the heifers had clinical signs consistent with acute ruminal acidosis on the day of, and subsequent to, the challenge. Within 24 h of challenge, 6 of 7 rumen volatile fatty acids measured were not detectable in this heifer and her rumen total lactate concentration was > 70 mM.

Résumé — Acidose spumeuse chez une génisse Holstein âgée de 21 mois. Les profils du rumen et de l'hématologie biochimiques ont été surveillés chez 8 génisses Holstein exposées à une épreuve d'alimentation en glucides. L'une des génisses avaient des signes cliniques conformes à une acidose spumeuse aiguë le jour et le lendemain de l'épreuve. Dans un délai de 24 h après l'épreuve, 6 des 7 acides gras volatils du rumen mesurés n'étaient pas détectables chez cette génisse et la concentration de lactate totale de son rumen était de > 70 mM.

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The objective of this report was to describe findings, especially ruminal conditions, that were present in a heifer with clinical signs consistent with ruminal acidosis. Clinical definitions of ruminal acidosis are inconsistent and largely based on arbitrary rumen pH cut points (1,2). Lean et al (3) note the need for definitions of metabolic disease to be accurate, standardized, repeatable, and based on clinical outcomes including measurable changes in metabolism, morbidity, mortality, or production.

Sporadic disorders such as acute ruminal acidosis are only rarely documented in detail that provides a combination of clinical signs, feed intake, nutritional composition of the diet, and rumen and blood measures; therefore, there is merit in presenting observations on a clinical case, particularly when detailed comparative data on other cattle were available.

We report feed intake, clinical signs, and rumen and blood biochemical profiles of a heifer exposed to a diet high in readily fermentable carbohydrates for 20 d preceding challenge with a diet containing 19.1% sugar and 54.1% starch on a dry matter (DM) basis. Her rumen and blood biochemical profiles are compared to those of 7 cohort heifers.

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Case description

A 21-month-old pregnant Holstein heifer [Identification No. 1250; body weight (BW): 488 kg] showed clinical signs of ruminal acidosis within 10 h of consuming 4.8 kg DM of milled wheat and 960 g DM of fructose (1.0 and 0.2% of her BW, respectively). Heifer 1250 was 1 of 8 control heifers (mean BW: 382 ± 17 kg, excluding heifer 1250) enrolled in a 29 d readily fermentable carbohydrate challenge study evaluating the effectiveness of feed additives to reduce acidosis risk (4). The study was conducted at Cobbitty, New South Wales, Australia. All experimental procedures were approved by the SBS*cibus* Animal Ethics Committee (SBScibus 0512-0513).

The study consisted of 5 experimental periods: 1) preadaptation (days −2 to 0), 2) adaptation I (days 1 to 10), 3) adaptation II (days 11 to 20), 4) challenge (day 21), and 5) post-challenge (days 22 to 26). Rations offered at each of these periods are detailed in Figure 1 and Table 1. The 20-day adaptation period was considered adequate to study rumen perturbation. Rumen and blood samples were not collected from the cohort heifers during the post-challenge period. Heifers were randomly allocated to 1 of 4 blocks (A to D; 2 heifers/block), enrolment of each block of heifers in the study was staggered over 4 consecutive days. Heifer 1250 was allocated to block A.

All heifers were housed with the main study herd in a paddock containing dormant kikuyu *(Pennisetum clandestinum)* with no available pasture and *ad libitum* water access. An equal amount of feed was offered twice daily at 7:00 and 14:00 h in individual feed bins and separate feeding pens that prevented access between pens. The amount of ration that the heifers consumed before ceasing consumption and the time they took

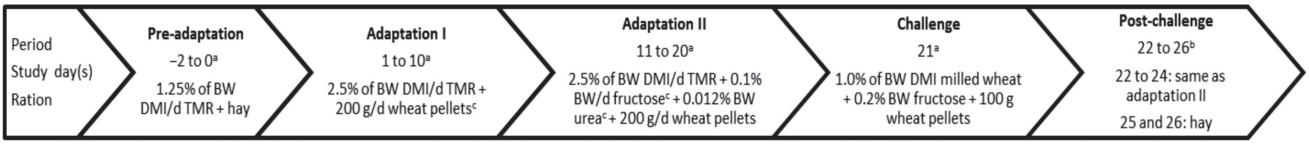

Figure 1. Experimental periods and their corresponding study days and rations offered for the study in which heifer 1250 and her cohort of heifers were enrolled.
BW – body weight; DMI – dry matter intake; TMR – total mixed ration (62:38 forage:concentrate, consisting of 31.5% wheaten hay, 30.5% alfalfa hay, and 38% milled wheat).
[a] Rumen and blood samples were collected on days 0, 7, 14, and 21 during their respective experimental periods from both heifer 1250 and her cohort of heifers.
[b] Rumen and blood samples were collected from heifer 1250 on days 22 and 23.
[c] Introductory doses were offered for the initial days before the full rate was offered.

