Treatment of cows with parturient paresis using intravenous calcium and oral sodium phosphate

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Summary
The goal of this study was to investigate whether intravenous infusion of 1000 ml 40% calcium borogluconate combined with the oral administration of 500 g sodium phosphate leads to a better cure rate and longer-lasting normocalcaemia and normophosphataemia than standard intravenous treatment with 500 ml calcium borogluconate in cows with parturient paresis. Forty recumbent cows with hypocalcaemia and hypophosphataemia were alternately allocated to group A or B. Cows of both groups were treated intravenously with 500 ml 40% calcium borogluconate, and cows of group B additionally received another 500 ml calcium borogluconate via slow intravenous infusion and 500 g sodium phosphate administered via an orogastric tube. Thirty-two cows stood within 8 hours after the start of treatment and 8 did not; of the 32 cows that stood, 18 belonged to group A and 14 to group B (90% of group A vs. 70% of group B; P = 0.23). Seven cows relapsed; of these and the 8 that did not respond to initial treatment, 10 stood after two standard intravenous treatments. Downer cow syndrome occurred in 5 cows, 3 of which recovered after aggressive therapy. The overall cure rate did not differ significantly between groups A and B. Twelve (60%) cows of group A and 14 (70%) cows of group B were cured after a single treatment and of the remaining 14, 11 were cured after two or more treatments. Two downer cows were euthanized and one other died of heart failure during treatment. Serum calcium concentrations during the first eight hours after the start of treatment were significantly higher in group B than in group A, and oral sodium phosphate caused a significant and lasting increase in inorganic phosphate. More cows of group B than group A were cured after a single treatment (P > 0.05). These findings, although not statistically significant, are promising and should be verified using a larger number of cows.

Keywords: cattle, parturient paresis, treatment, calcium infusion, oral sodium phosphate

Behandlung der Gebärparese von Kühen mit Kalzium intravenös und Natriumphosphat per os
Introduction

The standard treatment for parturient paresis is the intravenous administration of a calcium solution (Radosits et al., 2007). Forty-seven (Salis, 2002; Braun et al., 2004a) to 76% (Malz and Meier, 1992) of affected cows respond to a single treatment. Some cows relapse and require multiple treatments and some do not respond to treatment and are euthanized. For more than 10 years, we have been conducting studies aimed at improving the success rate of the treatment for parturient paresis using different protocols including slow intravenous infusion of calcium over several hours (Salis, 2002; Braun et al., 2004a, 2004b), doubling of the calcium dose (Jehle, 2004; Braun et al., 2006), oral (Dumelin, 2005; Braun et al., 2007) or intravenous administration of sodium phosphate in addition to intravenous administration of calcium (Zulliger, 2008; Braun et al., 2009) and intravenous and oral calcium and phosphorus (Blatter, 2011; Braun et al., 2012). Unfortunately, none of those treatments improved the success rate significantly; all treatments failed to produce a rapid increase in serum calcium and phosphorus concentrations that persisted for 48 to 76 hours. Doubling of the standard intravenous calcium dose caused hypercalcæmia or normocalcaemia that lasted for several hours (Braun et al., 2006), and the oral administration of 300 g monosodium phosphate resulted in transient normalisation of the inorganic phosphate concentration (Braun et al., 2007a). In an attempt to improve on those results, the same two treatments were combined in the present study and the amount of phosphate was increased to 500 g. The goal was to compare intravenous administration of 1000 ml of a 40% calcium borogluconate solution combined with oral administration of 300 g sodium phosphate with the standard single administration of 500 ml of the same calcium solution for the treatment of parturient paresis and generation of sustained normocalcaemia and normophosphataemia.

Animals, Material and Methods

Cows

Forty cows that had parturient paresis within 48 hours after parturition (median, 15.3 hours) were used. The cows originated from 33 herds serviced by the ambulatory clinic at the Department of Farm Animals, University of Zurich, were between 5 and 10 years of age (mean ± sd = 7.4 ± 1.7 years) and belonged to the Swiss Fleckvieh (n=25), Holstein Friesian (n=9) and Brown Swiss (n=6) breeds. The lactation numbers ranged from 1 to 8 (mean, 5.0 ± 1.6) and the production in the previous lactations ranged from 5'600 to 14'000 kg (mean, 8'448 ± 1'661 kg). The cows were treated 1 to 8 hours (median, 3 hours) after they had become recumbent; this time interval did not differ between treatment groups.

Clinical examination and diagnosis

All cows underwent a thorough clinical examination as described by Rosenberger (1990). Parturient paresis was diagnosed when cows became recumbent within 24 hours after normal parturition and had hypocalcaemia and hypophosphataemia.

Treatment

The cows were divided randomly into 2 groups of 20 cows each. All cows (groups A and B) received 500 ml of 40% calcium borogluconate supplemented with 6% magnesium hypophosphite (15.65 g calcium gluconate and borogluconate, 9.85 g magnesium hypophosphite: Calcamyl-40MP, Grüeb, Bern) via an indwelling jugular vein catheter. During the infusion, which was given over ten minutes, the heart rate and rhythm were monitored continuously. Cows of group B also received another 500 ml of the same calcium borogluconate solution added to 10 litres of a solution of 90 g sodium chloride and 500 g glucose and administered via a slow intravenous drip over a six-hour period (1.7 l/hour). Immediately after the start of the infusion, a solution of 500 g monosodium phosphate (NaH2PO4; Hänseler AG, Herisau) in 1 liter of water was administered into the rumen via an orogastric tube. The total amount of administered...
metabolisable electrolytes were 15.7 g calcium and 2.8 g magnesium per cow in group A and 31.4 g calcium, 5.6 g magnesium and 100 g phosphorus per cow in group B. Before the start of the treatment, all cows were turned onto their other side and the hind limbs were hobbled. After the initial rapid calcium infusion, heart rate, respiratory rate, rectal temperature, superficial body temperature, rumen motility, appetite and defecation were monitored every hour for 8 hours. Attempts to rise, the ability to stand and the progression of the disease were also noted. Cows that did not attempt to rise were encouraged to do so every hour by loud voice commands and manual slaps on the croup; when this was unsuccessful, a maximum of two gentle strikes with a cattle prod were used. Cows that remained recumbent were turned again after 4 hours.

Treatment of cows that did not respond or relapsed
Cows that did not stand within 8 hours after the beginning of the treatment or became recumbent again after successful initial treatment were given another 500 ml of 40% calcium borogluconate and 2 g ketoprofen (Rifen 10%, Streuli, Uznach), both administered intravenously.

Blood examination and urinalysis
Blood samples were collected from all the cows into vacuum tubes (Vacuette® Serum Sep. Clot Activator, 5 ml, Greiner bio-one GmbH, Kremsmünster, Austria) immediately before treatment and 10, 20, 40, 60, 90 minutes and 2, 3, 4, 5, 6, 7, 8, 24, 48 and 72 hours after the beginning of treatment for the determination of the concentrations of total and ionised calcium, inorganic phosphate and magnesium. The concentrations of calcium, inorganic phosphate and magnesium were determined at 37°C with a Cobas Integra 700 analyser (Roche Diagnostics, Rotkreuz), using Roche reagents under conditions defined by the International Federation of Clinical Chemists. The concentration of ionised calcium was measured using potentiometry (Rapidpoint 400, