Laminitis and dermatitis in heifers associated with excessive carbohydrate intake: skin lesions and biochemical findings

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ABSTRACT

The effects of a sudden addition of a large quantity of readily fermentable carbohydrate to the feed ration of pregnant heifers are described. Clinical and pathological changes caused by the resulting disease were confined to the digits and skin. The 4 acutely affected heifers were reluctant to get up or move (group II). They tended to lie down or stand with feet bunched together and the back arched, often shifting weight from limb to limb. They walked stiffly with great tenderness and pain in the digits. Extreme pain was noticed when the digits were examined. In 4 of 8 heifers, separation of the sole at the heel, with leakage of exudate, and under-running of the sole were observed. Necrotic dermatitis of the legs, alopecia and hyperkeratosis of the tail were noticed in all 8 heifers. Skin lesions appeared simultaneously. Four of the heifers (group I) recovered, and the other 4 (group II) were sent to slaughter. No *post mortem* examination was performed. The biochemical findings revealed a significantly higher concentration of total serum globulins and sodium, and increased activity, in CK, LDH and AST. A significantly decreasing pattern was noted in blood urea concentration, cholesterol, triglycerides, albumin and calcium. No significant differences among the various groups were found in the activities of amylase, GGT, and concentration of creatinine, total bilirubin, inorganic phosphorus, magnesium and potassium.

Key words: biochemistry, carbohydrate, dermatitis, heifers, laminitis.

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INTRODUCTION

Claw lameness in cattle can be due to trauma, infection, nutritional deficiency or metabolic disturbances. Various risk factors have been reported: age and breed of the animal, terrain, climatic conditions and farm management[5].

Feeding on excessive grain ration or non-structural carbohydrate, which are rapidly fermented in the rumen, is a causative factor in the development of laminitis, because of the propensity of these materials for inciting ruminal acidosis[3]. Laminitis has been defined as diffuse, acute, subacute or chronic aseptic pododermatitis, usually involving the tissues of several claws and leading to local signs[10].

Laminitis in dairy cattle kept under intensive management systems is now considered as an important disease, because of considerable economic losses caused by decreased production and by culling of severely affected animals.

Clinical studies of laminitis in cattle have been carried out[1,2,7,9] but, despite this published material, there is still a remarkable lack of specific, well-organised information on the laminitis-dermatitis syndrome in cattle associated with feeding of carbohydrate.

The present report describes the clinical, biochemical and radiological findings in an episode of laminitis in heifers caused by feeding large amounts of readily fermentable carbohydrate.

HERD HISTORY

The event occurred in a small dairy herd comprising 40 heifers, 15 first-calving cows, 40 lactating cows and 4 dry cows. The cows were kept all year round in a loose housing system, in large, completely covered open sheds, under a zero-grazing management system. The herd was kept in accordance with adequate standards of basic hygiene and mastitis control, and the animals had a mean milk yield of 8900 ℓ/yr. The cattle were divided into 5 groups: the lactating and the dry cows, and 3 groups of replacement heifers, divided according to age. The heifers were housed separately from the lactating cows. Milk production, fertility and herd diseases were recorded. Eight pregnant heifers (5–8 months) aged 19–22 months, kept in a separate barn, were affected.

The heifers were fed a complete mixed ration based on wheat silage, wheat straw, wheat hay, wheat bran, soya bean meal, poultry litter, barley grain, minerals and vitamin premix (Table 1). During a period of 3 weeks, each heifer was also fed industrial waste comprising 2.5 kg biscuit fragments daily (Table 2). Feeding of the biscuit fragments was discontinued after the appearance of the first clinical signs.

Food analysis revealed that the heifers received 12.0 kg dry matter containing 13 % total protein. The amount of non-structural carbohydrates (NSC) in the ration increased by 77 % during the above-mentioned 3-week period, while the roughage component decreased to 40.6 %, the energy concentration increased to 9.53 megajoules of metabolisable energy (MJME) per kg dry matter, and the degradable organic matter increased by 41 % (Table 1).

