DIARRHOEA OF THE YOUNG CALF: AN UPDATE

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1. INTRODUCTION

Neonatal calf diarrhoea is a multifactoral disease which despite decades of research on the topic remains the most common cause of death in young calves. Even though major risk factors have long been identified, numbers of calf losses due to diarrhoea are not declining. Surveillance data of the National Animal Health Monitoring System showed that "there was essentially no change from 1995 to 2001 in either the overall mortality of preweaned calves or in mortality by specific causes. Scours and diarrhea still accounted for the largest percentage of deaths in preweaned calves, followed by respiratory problems". The overall mortality in preweaned heifer calves was 10.5% of heifer calves born alive in 2002, and diarrhoea accounted for 62.1% of calf losses (USDA, 2002a). In surveillances on dairies in Southern Germany incidence of diarrhoea in young calves was 15.4%, and 28.4% when data were based on questionnaires filled in by the farmers (Katikaridis, 2000; Biewer, 2001), and 47.8% when calves were examined by a veterinarian daily up to three weeks of age (Girnus, 2004).

This review is indented to highlight some major points of research or discussion during the last years involving this disease complex.

2. INFECTIOUS AGENTS

Though diarrhoea of the neonatal calf was referred to as "colibacillosis" up to the second half of the last century, there is widespread agreement now that enterotoxic *E. coli* are of minor importance in the complex of neonatal calf diarrhoea on most farms. Studies in different European countries revealed rotavirus and cryptosporidia as major pathogens to be found in faecal samples of diarrhoeic calves, and a low prevalence of coronavirus and enterotoxic *E. coli* (Fagan *et al.* 1995; Fuente *et al.* 1998; Luginbühl *et al.* 2005).

Since *Cryptosporidium parvum* is highly prevalent in the cattle population it has come into the focus of research during the last decade. On US dairy farms a prevalence of 90% is reported, with almost half of the 1 to 3 weeks old calves shedding oocysts (NAHMS, 1993). In diarrhoeic calves this parasite was detected in 43.8%, 71.9%, 63.2%, and 6.9% of faecal samples, respectively, in the

first four weeks of life (Fuente *et al.* 1999). Mixed infections with other enteropathogens are common in diarrhoeic calves (Joachim *et al.* 2003). Because of the large numbers of oocysts excreted even by asymptomatic calves together with a strong resistance to environmental conditions and disinfectants, the emphasis of prevention of cryptosporidiosis lies on good management practices. Halofuginone lactate so far is the only agent that has been shown to reduce oocyst shedding under field conditions, if administered prophylactically, and is licensed for use in calves in Europe. In light of the results of recent studies the question whether the prophylactic use of halofuginone lactate in the first 7 days of life is able to reduce the incidence of diarrhoea (Lefay *et al.* 2001), or only to delay the onset of diarrhoea in calves (Jarvie *et al.* 2005) remains controversial.

3. MANAGEMENT RISK FACTORS AND PREVENTION

In principle, management factors have to boost the immunity of the calf and to diminish the risk of infection in order to reduce the risk for diarrhoea in the newborn calf.

Many factors can affect passive transfer of immunoglobulins, especially the time of first colostrum intake as well as quality and amount of ingested colostrum. Since local immunity is crucial especially for prevention of infections with enteropathogens the time from birth to first feeding of colostrum is correlated with the incidence of diarrhoea (Katikaridis, 2000; Biewer, 2001). In general, the quality of colostrum increases with the lactation number of the dam which explains the higher risk of disease in calves born to first-lactation cows (Svensson *et al.* 2003). Incidence of diarrhoea can be further reduced by feeding a half-litre surplus colostrum of the dam twice a day with the conventional feeding up to the 10^{th} day of life (Gutzwiller, 2002). It is recommended to feed 2 litres of colostrum as soon after birth as possible (within three hours) by nipple bottle or bucket, and to feed a total of four litres within the first 6 to 12 hours of life. It is not recommended to leave calves to suck the dam after birth, because it is difficult to assure adequate colostrum intake, and the risk of infection increases. Force-feeding of colostrum by tube feeder has evoked controversial discussion in the last years. Studies have shown that force feeding of 4 litres of colostrum soon after birth is a simple and reliable method to achieve concentrations of serum IgG in calves that are well above the desired minimal value of 10 g/l (Hungerford *et al.* 1999; Heyn, 2002). On the other hand there are other methods to reach this target, and in countries like Germany force feeding of animals without medical indication is prohibited by the law for animal welfare (Erhard *et al.* 2005). We compared force feeding of colostrum to conventional administration of colostrum on 15 dairies in Southern Germany. IgG concentrations in serum of force-fed calves was 19.04 g/l (n = 124) vs. 17.16 g/l in conventionally fed calves (n = 134). There was no significant difference in occurrence of neonatal diseases between groups. Expenses for veterinary care were slightly but not significantly higher in force-fed calves (Ebert, dissertation in preparation).