to consume this amount were recorded. Consumption time did not exceed 2 h. Heifers were subsequently returned to the paddock with no access to feed until the next feeding session.

Heifer 1250's dry matter intake (DMI) over the 20-day adaptation period, in which she was offered 2.5% of her BW DM total mixed ration (TMR)/d (days 1 to 10) and additional 0.1% of her BW fructose (d 11 to 20), averaged 8.6 kg DM/d (84 ± 3.3% of the offered ration/d), compared to 8.3 ± 0.3 kg DM/d (91 ± 1.5% of the offered ration/d) for the cohort heifers. Qualitatively, she had a similar DMI pattern to the cohort heifers, but had more pronounced drops in DMI. She consumed only 4.9 kg DM/d (50% of her allocated ration) on day 4 and a physical examination showed that her rectal temperature, rumen contractions, and heart rate (80 beats/min) were within normal ranges. Her DMI also decreased during the 4 d before challenge (days 17 to 20), to approximately 9.6 kg DM/d (79% of her offered ration/d), from approximately 11.5 kg DM/d (95% of her offered ration) for days 13 to 16. On days 17 to 20 the cohort heifers consumed on average 9.3 ± 0.3 kg DM/d (95 ± 1.5% of their offered ration/d).

Rumen fluid collected using a customized stomach pump and tube, and blood samples were taken weekly before challenge on day 0 (pre-adaptation), day 7 (adaptation I), and day 14 (adaptation II). The stomach tube was inserted to a minimum length of 2 m and rumen fluid was scored for saliva contamination (5). Rumen pH (Merck, Kilsyth, Victoria, Australia; Figure 2I) was measured within 3 min of collection of rumen fluid and all other measurements were done at a later date on samples immediately placed on ice and later stored at −20°C. All rumen and blood measures from heifer 1250 were compared with those of the 7 other heifers and are referred to as higher or lower if they are outside the mean ± standard error of the mean (SEM) of these cohort heifers.

Heifer 1250 had a higher rumen pH (Figure 2I), 20 to 30 mM lower total VFA's, which comprised acetate, propionate, butyrate, valerate, isobutyrate, isovalerate, and caproate measured by gas chromatography (Figure 2A), lower major individual VFA's (Figures 2B, C, E, and F), and a similar acetate to propionate ratio (Figure 2D) than the cohort heifers before challenge day. The heifer's ammonia concentration measured by a direct enzymatic method was lower in the adaptation II sampling (Figure 2G), while her total rumen lactate concentration (D-lactate; Boehringer Mannheim kit: cat no. 11 112 821 035; R-Biopharm-Laboratory Diagnostics, Taren Point, New South Wales, Australia; L-lactate; cat no. OSR 6193; Beckman Coulter Australia, Gladesville, New South Wales, Australia) was higher in the adaptation I sampling (Figure 2H).

Plasma reactive oxygen metabolites (dROMs; d-ROMS Test; Diacron International, Grosseto, Italy) and biological antioxidant potential (BAP; Bap Test; Diacron International) (6) were increased in heifer 1250 compared to the cohort heifers at the adaptation I and II samplings, respectively (202 compared to 138 ± 14 Carratelli units, and 4351 compared to 3884 ± 280 μmol/L). Heifer 1250's plasma oxidative stress index [OSI; (dROMs/BAP) × 100 (6)] (Figure 2J) was increased in the pre-adaptation and adaptation I samplings. Her plasma ceruloplasmin (7) dropped 133.7 mg/L between the pre-adaptation and adaptation I samplings (Figure 2K).