The affected heifers were classified into 2 groups: sub-acute (4 heifers, group I) and acute (4 heifers, group II) laminitis, respectively. Ten unaffected heifers of similar age and weight, randomly chosen from a neighbouring herd kept under similar management conditions but with a different feed ration, served as a control group.

CLINICAL FINDINGS

The first visit to the farm was made 1 week after the appearance of the clinical signs. Eight heifers were affected; 4 of those (group II) tended to spend most of the time lying down. When standing they

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assumed a posture typical of acute laminitis: arched back, feet bunched together under the belly and shifting weight frequently. When aroused to walk, the gait was stiff and tender, indicating acute pain in the claws. When the claws were examined by pressure exerted on the sole with pincers, the heifers exhibited severe pain. The claw lesions included separation of the sole at the heel, with leakage of exudate, and underrunning of the sole (Fig. 1).

The other 4 heifers (group I) had less severe signs of laminitis but did have lesions of diffuse dermatitis. The skin lesions occurred in all 8 affected heifers, started above the coronary band and extended up to the stifle (Fig. 2). The tail was also affected (Fig. 3). The nature of the dermatitis was ulcerative, necrotic skin thickening and wrinkling with areas of alopecia. All affected heifers were emaciated.

LABORATORY EXAMINATION

Blood analysis

Blood samples were collected from affected and control groups by jugular venipuncture in plain evacuated tubes for recovery of serum. Following blood clotting, the serum was separated and biochemically analysed within 24 h of sampling.

Enzymes, metabolytes and minerals were analysed spectrophotometrically with a selective chemistry analyser (Kone: Kone Corporation, Finland) at 30 °C, according to standard methods.

The activities of the following enzymes were determined: creatine phosphokinase (CK), alkaline phosphatase (ALP), amylase (Amyl), lactate dehydrogenase (LDH), γ-glutamyl-transferase (GGT), L-alanine aminotransferase (ALT) and L-aspartate aminotransferase (AST).

Total globulin content was calculated by subtracting the albumin (ALB) value from the total protein. The following metabolytes and mineral concentrations were measured: urea, triglyceride, cholesterol, creatinine, total bilirubin, Calcium (Ca), inorganic phosphorus (Pi) and magnesium (Mg). The electrolytes, sodium and potassium, were determined by using specific electrodes (Kone Microlyte Ion Selective Analyzer, Kone Corporation).

Histology

Skin biopsies were taken from the legs and tails of the clinically affected heifers, and were fixed in 10 % neutral buffered formalin, embedded in paraffin wax, sectioned at 4–5 μm thickness, stained with haematoxylin and eosin (HE) and microscopically evaluated.

Table 1: **Composition of the daily feed ration of heifers in the affected and control herds (kg dry matter).**

	Control herd	Affected herd
Wheat silage	0.197	0.470
Wheat straw	4.500	3.346
Wheat hay	1.305	1.060
Wheat bran	0.620	1.066
Orange peel	1.149	—
Soya bean meal	—	0.553
Poultry litter	2.534	2.058
Gluten	1.695	—
Barley grain	—	1.215
Biscuit fragments	—	2.225
Mineral and vitamin premix	0.004	0.004
Dry matter	12.000 kg	12.0 kg
Wet matter	21.336 kg	14.692 kg
Total protein	1.495 kg	1.558 kg
Megajoules of metabolisable energy (MJME)	105.275	114.378
Non-structural carbohydrate	1.803 kg	3.190 kg
Protein %	12.45 %	13 %
Energy concentration (MJME/kg dry matter)	8.77	9.53
Roughage component	50.0 %	40.6 %
Degradable organic matter	3.371 kg	4.747 kg

Table 2: **Composition of the biscuit fragments (based on kg dry matter).**

Dry matter	89 %
Protein	9.7 %
Megajoules of metabolisable energy	12.82
Non-digestible fibre	9 %
Calcium	1.7 g
Phosphorus	2.5 g
Fat	115 g
Non-structural carbohydrate	675 g
Ash	28 g
Degradable organic matter	750 g

Fig. 1: **Separation of the sole at the heel with leakage of exudate and under-running of the sole.**

Fig. 2: **Ulcerative dermatitis of the legs.**

Radiological examination

The digits of the severely affected heifers (group II) were examined radiographically.