The key to reducing the risk of infection is cleanliness of the calving area, calf housing, and feeding equipment. So it is not amazing that the use of individual maternity areas and regular removal of bedding from the maternity area between calvings reduces the risk of neonatal diarrhoea in the calf (Curtis *et al.* 1988; Frank & Kaneene, 1993). Calves should be housed individually in the first weeks of life, preferable in fibreglass hutches, where the incidence of diarrhoea is lower than in individual calf pens (Biewer, 2001; Girnus, 2004). The advantage of individual hutches is that high-pressure cleaning can easily be performed, and hutches can be moved to new areas between calves. Unfortunately acceptance of this housing method is still not as widespread in Germany and probably in other European countries as in the US dairy industry, where in 2002 on almost half of the dairy operations heifer calves were housed in hutches (USDA, 2002b). For two areas in Southern Germany the respective percentages were 9.9% and 11.6% at the end of the last century (Katikaridis, 2000; Biewer, 2001). The study of Girnus (2004) has shown that the benefits of housing calves in hutches are easily lost if more than one calf is housed in one hutch.

4. THE ROLE OF D-LACTIC ACID IN NEONATAL CALF DIARRHOEA

The pathogenetic mechanisms of acidosis in diarrheic calves in general are intestinal losses of bicarbonate and accumulation of organic acids. In case of dehydration, reduction of renal perfusion and hence of excretion of hydrogen ions can worsen the situation. Formation of L-lactate from anaerobic glycolysis following tissue hypoperfusion has long been considered to be the major cause of high anion gap acidosis in calves with neonatal diarrhoea.

In the last decade D-lactic acid has been identified as a considerable factor in pathogenesis of metabolic acidosis in the diarrhoeic calf (Grude *et al.* 1999; Omole *et al.* 2001; Ewaschuk *et al.* 2004; Lorenz, 2004a). When a reference range was established using clinically healthy bucket-fed Simmental calves up to three weeks old from dairy farms in southern Germany, the upper limit of the 95% percentile was found to be 3.96 mmol/l (Lorenz *et al.* 2003). Since mammals are not able to synthesise appreciable amounts of D-lactate this value was somehow astonishingly high, which was attributed to the feeding regime. Figure 1 shows that D-lactataemia is a common occurrence in calves with diarrhoea, and that there is a good correlation (r = .685) between D-lactate levels and base excess values. Thus, even if most clinical signs of acidosis are attributable to D-lactate (Lorenz, 2004b; Lorenz *et al.* 2005), this correlation allows estimation of the degree of acidosis on the basis of clinical signs. The current understanding of the influence of D-lactate on clinical signs in diarrhoeic calves is dealt with by Naylor *et al.* elsewhere in this issue.

