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Field Study of Dairy Cows with Reduced Appetite in Early Lactation: Clinical Examinations, Blood and Rumen Fluid Analyses

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Steen A: Field study of dairy cows with reduced appetite in early lactation: clinical examinations, blood and rumen fluid analyses. Acta vet. scand. 2001, 42, 219-228. – The study included 125 cows with reduced appetite and with clinical signs interpreted by the owner as indicating bovine ketosis 6 to 75 days postpartum. Almost all of the cows were given concentrates 2 to 3 times daily. With a practitioners view to treatment and prophylaxis the cows were divided into 5 diagnostic groups on the basis of thorough clinical examination, milk ketotest, decreased protozoal activity and concentrations, increased methylene blue reduction time, and increased liver parameters: ketosis (n=32), indigestion (n=26), combined ketosis and indigestion (n=29), liver disease combined with ketosis, indigestion, or both (n=15), and no specific diagnosis (n=17). Three cows with traumatic reticuloperitonitis and 3 with abomasal displacement were not grouped. Nonparametric methods were used when groups were compared. Aspartate aminotransferase, glutamate dehydrogenase, gamma-glutamyl transferase and total bilirubin were elevated in the group with liver disease. Free fatty acids were significantly elevated in cows with ketosis, compared with cows with indigestion. Activity and concentrations of large and small protozoas were reduced, and methylene blue reduction time was increased in cows with indigestion. The rumen fluid pH was the same for groups of cows with and without indigestion. Prolonged reduced appetite before examination could have led to misclassification. Without careful interpretation of the milk ketotest, many cases with additional diagnoses would have been reported as primary ketosis. Thorough clinical examination together with feasible rumen fluid examination and economically reasonable blood biochemistry did not uncover the reason(s) for reduced appetite in 14% of the cows. More powerful diagnostic methods are needed.

bovine ketosis; indigestion; rumen acidosis; dairy cattle.

Introduction

Reduced appetite in dairy cows in early lactation is one of the clinical signs of bovine ketosis and indigestion caused by simple indigestion or subacute ruminal acidosis (*Radostits et al.* 2000). After indigestion was defined as a separate diagnosis in the Norwegian Disease Recording System in 1989, the number of reported cases of bovine ketosis decreased, although the incidence is still high. Several investigations indicate that the figures from the health card statistics may be inaccurate (*Cote et al.* 1969, *Øverby et al.* 1974, *Simensen et al.* 1990). Terms like "doubtful", "false", physiologic, spontaneous, alimentary, type I and II, and secondary ketosis have been used. In many thoroughly examined ketosis patients various concurrent diseases can be found (*Cote et al.* 1969, *Dale* 1978, *Dirksen* 1992), i.e. secondary ketosis is more frequent than usually thought. Some investigations detected subclinical keto-

sis and discussed its connection to milk yield and fertility (*Baird* 1982, *Qvesel* 1983, *Andersson & Emanuelson* 1985).

Veterinarians in clinical practice are often faced with the problem to distinguish between bovine ketosis and indigestion in cows with reduced appetite in early lactation. The purpose of this investigation was to use established metods in clinical examination, clinical pathology and rumen fluid analyses and evaluate the results from examining cows with reduced appetite in the most critical period for developing ketosis postpartum.

Materials and methods

Patients

The patients were located in a clinical practice on 5 islands, "Nordøyane", in the northwest of Norway. The selection of patients was determined by the practical working situation: number of visits and the workload of the veterinarian and the ambulance boat. Cows with reduced appetite the first 3 months of lactation, and with clinical signs interpreted by the owner as ketosis, were examined. Of a total of 125 cows, 24, 36, 32 and 33 were in first, second, third, or fourth to eight lactation, respectively. All cows were from tie stall barns.

Anamnesis

Patient data. Start of clinical signs, appetite, feed refused and milk production. Lactation number, days postpartum, milk yield during previous lactation, milking curve, clinical signs of ketosis earlier in present lactation.

Herd data. Cows with similar clinical signs. Type of concentrates and quantity fed, number of meals per day, silage feeding, other roughages, minerals. Prophylactic feeding or medication.