Statistical analysis

This was carried out with SPSS 6.1 for Windows; *P* values of less than 0.05 were considered significant.

RESULTS

Blood analysis

A significantly higher concentration of total serum globulins was recorded in the severely laminitic heifers (group II) (40.2 ± 7.5 g/ℓ) than in the control heifers (27.2 ± 1.8 g/ℓ) ($P < 0.001$), while in the subacute group (group I) only a small increase, with a mean concentration of 28.7 ± 3.3 g/ℓ, was observed (Fig. 4). In contrast to the globulins, a gradual, non-significant decrease in the albumin concentration was noted, from 38.2 ± 1.5 g/ℓ in control heifers to 36.2 ± 1.7 g/ℓ in group I and to 31.8 ± 2.5 g/ℓ in the severely affected heifers (group II) (Fig. 4).

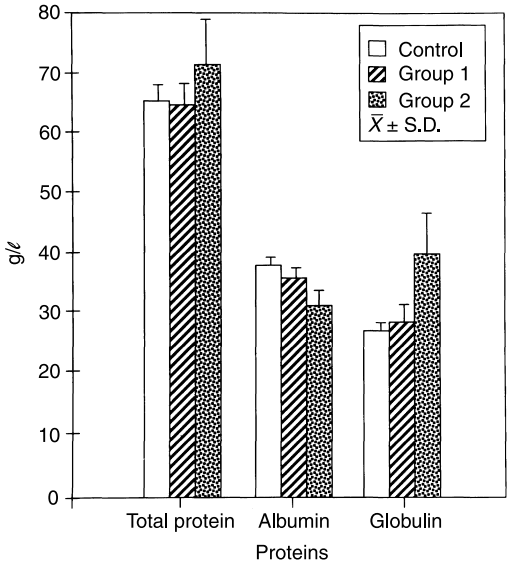

Fig. 4: **Serum proteins of heifers with laminitis caused by excessive carbohydrate intake.**

A significantly decreasing pattern was seen in the blood urea concentrations: 37.9 ± 3.5 mg/dℓ for the control heifers; 31 ± 2.1 mg/dℓ for group I; and 25.9 ± 5 mg/dℓ for group II ($P < 0.001$) (Fig. 5). A marked decrease was seen in cholesterol concentration, from 119 ± 18 mg/dℓ in the control group to 102 ± 14 mg/dℓ in group I and to 68 ± 19 mg/dℓ in group II ($P < 0.001$) (Fig. 5). A decrease was noted also in triglyceride concentration; 43.9 ± 14.7 mg/dℓ in the control group *vs* 39.4 ± 9.7 and 26.8 ± 7.1 mg/dℓ in groups I and II respectively.

The mean activities of the enzymes CK, LDH and AST were higher ($P < 0.001$) in the severely laminitic heifers (group II) than in the control group (Figs 6, 7): 348 ± 88 *vs* 64 ± 13 U/ℓ for CK; 2178 ± 333 *vs* 1535 ± 166 U/ℓ for LDH; and 85.5 ± 25.5 *vs* 43.6 ± 6.2 U/ℓ for AST.

Moderate increases in CK, LDH, ALT, AST and ALP were also seen in group I (Figs 6, 7). Moderate decreases in the mean concentrations of the enzymes ALP and ALT were noted in group II (145 ± 34 *vs* 120 ± 13) ($P < 0.01$) and (18.9 ± 3.2 *vs* 13.5 ± 5.4) ($P < 0.05$), respectively.