Serum non-esterified fatty acid (NEFA) concentrations (Randox Laboratories, Crumlin, Antrim, UK) for heifer 1250 were similar to those of the cohort heifers before challenge. Her serum β-hydroxybutyrate concentration (Randox Laboratories) was higher in adaptation II than that of the cohort heifers (Figure 2L). Heifer 1250's serum glucose concentration (Infinity™ Glucose Hexinase Liquid Stable Reagent; Thermo Scientific, Scoresby, Victoria, Australia) was lower in the adaptation II sampling than that of the cohort heifers (4.3 compared to 4.8 ± 0.2 mM, respectively). Serum urea concentrations for heifer 1250 (Infinity™ Urea Liquid Stable Reagent; Thermo Scientific) were lower than the cohort heifers' in the pre-adaptation (2.5 compared to 3.8 ± 0.2 mM, respectively) and higher in the adaptation II samplings (5.2 compared to 3.1 ± 0.1 mM, respectively).

Heifers were scored for locomotion using a 5-point scoring system (8) after rumen and blood collection and heifer 1250 was scored as < 2 (sound) during all scoring sessions leading up to challenge day, similar to her cohorts.

On the challenge day (day 21) each heifer was offered 200 g of alfalfa hay and immediately after its consumption each heifer was offered 1.0% and 0.2% of their BW DM in milled wheat and fructose, respectively, and 200 g of wheat pellets. The challenge ration had 19.1% DM sugar and 54.1% DM starch content (CPM Dairy Analyzer V3.10; Cornell-Penn-Miner, Cornell University, Ithaca, New York, USA; Table 1). Feeding was staggered within each block with the first heifer fed at 7:00 h. Heifer 1250 consumed 100% of her offered 4.8 kg DM of milled wheat, 960 g DM of fructose, and 200 g of wheat pellets in 36 min (total intake 6.0 kg DM). The mean DMI, percentage of offered ration consumed, and consumption time for the cohort heifers were 4.2 ± 0.4 kg DM, 90 ± 9.6%

Table 1. Estimated chemical composition of the ration offered during the following periods: 1). adaptation I [comprising 2.5% of body weight (BW) DMI/d total mixed ration (TMR) + 200 g/d wheat pellets], 2). adaptation II (comprising 2.5% of BW DMI/d TMR + 0.1% of BW/d fructose + 200 g/d wheat pellets), and 3). challenge (comprising 1.0% of BW DMI wheat + 0.2% of BW fructose + 100 g wheat pellets)[a]. Estimations were based on a 400 kg BW heifer with a body condition score of 3.25 and a growth rate of 0.73 kg/d. The TMR (62:38 forage:concentrate) consisted of 31.5% wheaten hay, 30.5% alfalfa hay, and 38% disc milled wheat

Item (% DM)	Adaptation I (days 1 to 10)	Adaptation II (days 11 to 20)	Challenge (day 21)
DM	89.6	89.9	90.2
CP	13.4	14.1	9.8
RUP (% of CP)	27.6	21.3	11.8
RDP (% of CP)	72.4	78.7	88.2
RDP	9.70	11.1	8.60
Soluble protein (% of CP)	39.8	45.0	38.5
ADF	22.4	21.6	4.49
NDF	35.0	33.6	10.7
Forage NDF (% of NDF)	85.8	85.8	0.0
Forage NDF (% of DM)	30.0	28.8	0.0
Physically effective NDF	30.6	29.4	4.2
Lignin	3.6	3.5	0.9
NFC[b]	46.1	47.7	76.3
Silage acids	0.0	0.0	0.0
Sugar	9.4	12.6	19.1
Starch	27.5	26.4	54.1
Soluble fiber	9.1	8.7	3.2
Total ether extract	1.95	1.88	1.74
Total LCFA	1.39	1.34	1.57
Ash	5.98	4.94	1.82
DCAD (mEq/100 g)	24.7	23.7	−0.25
Minerals (mg/kg)			
Chloride	4400	4300	1000
Calcium	4800	4500	900
Copper	21	19	20
Iron	128	122	42
Phosphorus	3000	2900	2800
Potassium	1700	16 300	4100
Magnesium	1400	1300	1000
Manganese	75	70	58
Sodium	1300	1200	100
Sulfur	1900	1800	1300
Zinc	64	58	62

[a] Estimations were performed using CPM Dairy Analyzer V3.10 (Cornell-Penn-Miner, Cornell University, Ithaca, NY, USA).
[b] NFC = 100 — [(NDF — NDICP) + CP + crude fat + ash].
DM — dry matter, DMI — dry matter intake, TMR — total mixed ration, CP — crude protein, RUP — rumen undegradable protein, RDP — rumen degradable protein, ADF — acid detergent fiber, NDF — neutral detergent fiber, NFC — non-fiber carbohydrates, LCFA — long-chain fatty acids, DCAD — dietary cation-anion difference.

and 62 ± 9.8 min, respectively. At approximately 3.6 h after consumption of the ration a rumen fluid sample and a blood sample were collected from all heifers.