Clinical examination

The clinical examination included check of body temperature, heart rate, inspection of mucous membranes in mouth and vagina, teeth, palpable lymph nodes, auscultation of the heart and lungs, bilateral percussion and auscultation for diagnosis of abomasal displacement, rumen sounds and motor activity, auscultation over trachea during ructus and during deep palpation in the reticulum area for diagnosis of traumatic reticuloperitonitis, palpation of claws for diagnosis of laminitis, rectal exploration of uterus, ovaries, kidney, rumen and intestines with evaluation of consistency of rumen contents and faeces, finally udder and milk.

Clinical pathology

Milk was tested for ketone bodies with Rothera test powder, consisting of sodium nitroprusside 50mg, anhydrous sodium carbonate 10g, and ammonium sulphate 20g. Results were graded from 0 (negative) to 3 (maximum).

Rumen fluid was collected through the Thygesen stomach tube (*Sørensen & Schambye* 1955), with a modification to avoid contamination from saliva: a beet cup replaced the pipe-formed metal sieve. The inner polyethylene tube was plugged with a cotton plug that was blown out with a bicycle tire pump after positioning of the stomach tube. A suction pump was used to withdraw 100-200 ml of rumen fluid.

The rumen fluid was immediately examined with respect to colour, odour and consistency. The pH was measured with portable electronic pH-meter (pHep, Hanna Instruments, Italy). Two test tubes were filled with 20 ml rumen fluid, and the content in one of the tubes was mixed with 1 ml methylene blue 0.3 mg/ml. The test tubes were placed in a water bath at 38 °C (thermos). After 3 and 6 min the test tubes were examined to determine the methylene blue reduction time and the sedimentation time (*Dirksen* 1990).

Protozoan activity was observed in the control

test tube with a magnifying lens (× 20). The number of large and small protozoa was cathegorized to many, few or none in a drop of mixed rumen fluid. Rumen bacteria were examined in the home laboratory on Gram stained smears.

Blood samples were drawn from the coccygeal vein. Heparinised plasma was analysed for aspartate aminotransferase (AST), glutamate dehydrogenase (GD), gamma-glutamyl transferase (GGT), total bilirubin, free fatty acids, total protein, calcium (Ca), magnesium (Mg) and phosphorous (P) at the Central Laboratory, The Norwegian School of Veterinary Science, on a Technicon RA-1000® with methods approved by the manufacturer (Bayer Corp., N.Y., USA). All enzymes were analysed at 37°C. GD and GGT were not analysed from all patients.

Treatment and response

All patients were treated with 100mg prednisolon (Prednisolon® Leo Vet., Løvens Kemiske Fabrik, Denmark), 10mg dexamethasone (Vorenvet® vet., Boehringer Ingelheim, Germany), and multivitamin B (Becoplex® vet., Boehringer Ingelheim, Germany). Cows with indigestion were in addition given a rumen fluid stimulant (Proviton®, Løven Agro, Denmark). One cow which collapsed during the examination was given calcium and magnesium chloride intravenously.

The farmer recorded the time the cow started eating the whole ration as a measure of response upon treatment.

Statistical analysis

Nonparametric descriptive and analytic methods were used. Enzymatic and metabolic values were plotted and central tendency and variability were given with medians, interquartile ranges, and the 10^{th} and 90^{th} percentiles. Differences between groups were tested with nonparametric one way analysis of variance, Kruskal-Wallis test, using JMP® (*SAS Institute Inc.* 1995), $\alpha = 0.05$. Pairwise comparisons were performed by means of Wilcoxon two sample test when the Kruskal-Wallis test was significant. A chi-square test was used when ordinal variables were compared.

Table 1. Diagnostic criteria for 5 disease categories.