A marked decrease in the mean concentration of calcium and an increase in that of sodium were noted for both groups. Calcium concentration decreased from

Fig. 3: **Alopecia and thickening of the tail skin.**

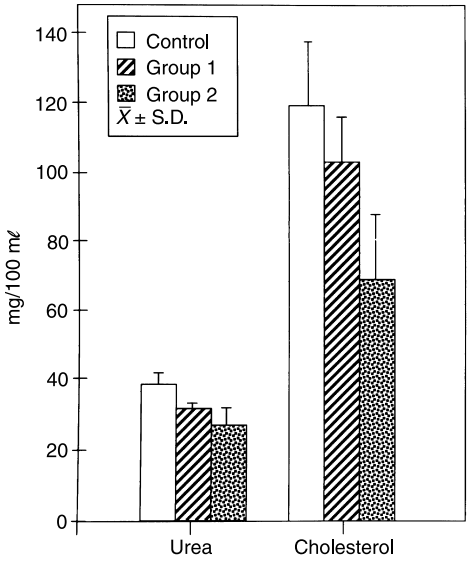

Fig. 5: **Serum urea and cholesterol of heifers with laminitis caused by excessive carbohydrate intake.**

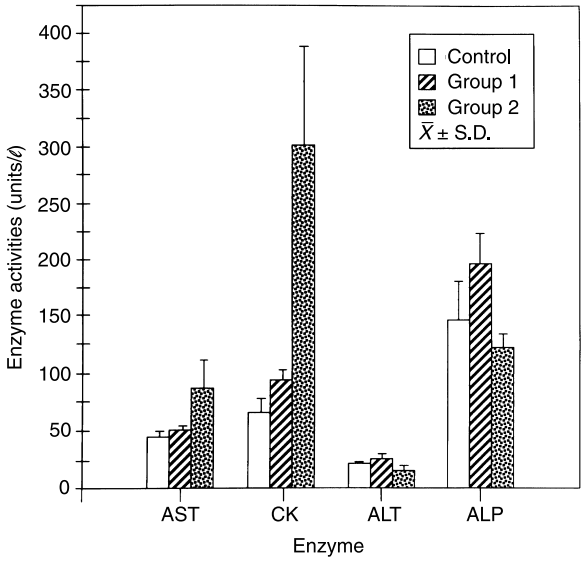

Fig. 6: **Serum AST, CK, ALT and ALP of heifers with laminitis caused by excessive carbohydrate intake.**

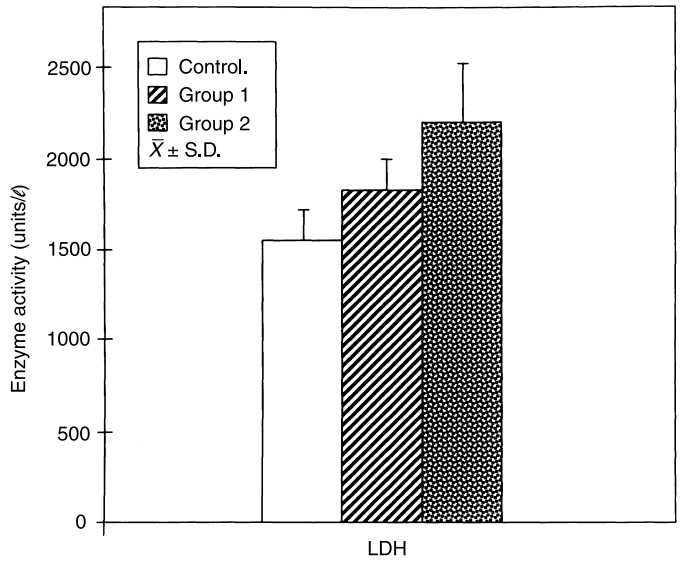

Fig. 7: **Serum LDH of heifers with laminitis caused by excessive carbohydrate intake.**