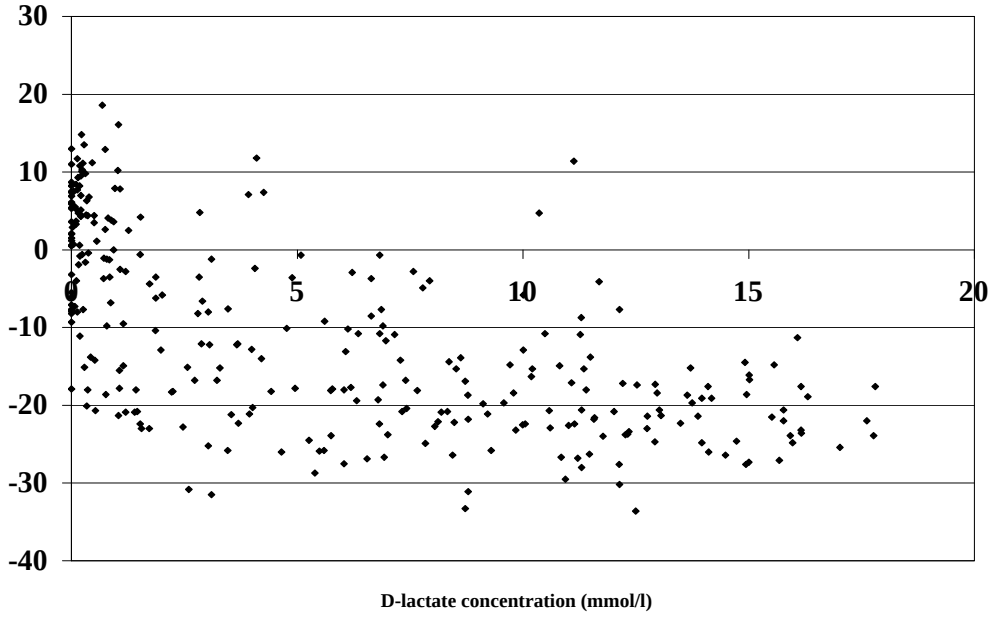

Figure 1. **Base excess and serum D-lactate concentrations of 300 hospitalised calves with neonatal diarrhoea**

We performed a prospective study to elucidate whether amounts of bicarbonate needed for correction of acidosis and normalisation of clinical signs is influenced by blood D-lactate concentrations in calves with diarrhoea (Lorenz & Vogt, 2006). In seventy-three calves up to three weeks old with acute diarrhoea and base excess values below -10 mmol/l correction of acidosis was carried out within 3.5 h by IV administration of an amount of sodium bicarbonate which was calculated using the formula: HCO3- (mmol) = body mass (kg) * base deficit (mmol/l) * 0.6 (l/kg). Clinical signs, venous base excess, and plasma D-lactate concentrations were monitored immediately following admission, following correction of acidosis at 4 h, and 24 h after admission. Figure 2 shows the base excess and plasma D-lactate concentrations throughout the study.

Metabolic acidosis was not corrected in more than half of the calves (n = 43) by the calculated amount of bicarbonate, and the risk of failure to correct acidosis increases with D-lactate concentrations. The study shows that calves with elevated D-lactate concentrations do not need additional specific therapy, as D-lactate concentrations regularly fall following correction of acidosis and restitution of body fluid volume. However, calves with distinct changes in posture and demeanour need higher doses of bicarbonate than calculated with the factor of 0.6 in the formula mentioned above probably because of hyper D-lactataemia.