Group	Diagnosis	Criteria
K	Ketosis	Ketotest = 2 or 3
I	Indigestion	Ketotest = 0 or 1 At least one deviating rumen fluid parameter: -Methylene blue reduction time > 3 min -Few large or small protozoa -Reduced protozoan activity
K+I	Ketosis and indigestion	As above for both "Ketosis" and "Indigestion"
L	Liver disease in addition to ketosis, and/or indigestion	In addition to "Ketosis", "Indigestion", or both - Two or more increased liver parameters: -AST >125 U/l -GD > 63 U/l -GGT > 46 U/l -Total bilirubin > 6 μmol/l
N	No specific diagnosis	Ketotest = 0 or 1. No deviating rumen fluid or liver disease parameters

Results

On the basis of clinical examination, rumen fluid examination, and clinical biochemistry the material was divided into 5 diagnostic groups: ketosis (K), indigestion (I), combined ketosis and indigestion (K+I), liver disease in addition to ketosis, indigestion, or both (L), and a group with reduced appetite but none of the above diagnoses (N). Diagnostic criteria for the groups are given in Table 1 and the distribution of patients into each group is given in Fig. 1. Three

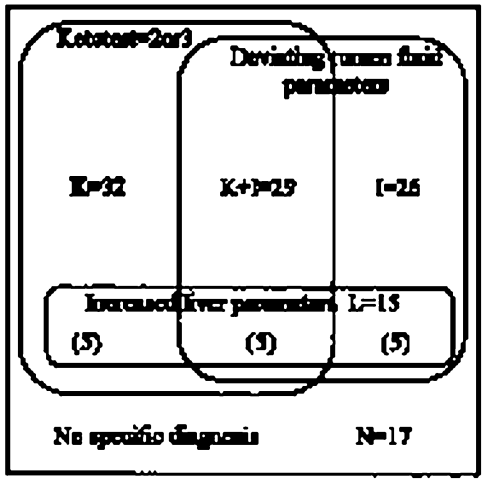

Figure 1. Distribution of cows into diagnostic groups based on ketotest = 2 or 3, deviating rumen fluid parameters and increased liver parameters, N = 125. K = Ketosis (n=32), I = Indigestion (n=26), K+I = Ketosis and indigestion (n=29), L = Liver disease in addition to ketosis, indigestion, or both (n=15), N = No specific diagnosis (n=17). See Table 1 for diagnostic criteria.

cows with traumatic reticuloperitonitis and 3 cows with abomasal displacement were not grouped.

Grass silage was used as the sole roughage among 77% of the cows in the material, and 8% and 2% of the cows received turnips and hay in addition to grass silage. Fresh grass was given to 13% of the cows. Half of the cows were given roughage in restricted amounts twice daily, while the other half received roughage ad libitum. Concentrates were given according to milk production level, 2 kg for the first 10 kg of milk with additional 1 kg per 2.5 kg milk. Most of the cows (61%) were fed concentrates twice daily, 37% 3 times daily and only 2% 4 times daily. There were no differences between the diagnostic groups. Median milk yield the previous lactation for multiparous cows was 6 400 kg, the 10th and 90th percentiles were 5 000 and 7 000 kg, respectively. There were no differences between the diagnostic groups.

Table 2. Duration of reduced appetite before examination, percentage distribution within the diagnostic groups defined in Table 1 and Fig.1. Same day or 0, 1, 2, and ≥3 denotes that the cows had reduced appetite in the morning, since yesterday, the day before yesterday or longer, respectively.

Days	K	I	K+I	L	N
≥3	30	54	38	46	43
2	20	9	12	0	14
1	43	29	46	46	36
0	7	8	4	8	7

Median examination time postpartum was 28 days, see Fig. 2. There were no differences between the diagnostic groups.

Thirty to 54% of the cows in the different diagnostic groups had reduced appetite more than 2 days before examination, see Table 2.

The results of the analyses on enzymes and metabolites are plotted in Fig. 3. Increased val-

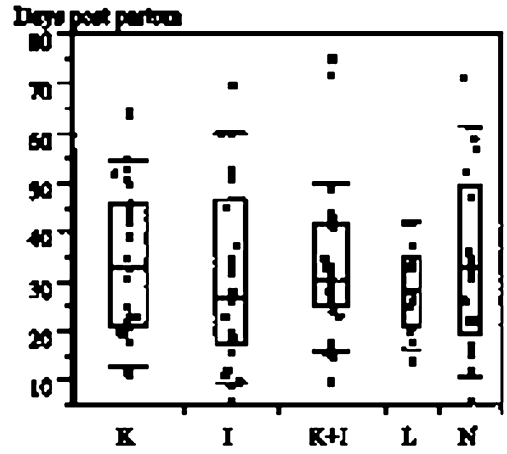

Figure 2. Examination time postpartum, percentage distribution within the diagnostic groups defined in Table 1 and Fig.1.