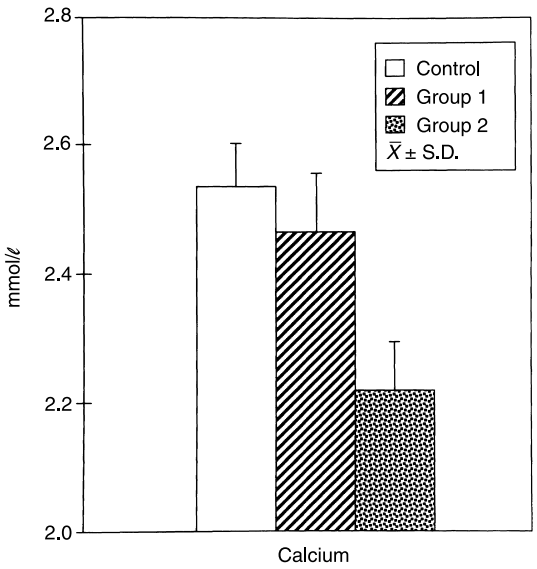

Fig. 8: **Serum calcium of heifers with laminitis caused by excessive carbohydrate intake.**

2.54 ± 0.07 mmol/ℓ in the control group to 2.22 ± 0.08 mmol/ℓ in group II ($P < 0.001$) (Fig. 8), and sodium increased from 137.6 ± 0.6 mmol/ℓ in the control group to 144 ± 2.4 mmol/ℓ in group II ($P < 0.001$) (Fig. 9).

No significant differences among the various groups were found in the concentrations of amylase, GGT, creatinine, total bilirubin, inorganic phosphorus, magnesium and potassium.

Histology

Histology of the skin biopsy samples revealed necrotic dermatitis, hyperkeratosis and dermal oedema.

Radiology

Radiological examination included dorsoventral and lateral radiographs. No evidence of rotation of the distal phalanx was seen, but there was marked soft tissue swelling on both projections.

DISCUSSION

Since these animals had been kept on deep straw bedding, it would appear that the claw lesions described were not of traumatic origin, but were due to a metabolic insult, possibly originating from carbohydrate overfeeding.

The sudden change in feed appears likely to have been a contributory factor in this lameness-dermatitis syndrome. The addition of a large quantity of readily fermentable carbohydrate that corresponds with degradable organic matter (DOM) to the feed ration of animals not accustomed to it, and consequent excess production of lactic acid, may have caused rumen acidosis and release of histamine and other vaso-active agents[11]. This process may have led to subclinical and clinical laminitis, emaciation and severe skin lesions on the legs and tail.

The findings in the present investigation, that laminitis is related to the metabolic disturbance caused by carbohydrate overfeeding in cattle, confirmed previous findings[2,9].

Feeding rations high in easily digestible carbohydrates is regarded as the most important predisposing cause of laminitis in cattle[4,8,11]. There is consensus worldwide that the vascular system of the bovine digit plays a significant role in the pathogenesis of bovine laminitis. The normal function of the bovine claw can be disrupted by a change in its blood flow. Inappropriate activity of the peripheral digital vessels contributes to the development of tissue anoxia and an inadequate supply of nutrients to keratin-producing cells and the epidermis, followed by ischaemic necrosis and degeneration of the horn-producing structures[6,12]. Periph-

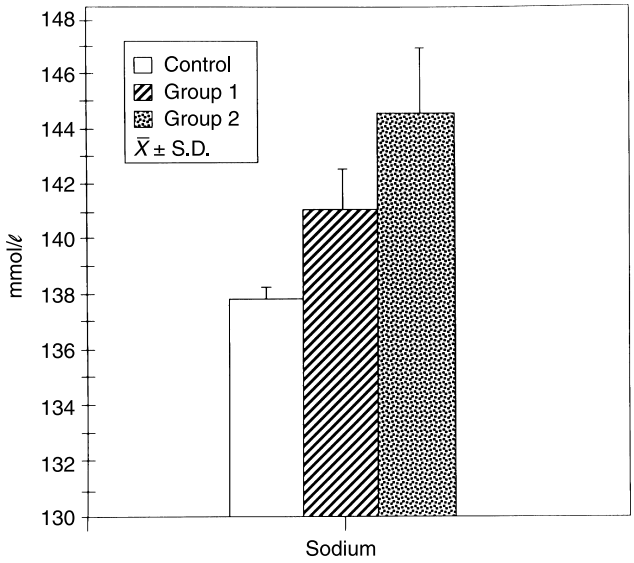

Fig. 9: **Serum sodium of heifers with laminitis caused by excessive carbohydrate intake.**

eral circulatory failure may be caused by toxic vaso-active substances such as histamine, lactic acid and endotoxin[11].