Heifer 1250's rumen pH was 5.1 on the challenge day, which was a decline of 1.7 pH units from her pre-adaptation sample pH. Mean rumen pH of the cohort heifers on the challenge day was 5.7 (Figure 2I).

On the challenge day heifer 1250's total VFA concentration was lower (82.9 mM) compared with her cohorts (114.7 ± 13.4 mM; Figure 2A). Her acetate concentration was similar to the cohort heifers' (Figure 2B); however, her propionate concentration was lower at 7.7 mM compared with 24.1 ± 4.1 mM for her cohorts (Figure 2C), and her acetate to propionate ratio was 8.2 compared with 3.1 ± 0.4. Heifer 1250's butyrate concentration was lower (10.7 mM) than the cohort heifers' (19.3 ± 3.6 mM; Figure 2E). Her valerate concentration was lower at 0.5 mM compared with 3.0 ± 0.9 mM for the cohort heifers (Figure 2F), and ammonia concentration was over 6-fold higher than that of the cohort heifers (Figure 2G).

Heifer 1250's rumen histamine concentration (human histamine ELISA kit; IBL International, Hamburg, Germany) was more than double that of the cohort heifers' on challenge day, 291.6 and 124.2 ± 40.5 mM, respectively. Her dROMs were higher over the challenge period than that of the cohort heifers (147 compared to 120 ± 10 Carratelli units) and her OSI measure was 4.1 compared to 3.4 ± 0.4 for the cohort. Her ceruloplasmin concentrations were lower (157.5 mg/L) than those of the cohort heifers (247.1 ± 43.2 mg/L; Figure 2K).

Heifer 1250's β-hydroxybutyrate (Figure 2L) and urea concentrations were higher than those of the cohort heifers on the challenge day (4.1 compared to 2.9 ± 0.3 mM, respectively), while her glucose was lower (3.7 and 4.8 ± 0.3 mM, respectively) and her NEFA was similar (0.07 and 0.05 ± 0.02 mM, respectively).

In the afternoon after the challenge all heifers were scored for locomotion and offered *ad libitum* access to wheaten and millet hay 7 h after the challenge with close monitoring of feed intake. Heifer 1250's locomotion score was 1.5, compared to 1.1 ± 0.1 for the cohort heifers.

The first clinical sign that heifer 1250 may have had acute ruminal acidosis was a different behavior from her cohort heifers when hay was offered. The cohort heifers rushed to eat the hay, whereas heifer 1250 showed no interest in eating. At 17:30, 20:00, and 22:30 h checks she stood at the rear of the paddock, continued to show no interest in hay, had very loose diarrhea, and had dull mentation, but was sufficiently alert to not allow contact within 3 m.

On the morning after challenge (day 22), heifer 1250 stood in an area surrounded by watery, bubbly diarrhea, was dull, and walked tentatively. Her heart rate was 66 beats/min, respiration rate was 18 breaths/min, and her rumen was static and distended, as assessed by auscultation and ballotment of the paralumbar fossa. Rumen fluid and a blood sample were taken at 7:00 h and the immediate rumen pH measure was 5.5. Later VFA analysis (in duplicate) revealed heifer 1250 had no detectable concentrations of propionate, butyrate, valerate, isobutyrate, isovalerate, or caproate (Figure 2 C,E,F). The only VFA detected was 15.2 mM acetate (Figure 2B). Her ammonia concentration was 13.5 mM (Figure 2G) and total rumen lactate concentration was 67% (70 mM; Figure 2H) and rumen histamine concentration was 77% (224 mM), of the respective concentrations measured 20 h earlier. Her OSI had returned to a similar level to that reported at the adaptation II sampling (Figure 2J), but plasma ceruloplasmin was 266 mg/L (Figure 2K). Heifer 1250's β-hydroxybutyrate concentration was approximately 6-fold lower than 20 h earlier (Figure 2L). Her NEFA concentration was 58% higher, and her urea and glucose were approximately 53 and 9% lower, respectively, than 20 h earlier.