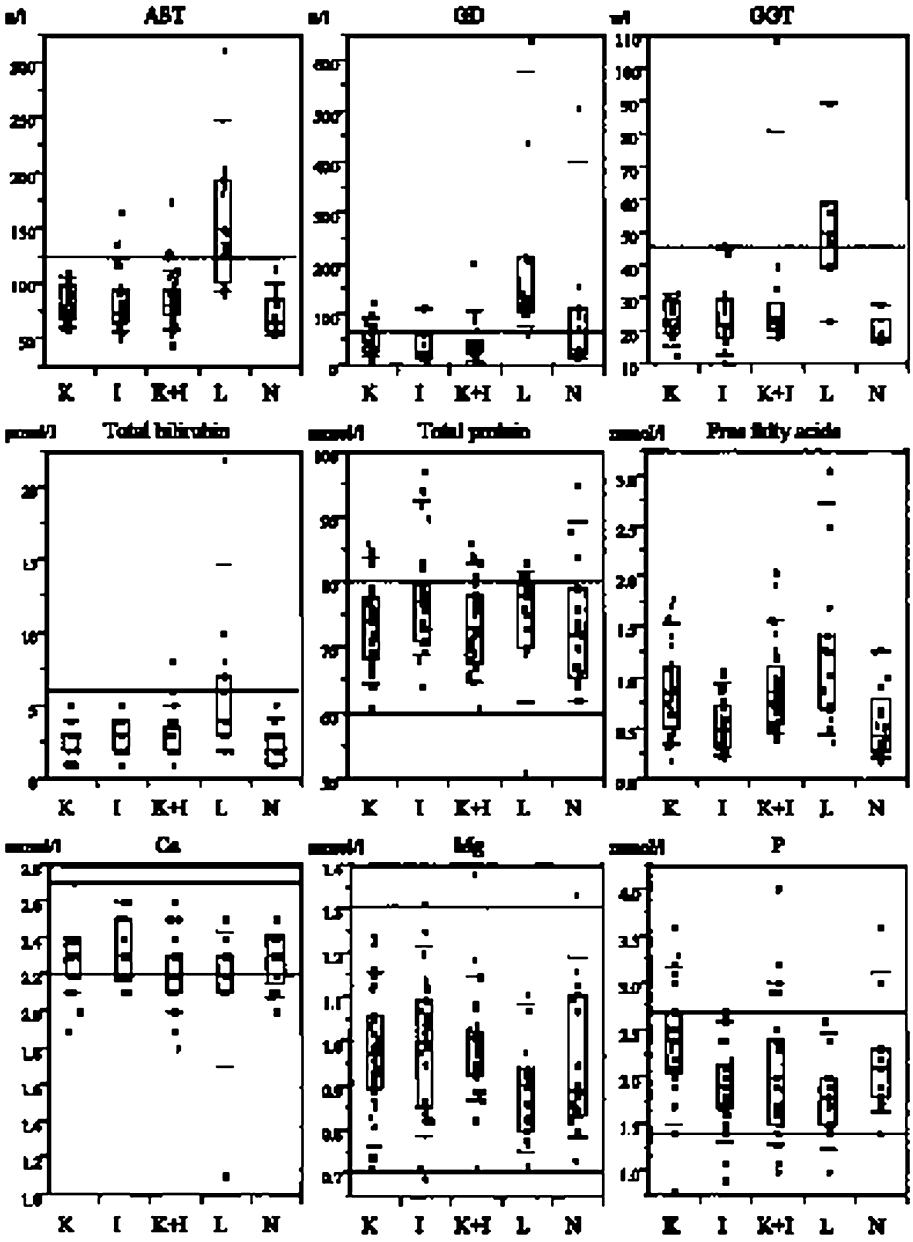

Figure 3. Quantile box plots of clinical biochemical parameters in the 5 diagnostic groups defined in Table 1 and Fig.1: The box shows the group median as a line across the middle and the quartiles (25th and 75th percentiles) as its ends. The 10th and 90th percentiles are shown as lines above and below the box. Dotted lines show the reference range (95% confidence interval) established at The Norwegian School of Veterinary Science.