The inflammatory process of laminitis resulted in an increase in total protein concentration due to increased levels of globulins in the blood. The severe laminitis and dermatitis of the legs, and consequent prolonged periods of recumbency, may have led to considerable muscle damage that resulted in increased activities of CK, LDH and AST in serum.

Our findings of decreased urea concentration in the laminitic heifers are in contrast to a previous report of increased urea concentration[9]. The decreased urea concentration that we found could be due to the utilisation of the ammonia for amino-acid production. The protein concentration was quite low in relation to the energy content of the ration.

The lower blood urea concentration seen in the sick animal groups could also be a result of lower food and protein consumption, a hypothesis supported by the lower cholesterol and triglyceride levels.

The decrease in calcium concentration may be related to both the decrease in serum albumin, which binds calcium, and the lower feed intake. It could also be due to higher concentrations of lactate, which would bind some of the ionised calcium, leading to a lower serum calcium concentration. The increase in sodium concentration could be a result of decreased water intake due to lameness of the affected animals.

The pathogenesis of associated dermatitis in the affected heifers is not yet understood. Outbreaks of laminitis and associated dermatitis were previously seen in Israel, but have not been recorded. Future similar outbreaks should be carefully recorded and investigated.

This outbreak is a solid reminder of the need to exercise control measures to prevent abrupt changes in the feed ration and consequent development of ruminal acidosis. It appears that animals that undergo abrupt feed changes are more prone to metabolic disorders, as occurred in the present case. All dietary changes, especially to the carbohydrate-roughage ratio, should be made gradually and in accordance with the age and lactation stage of the animal.

REFERENCES

1. Bargai U, Shamir I, Lublin A, Bogin E 1962 Winter outbreaks of laminitis in dairy calves: aetiology and laboratory, radiological and pathological findings. *Veterinary Record* 131: 411–414
2. Bradley H K, Shannon D, Neilson D R 1989 Subclinical laminitis in dairy heifers. *Veterinary Record* 125: 177–179
3. Dunlop R H, Hammond B P 1965 D-lactic acidosis in ruminants. *Annals of the New York Academy of Science* 119: 1108
4. Greenough P R 1990 Observations on bovine laminitis. *In Practice* 12: 169–173
5. Harris D J, Hibburt C D, Anderson G A, Younis P J, Fitzpatrick D H, Dunn A C, Parsons I W, McBeath N R 1988 The incidence cost and factors associated with foot lameness in dairy cattle in south-western Victoria. *Australian Veterinary Journal* 65: 171–176
6. Hood D M, Stephens K A 1981 Physiopathology of equine laminitis. *Compendium of Continuing Education for Practicing Veterinarians* 3: 454–459
7. MacLean C W 1965 Observations on acute laminitis of cattle in South Hampshire. *Veterinary Record* 77: 662–672
8. MacLean C W 1966 Observations on laminitis in intensive beef units. *Veterinary Record* 78: 223–231
9. Mullen P A 1976 Overfeeding in cattle: clinical, biochemical and therapeutic aspects. *Veterinary Record* 98: 439–443
10. Nilsson S A 1963 Clinical, morphological and experimental studies of laminitis in cattle. *Acta Veterinaria Scandinavica* (Supl) 4: 9–304
11. Vermunt J J 1992 'Subclinical' laminitis in dairy cattle. *New Zealand Veterinary Journal* 40: 133–138
12. Vermunt J J, Leach D H 1992 A scanning electron microscopic study of the vascular system of the bovine hind limb claw. *New Zealand Veterinary Journal* 40: 146–154