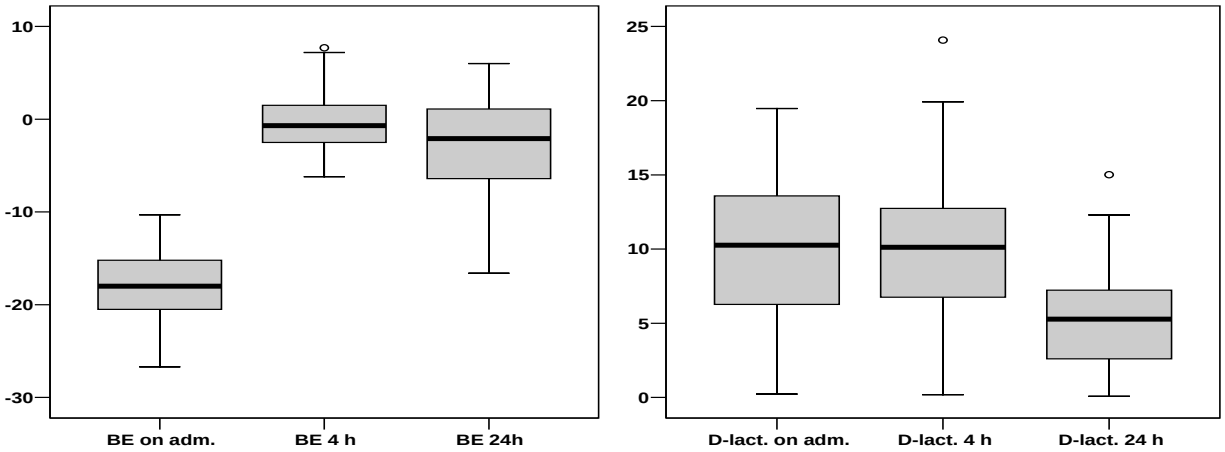

Figure 2. **Course of base excess (mmol/l) and serum D-lactate concentrations in 73 acidotic calves with diarrhoea after correction of acidosis within 3.5 hours**

The reasons for the phenomenon of decreasing D-lactate values after conventional treatment of dehydration and acidosis is not yet clear, but an impairment of metabolism of D-lactate by acidosis can be assumed. That was the reason for another study which compared two different regimes (slow vs. fast) of treating metabolic acidosis in diarrhoeic calves (Haase, 2006). Following the formula "bodyweight * base deficit * 0.6" the calves of both groups were given the respective amount of sodium bicarbonate. The animals belonging to group 1 received the calculated amount of bicarbonate within one hour as a 4.2 % solution, whereupon 0.9% sodium chloride was given in the following 23 hours in volumes corresponding to the estimated fluid loss. The animals of group 2 were treated with a constant drip infusion over 24 hours which contained both the calculated amount of bicarbonate as a 1.4% solution and 0.9% sodium chloride which was estimated on the basis of clinical dehydration. Calves in which acidosis was corrected rapidly showed significantly lower pH, base excesses and HCO_3^--concentrations on the second day compared to the calves in which acidosis was corrected slowly; but these variations did not appear clinically. No differences between the two groups could be diagnosed on the basis of the general condition. Likewise, no difference between groups could be detected in the milk intake after the end of fluid therapy. These results do not support the assumption that D-lactate levels would decline faster in calves when acidosis was corrected rapidly, as no significant differences of the D-lactate-values could be diagnosed between the two groups on the second day.

Figure 3 shows that elevation of D-lactate concentration on admission has no negative effect on prognosis in diarrhoeic calves.

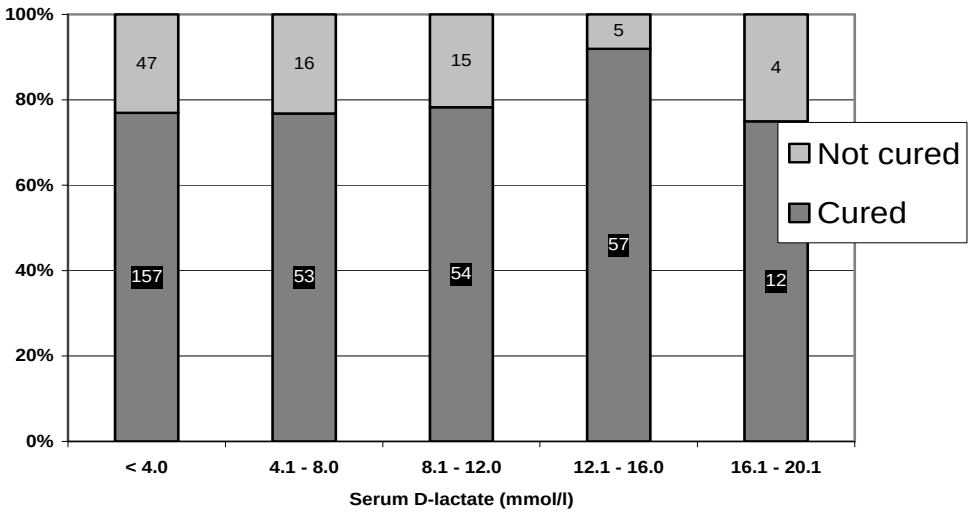

Figure 3. **Serum D-lactate concentration on admission and outcome in 420 hospitalised calves with neonatal diarrhoea**