Table 3. Deviating rumen fluid parameter details from cows classified as having indigestion (I), ketosis and indigestion (K+I), liver disease and indigestion but not ketosis (L-K), see Fig. 1. Percentage distribution of gradings for each parameter.

Rumen fluid parameter n	I 26	K+I 29	L-K 10
Protozoan activity			
Good	50	59	60
Reduced	33	38	40
None	15	3	0
Large protozoa:			
Many	21	42	40
Few	42	50	60
None	37	8	0
Small protozoa:			
Many	26	37	20
Few	53	46	70
None	21	17	10
Methylene blue reduction time:			
<3 min	85	61	60
<6 min	15	28	40
>6 min	0	11	0

ues of AST, GD, GGT and total bilirubin were used to group cows with liver disease. Free fatty acids were significantly increased in cows with ketosis compared with the cows with indigestion, irrespective of other diagnoses. Concentrations of other enzymes and metabolites did not differ significantly.

Cows were diagnosed as having indigestion based on at least one deviating rumen fluid parameter. Rumen fluid parameter details from cows in the I group, the K+I group, and from cows in a subgroup of the L group, with indigestion but without ketosis (L-K), are given in Table 3.

Range of rumen fluid pH was 5.9 to 7.9. There was no difference in rumen motor activity in cows with or without deviating rumen fluid parameters. Microscopic evaluation of Gram-stained rumen fluid showed only Gram negative bacteria and no differences between groups. Five of the cows in the I group and one in the K group had diarrhoea. Percentage of cows within the groups with dry and firm feces was 38% (K), 8% (I), 41% (K+I), 20% (L), and 12% (N). Within the groups, 13% (K), 23% (I), 10% (K+I), 7% (L), and 6% (N) of the cows had been treated for ketosis earlier in the lactation.

Left abomasal displacement was diagnosed in 3 cows, and occured on days 8, 11 and 31 postpartum, respectively. Only the cow diagnosed 31 days postpartum had increased concentrations of liver enzymes. There were no deviating rumen fluid parameters. Two of the cows had a maximal ketotest reaction and strongly increased free fatty acid concentrations (2.00 and 2.62 mmol/l), the third cow (11 days postpartum) had not ketonemia.

Two of the 3 cows with traumatic reticuloperitonitis had increased body temperature, 39.3 and 39.9 °C. All 3 had a maximal ketotest reaction and increased free fatty acids concentrations (1.40-1.83 mmol/l). The frequency of rumen contractions was decreased ($p>0.05$), 2-3 rumen contractions per 2 min against a median value of 4 for all the cows in the material.

One cow in the L group with a maximal ketotest reaction, increased total bilirubin and diarrhoea 43 days postpartum, had a calcium concentration of 1.1 mmol/l. This cow collapsed during

Table 4. Response upon treatment: Percentage distribution within the diagnostic groups when the cows started eating the whole ration, within one (<1) or within two (<2) days. Cows which did not respond upon treatment are grouped together with those which started eating the whole ration after 3 days (≥3).

Days	K	I	K+I	L	N
≥3	12	22	28	46	33
<2	50	45	50	36	42
<1	38	33	22	18	25

examination and was treated with calcium and magnesium chloride.

One of the cows in the K group showed signs of cerebral disturbance with manic chewing movements with salivation, licking of inanimate objects, and head pushing and leaning into the stanchion.

Most of the cows in the ketosis group responded well to treatment, 38% started eating their whole ration the same evening, 88% had started the day after, see Table 4. Nearly half of the cows with liver disease and a third of the cows with no specific diagnosis had continued depressed appetite and did not resume normal appetite within one week.