Heifer 1250 was treated by stomach tube with 200 g of sodium bicarbonate (Penice Soda Products; Osborne, South

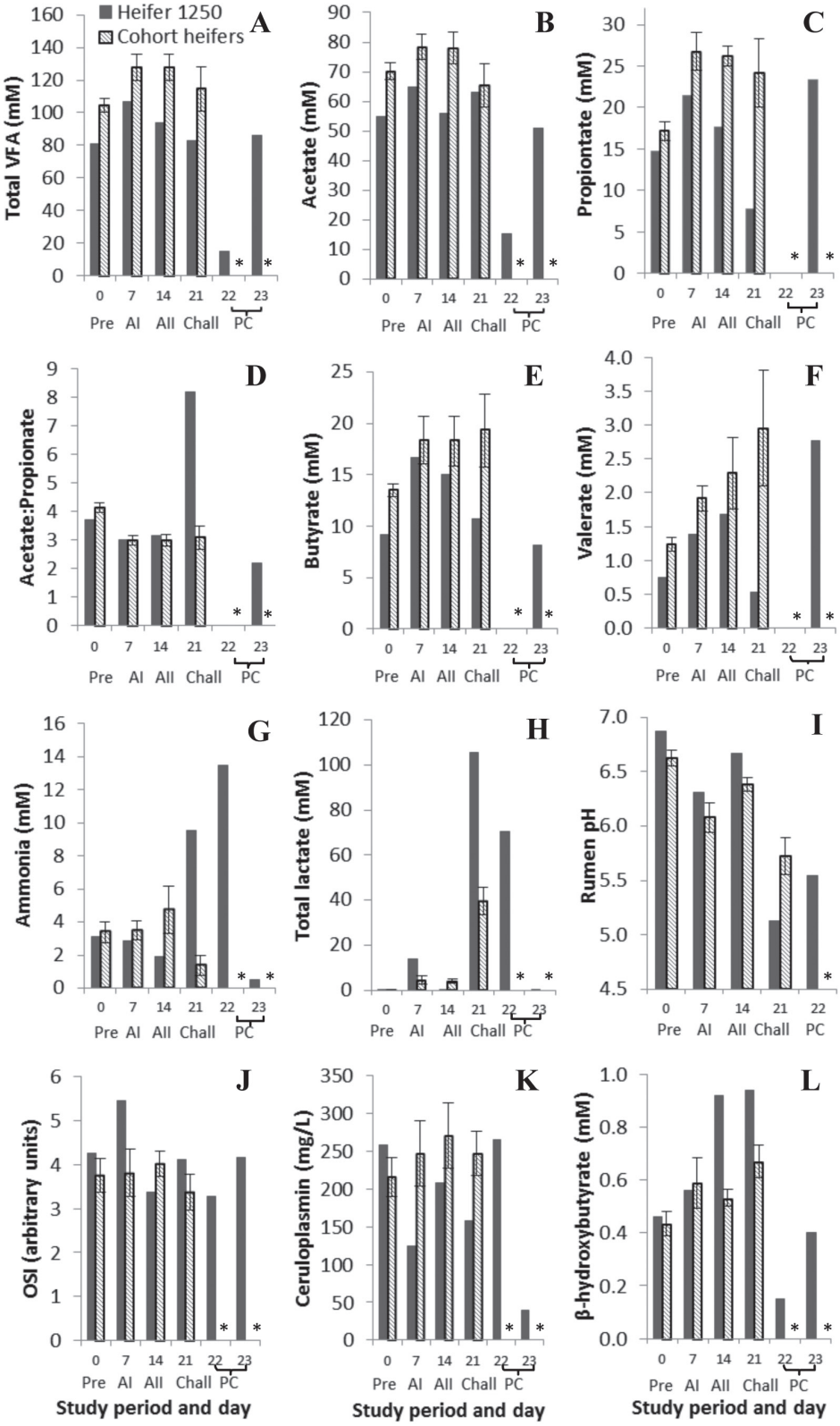

Figure 2. Ruminal total volatile fatty acid (VFA) (A); acetate (B); propionate (C); acetate:propionate (D); butyrate (E); valerate (F); ammonia (G); and total lactate (H); concentrations, and pH (I); plasma oxidative stress index (OSI) (J); plasma ceruloplasmin (K); and serum β-hydroxybutyrate (L) concentrations in heifer 1250 that was diagnosed with acute ruminal acidosis and the mean (± SEM) of 7 cohort heifers. Rumen fluid samples were collected using a stomach tube over the following 5 experimental periods on the study day indicated in parentheses: Pre – pre-adaptation (d 0); AI – adaptation I (d 7); AII – adaptation II (d 14); Chall – challenge (d 21); and PC – post-challenge (days 22 and 23). Refer to Figure 1 for rations offered during each experimental period. On challenge (day 21) rumen fluid samples were collected approximately 3.6 h after the challenge rations were consumed. *Rumen fluid was not collected from the 7 cohort heifers during the post-challenge period.

Australia, Australia) in 20 L of water at 7:30 h (d 22), before being scored for locomotion. She was reluctant to walk; her gait was slow and tentative and was scored at 2.5, 1 score higher than the previous day. No other heifers in the cohort had locomotion scores greater than 1.5. Heifer 1250 was housed in a separate yard where she was offered wheaten and millet hay and water, none of which she consumed. Later she was offered TMR but there was no detectable intake. She was given Flunixin meglumine (Ilium Flunixil; Troy Laboratories, Smithfield, New South Wales, Australia), 2.2 mg/kg BW, IM, at 14:30 h on the same day. She was examined on day 23, 48 h after challenge. At this time her appetite had resumed and she displayed no abnormal signs during a physical examination. She was subsequently returned to the main herd. Rumen fluid and blood measures from samples collected at 14:00 h on the same day (day 23) revealed that her total VFA concentrations had returned to a similar concentration to those on the challenge day. Her