5. FEEDING OF THE DIARRHOEIC CALF

Traditionally continued feeding of milk to diarrhoeic calves was thought to aggravate diarrhoea because malabsorbtion would provide substrate to the intestinal flora for fermentation and thus lead to osmotic diarrhoea. Consequently withdrawal of milk during the first days of diarrhoea was recommended. In later studies secretory mechanisms were found to play the major role even in diarrhoea caused by viruses or cryptosporidia (Doll, 1994), and evidence was produced that diarrhoeic calves have sufficient capacity to digest milk (Heath *et al.* 1989; Garthwaite *et al.* 1994). Furthermore the ingestion of adequate amounts of milk showed no negative effect on duration of diarrhoea (Deischl, 1992; Niemeyer, 1992; Doll, 1994). Hinderer *et al.* (1999) evaluated two different feeding regimes in young calves with diarrhoea. The patients were given whole milk *ad libitum* 3 times a day (experimental group) or were offered whole milk at a rate of 13% their body mass per day divided into 3 meals (control group, 30 calves per group). Both groups were also offered an oral rehydration solution at 9.5% of body mass per day, also divided into 3 portions. There was no difference in duration of illness between groups. The daily gain in body mass averaged 0.607 ± 0.452 and 0.230 ± 0.231 kg, respectively. This study shows that diarrhoeic calves can achieve daily weight gains similar to those of healthy calves without negative effects on duration of illness. On the basis of these findings we recommend to supply calves (healthy as well as diarrhoeic) with an amount of milk that equals about 12% of their body weight. In case of diarrhoea oral rehydration solutions must be provided additionally.

6. USE OF ANTIBIOTICS IN CALF DIARRHOEA

Antibiotics are still frequently used for the treatment of uncomplicated and undifferentiated diarrhoea in calves. Since indiscriminate use of antibiotics promotes the selection, and subsequent proliferation, of antibiotic-resistant strains of bacteria, this general practice has been scrutinised. The benefit of antimicrobial treatment in neonatal calf diarrhoea has recently been reviewed (Constable, 2004). Routine use of oral and injectable antibiotics cannot be recommended in calves without systemic illness. In calves with diarrhoea and severe systemic involvement antimicrobial therapy must be pondered carefully as intercurrent disease is not uncommon and the risk of bacteremia or septicaemia is increased. High risk calves in this respect are under 5 days of age, have failure of passive transfer, are recumbent, and have no sucking reflex (Lofstedt *et al.* 1999). In this indication parenteral administration of broad-spectrum β-lactam antimicrobials (ceftiofur,

amoxicillin or ampicillin), potentiated sulfonamides, or fluoroquinolones (where permitted) is recommended (Constable, 2004).

7. SUMMARY

Neonatal calf diarrhoea is a multifactoral disease which despite decades of research on the topic remains the most common cause of death in young calves. An infectious aetiology can be assumed in operations where diarrhoea is a common occurrence. The most frequently found enteropathogens in faecal samples are rotaviruses and cryptosporidia. To reduce the risk of neonatal diarrhoea management factors have to boost the immunity of the calf and to diminish the risk of infection:

- provision of 2 litres of colostrum within the first 3 hours of life; provision of a total of 4 litres within the first 6 to 12 hours,
- individual maternity areas and regular removal of bedding from the maternity area between calvings,
- immediate separation from the dam and housing of calves in individual hutches; hutches should be high-pressure cleaned and moved to new areas between calves,
- cleanliness of feeding equipment,
- feeding of adequate amounts of milk to both healthy and diarrhoeic calves.

8. KEY WORDS

Diarrhoea, calves, risk factors, D-lactic acidosis, feeding regime.