Discussion

In the present study cows with the same main diagnosis were grouped with a practising veterinarians view to treatment and prophylaxis, but distinctions between the groups were not clear-cut. Based on milk ketotest score, rumen fluid parameters, liver parameters, and the total clinical picture the material was divided into 5 groups (Table 1). Primary ketosis (K) and indigestion (I) cases were grouped separately. Because of overlapping 2 further groups were required to make homogenous groups, one was a combination of the first 2 (K+I), and the other with additional liver disease (L). The rest of the material that did not fit into any of these groups was lumped together in the N group. The traumatic reticuloperitonitis and abomasal dislocation patients that had high scores on the ketotest were diagnosed separately and not grouped.

The cows in the present study were feed concentrates according to requirement and only 2 or 3 times daily. The main ingredient in the concentrate was barley produced in Norway with starch that is easily degraded in the rumen (*Nocek & Tamminga* 1991). Low rumen pH occurs when processed concentrate is given in large quantities a limited number of times per day (*Owens et al.* 1998). Low rumen pH depresses cellulose digestion and intake of roughage, leading to problems of acidosis and secondary ketosis due to off-feed conditions (*Ørskov* 1999).

Rumen fluid pH drops during and after a meal and usually returns to the inital level within 12 h (*Nordlund & Garrett* 1994). The best diagnostic power will be obtained when rumen fluid is sampled when pH is expected to be at or near its nadir. In herds where roughage and concentrates are fed separately, samples should be collected 2-4(5) h following the concentrate meal (*Nordlund & Garrett* 1994, *Nordlund et al.* 1995). pH-measurements on rumen fluid sampled by the present procedure that excluded saliva contamination did not point out the cows in the I group. In cows fed concentrates only 2 times daily, low pH values could be expected in many cows. Only few cows were examined at an early stage (Table 2) and the pH could have been normalised because of sodium bicarbonate from saliva or because of reduced fermentation activity by the rumen bacteria.

The concentration of protozoa in rumen contents, especially small entodiniomorphs (oligotrichs), generally increases with the addition of concentrates to roughage diets (*Franzolin & Dehority* 1996, *Dehority & Orpin* 1997). A fall in the pH of rumen contents is generally accompanied by a decrease in the protozoal concentrations. The large entodiniomorphs are the most sensitive of the protozoal species, whereas the trichostomatids (holotrichs) are the most tolerant to low pH. Nearly all protozoa die when pH declines to 5.0 (*Belknap & Navarre* 2000). When subacute rumen acidosis was induced experimentally during stepwise adaptation to high concentrate diets after parturition, the population of large entodiniomorphs was successively decimated, while the trichostomatids survived (*Steen et al.,* to be published). *Goad et al.* (1998) concluded that a decline in

the concentration of ciliated protozoa may be the only microbial indicator of subacute ruminal acidosis. Underfeeding will also cause a reduction in protozoal concentration (*Dirksen* 1990). It seems that several other factors are involved in defaunation. These factors could include rate of feed consumption, rate of passage, and salivary production (*Franzolin & Dehority* 1996).

The methylene blue reduction time is an indirect measure of the redox potential and bacterial activity of rumen fluid (*Dirksen* 1969). By standardised reading after 3 and 6 min and keeping the probes in a thermos, the test was relatively fast and simple to perform. When feeding a mixed ration of roughage and concentrates methylene blue will be decolourised in less than 3 min. *Dirksen* (1969) showed that in experimentally induced rumen acidosis the reduction time increased moderately the first 4-5 h after adding 4 kg sugar through a rumen fistula, and when acidity dropped to values below pH 5.1 a long delay could be observed. Reduced microbial activity in the rumen after a prolonged period with reduced appetite will also cause an increase in reduction time (*Dirksen* 1969).

The sedimentation time was difficult to assess with the method used, and rejected as a diagnostic parameter.

From the above discussion it seems that reduced protozoal concentrations and increased methylene blue reduction time can have 2 main causes, rumen acidosis or starvation. In the present material there was a reduction in the concentrations of both large and small protozoan in 60% to 80% of samples from the patients with deviating rumen fluid parameters, the I group, the K+I group and L-K subgroup (Table 3). It was not possible to distinguish between large entodionimorphs and trichostomatids with the magnifying lens. Many of these cases could have been misclassified based on the reduced protozoal numbers, because 54% of the cows in the I group and 38% in the K+I group had reduced appetite ≥3 days (Table 2). The reduced numbers of protozoa could have been a result of the reduced feed intake. Some of the cases that were classified into the K+I group based on increased methylene blue reduction time (28%+11%, Table 3) were probably primary ketosis cases (K) of longer duration or with late treatment. Because of the workload of the practising veterinarian, time did not permit examination of a control patient for each clinical case. Measurements on "normal" cows from the same farm as close as possible in stage of lactation could have removed some of the diurnal, farm and diet effects from the data analysis.