acetate to propionate ratio (Figure 2D) was 2.2, valerate concentration was much higher (2.77 mM) than her previous recordings (Figure 2F), ammonia had dropped markedly to 0.53 mM (Figure 2G), and total lactate concentration was very low (Figure 2H). Her rumen histamine concentration had decreased from the previous sampling by 70%, OSI increased by 21% (Figure 2J), BAP decreased by 15%, and plasma ceruloplasmin had decreased dramatically from 265.9 to 40.0 mg/L (Figure 2K). Heifer 1250's NEFA concentrations decreased from 0.17 to 0.09 mM, while her β-hydroxybutyrate (0.15 to 0.40 mM; Figure 2L), glucose (3.4 to 4.1 mM), and urea concentrations (1.9 to 2.7 mM) increased compared to the previous day. Her DMI (11.1 ± 1.1 kg DM; 91 ± 9.4% of offered ration) on the 2nd and 3rd day after challenge was similar to the cohort heifers' (8.9 ± 0.6 kg DM; 91 ± 6.2% of offered ration). Her locomotion score was normal on day 26 and consistent with that of the cohort heifers.

Over the study, heifer 1250 gained 34 kg BW and on average the cohort heifers gained 37 ± 4 kg BW. After being returned to the farm of origin heifer 1250 calved with no complications and the herd manager reported that she has been in excellent health since her return.

Discussion

The clinical signs of diarrhea, inappetence, and reluctance to move observed within 10 h of consumption of readily fermentable carbohydrate are consistent with the diagnosis of acute ruminal acidosis (1). Total lactic acid concentrations were within the range for acute acidosis (2), throughout the challenge and remained within this range 1 d after, until these returned to near zero 2 d after challenge. When considering the difference between rumen pH values collected by stomach tube and fistula, the observed pH of 5.1, 3.6 h after challenge ration consumption is consistent with acute acidosis definitions (9). However, rumen pH was 5.5 when the clinical signs were observed. It is possible rumen pH continued to decline after 3.6 h; however, rumen fluid collection at 2 to 5 h after the primary concentrate meal has been reported as the optimum sampling time (10) and symptoms were most visible at the time pH was 5.5.