9. RESUME

La diarrhée chez le veau nouveau-né est une maladie multifactorielle qui occasionne des pertes importantes. L'étiologie est généralement d'origine infectieuse quand les diarrhées sont fréquentes dans un troupeau. Les agents pathogènes détectés le plus souvent sont de nature virale (Rotavirus) et des cryptosporidies. La genèse d'épizooties dans un troupeau est toujours associée à des erreurs de la gestion. Les facteurs diminuant le risque de diarrhée sont ceux qui renforcent le système immunitaire du veau et qui réduisent la concentration de germes infectieux :

- distribution de deux litres de colostrum dans les 3 heures suivant la naissance, et de 4 litres de colostrum entre 6 et 12 heures après la naissance,
- vêlage dans un box séparé, nettoyage régulier du box,
- séparation du nouveau-né et de la mère dès la naissance, isolement du nouveau-né dans un igloo qui a été nettoyé à l'aide d'une nettoyeuse de forte pression, déplacement de l'igloo sur un nouvel emplacement avant d'y mettre un nouveau veau,
- nettoyage régulier et en profondeur de l'appareillage servant à l'abreuvage entre les repas,
- maintien de l'abreuvage en lait en quantité appropriée à ses besoins habituels, bien que le veau soit diarrhéique.

10. MOTS CLES

Diarrhée, veaux, facteurs de risque, D-Lactatémie, diète.

11. ZUSAMMENFASSUNG

Durchfall bei neugeborenen Kälbern ist eine multifaktorielle Erkrankung, die weltweit erhebliche Kälberverluste verursacht. Es ist davon auszugehen, dass den meisten bestandsweise gehäuft auftretenden Durchfallerkrankungen junger Kälber eine infektiöse Ätiologie zugrunde liegt. Die hierbei am häufigsten nachgewiesenen Erreger sind Rotaviren und Kryptosporidien. Immer jedoch sind auch Fehler im Management an der Entstehung eines Bestandsproblemes Neugeborenendiarrhoe beteiligt. Die Faktoren, die das Auftreten von Durchfall vermeiden helfen, sind solche, die die Abwehrlage des Kalbes stärken und den Keimdruck vermindern:

- Versorgung des Kalbes mit zwei Litern Erstkolostrum innerhalb der ersten drei Lebensstunden, insgesamt Verabreichung von vier Litern Kolostrum innerhalb der ersten sechs bis zwölf Lebensstunden,

- Abkalbung in einer dafür vorgesehenen Abkalbebox, die regelmäßig gereinigt wird,

- Unmittelbar nach der Geburt: Verbringen des Kalbes in ein mittels Hochdruckreiniger gereinigtes Iglu, dessen Standplatz zwischen den Belegungen geändert wird,

- Gründliche Reinigung der in ausreichender Menge vorhandenen Tränkegerätschaften zwischen den Mahlzeiten,

- Tränkung des Kalbes mit bedarfsdeckenden Volumina an Milch, auch wenn Durchfall auftritt.

12. SCHLÜSSELWÖRTER

Durchfall, Kälber, Risikofaktoren, D-Laktatazidose, Diätfrage.

13. RESÚMEN

La diarrea del ternero recién nacido es una enfermedad multifactorial, la cual mundialmente produce grandes pérdidas. Sabemos que en la mayoría de los casos, en los cuales se observa una diarrea de los terneros jóvenes, esta tiene una etiología infecciósa. Responsible son generalmente rotavirus y criptosporidios. También errores en el manejo son frecuentes. Factores que evitan la diarrea del ternero son aquellos que aumentan las defensas y reducen la presión por parte de los gérmenes:

- administración des dos litros de calostro durante las primeras tres horas de vida,

- administración total de cuatro litros de calostro durante las primeras seis a doce horas de vida,
- partos en boxes para partos, los cuales se pueden limpiar adecuada- y regularmente,

- transpasar a los terneros inmediatamente despues de nacidos a un iglu que se puede limpiar con vapor a alta presión y el cual se puede cambiar de lugar entre las distintas ocupaciones nuevas,

- limpieza prolija de los utensilios de alimentación (disponibles en cantidad suficiente) entre las comidas,

- alimentar los terneros con cantidades adecuadas de leche en caso que surgan diarreas.

14. PALABRAS CLAVES

Diarrea del ternero, errores en el manejo, D-lactacidemia-alimentación.

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