In early lactation energy loss due to milk production is greater than the energy intake. In this metabolic state the cow mobilise body fat as free fatty acids. Some of these free fatty acids are incorporated into milk fat, the remainder is used as energy substrates through oxidation in the citric acid cycle and through production of ketone bodies in the liver. Ketone bodies are an important energy source in many tissues and may have a glucose-sparing effect (*Herdt* 1988). Moderate hyperketonaemia in early lactation is normal (*Halse et al.* 1983). Blood glucose concentrations are reduced in primary ketosis, in ketosis secondary to other diseases blood glucose concentrations are only moderately reduced or often above reference range (*Radostits et al.* 2000 pp.1452-1462). Reliable cowside equipment for blood glucose measurements was not available during the present study, but could have been a useful additional diagnostic parameter.

The cows in the relatively large group with no specific diagnoses (N) resembled the K group, except for a negative or slightly positive ketotest score. Without careful interpretation of the milk ketotest, many cases would have been reported as primary ketosis. Thorough clinical

examination together with additional rumen fluid examination and clinical pathology did not uncover the reason(s) for the reduced appetite in these cows.

The conclusion from the present study was that established methods in clinical examination, clinical pathology and rumen fluid analyses that were feasible for the practitioner did not lead to clear-cut diagnoses in many of the cows when used on cows with reduced appetite in the most critical period for developing ketosis postpartum. The methods used did not uncover the reason(s) for the reduced appetite in 14% of the cows in the material. More research is needed to develop more powerful diagnostic methods, especially in the field of rumen acidosis.

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Sammendrag

Feltstudie av kyr med nedsatt matlyst i tidlig laktasjon: Kliniske undersøkelser, blod og vomsaftanalyser.

Undersøkelsen omfattet 125 kyr med nedsatt matlyst og med kliniske tegn tolket av eier som tegn på ketose 6 til 75 dager etter kalving. De fleste av kyrne fikk tildelt kraftfôr etter behov 2 eller 3 ganger per dag. Med tanke på behandling og profylakse ble kyrne på grunnlag av klinisk undersøkelse, ketotestutslag, nedsatt aktivitet og konsentrasjon av protozoer, forøket reduktasetest og forøkede leverparametre delt i 5 diagnosegrupper: ketose (n=32), indigestion (n=26), kombinert ketose og indigestion (n=29), leverpåkjenning i tillegg til ketose, indigestion eller begge (n=15), og ingen spesifikk diagnose (n=17). Tre kyr med kvast og 3 med løpedislokasjon ble ikke gruppert. Nonparametriske metoder ble benyttet ved sammenligninger mellom gruppene. Aspartat aminotransferase, glutamat dehydrogenase, gamma-glutamyl transferase og totalbilirubin var forhøyd i gruppen med leverpåkjenning. Frie fettsyrer var signifikant forøket hos kyr med ketose sammenlignet med kyr med indigestion. Infusorieaktivitet og antall av store og små infusorier var nedsatt og reduktasetest forøket i gruppen med indigestion. Det ble ikke målt pH-forskjeller mellom kyr med og uten indigestion. Langvarig nedsatt matlyst før undersøkelse kan ha influert på grupperingen. Ved ukritisk bruk av ketotest ville mange av kyrne med tilleggsdiagnoser blitt rapportert som primær ketose. Grundig klinisk undersøkelse supplert med praktisk gjennomførbare vomsaftundersøkelser og økonomisk forsvarlig klinisk biokjemiske analyser kunne ikke avdekke årsaken(e) til den nedsatte matlysten hos 14% av kyrne i materialet. Bedre diagnostiske hjelpemidler, særlig med tanke på vomacidose, er nødvendig.

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