This is the first reported case in cattle, in which the individual VFA's, propionate, butyrate, isobutyrate, isovalerate, valerate, and caproate, were not detected during clinical ruminal acidosis. An absence of propionate and butyrate concentrations was similarly noted in wethers within 14 h of being administered a sucrose solution at 15 g/kg BW through a rumen fistula (11). Further, all wethers had acetate concentrations of < 5 mM within 24 h of treatment and lactate concentrations were > 70 mM (11). No detectable or very low concentrations of propionate, acetate, and valerate were reported in sheep as early as 10 h after 1.4 to 2.7 kg of cracked wheat was administered through a rumen fistula (12). A total VFA concentration of < 20 mM was reported in a wether 24 h after consuming 6.8 kg of mangolds (13). It has been suggested that in acute acidosis, VFA concentrations are initially increased and then decline below 100 mM (2); however, these observations suggest a more dramatic decline is present.

This study was planned to induce an acidosis of non-life threatening severity based on feeding an amount of grain similar to percentages that are commonly fed to beef and dairy cattle and 0.2% of BW DMI of fructose which was designed to increase the risk of acidosis and mimic large exposures to sugar sources from access to high fructan or sucrose sources such as some fresh forages, brassicas, or citrus pulp, molasses, whey, or crystalline sugar. The fructose would have been rapidly fermented and may have increased rumen fluid passage and hindgut fermentation. Thus, grain fed in the milking parlour in combination with sugar sources and inadequate effective fiber may place cattle at increased risk of acidosis.

Individual animals have different susceptibilities to acidosis (14). Heifer 1250 had a different rumen fermentation profile to her cohorts at study day 0 which may indicate an inherent increased risk of acidosis. Her reduction in DMI to approximately 9.6 kg DM/d (79% of her offered ration/d) during the 4 d leading up to the challenge day may further indicate a greater susceptibility to acidosis compared with the cohort heifers before challenge. Her rapid consumption of 100% of the ration in approximately half the time of the cohort heifers may have also increased her risk of acidosis. As a consequence of heifer 1250 developing acidosis, the 6 heifers allocated to blocks B to D that were challenged on subsequent days were drenched with 200 g of sodium bicarbonate 4 h after the challenge.

Bramley et al (5) defined acidosis on the basis of a combination of individual VFA's, ammonia, rumen pH, and lactate measures. Definitions based on rumen pH cut points alone are not consistent and need to be accurately defined to allow appropriate diagnosis and treatment. Definitions also need to be specific for rumen fluid collection method to account for the heterogeneous nature of the rumen and also need to better accommodate single samples. This case study supports the work of Bramley et al (5) suggesting that rumen pH is not the key indicator of acidosis. Although heifer 1250 had lower rumen pH values than the cohort heifers, her pH was borderline for diagnosis of acute acidosis, whereas the very high lactate, and histamine and low VFA levels were suggestive of acute acidosis. This suggests that high rumen lactate concentrations do not necessarily correspond to a very low rumen pH and cowside VFA tests may be important diagnostic tools for acidosis in the future.

The increases in histamine were consistent with the knowledge that histamine generation occurs after feeding and can accumulate in the rumen during acidotic conditions (15,16). The increase in locomotion score suggests an association with histamine. While net absorption of histamine from the rumen appears to be low, and it is inactivated either during or after absorption into the blood, low rumen pH, and gut lesions may favor absorption (17).

The increase in OSI after challenge suggests heifer 1250 was subject to increased oxidative damage, an observation which is consistent with elevated oxidative stress measures in cattle fed high starch diets (18). The decrease in ceruloplasmin and BAP concentrations 2 d after the challenge may suggest they are involved in the pathogenesis of acidosis (19). The decrease in β-hydroxybutyrate, glucose, and urea and increase in NEFA concentrations 24 h after challenge support the presence of a metabolic disturbance. These measures may also be useful ancillary diagnostic measures for acidosis that could be developed for cowside use in the future.

In conclusion, this study emphasizes the variability in susceptibility to acidosis of individual cattle and the importance of detection of early clinical signs such as decreases in DMI, diarrhea, and change in demeanor. It also draws attention to the potential dangers of sugars in the diet, particularly in combination with grain. Clinicians should be aware that rumen pH is not always an adequate diagnostic method for evaluating acidosis, especially when a single sample is obtained. Other rumen measures including rumen VFA, lactic acid, and ammonia can provide a greater understanding of rumen function. Recovery of rumen function can occur rapidly after treatment despite severe perturbation. CVJ

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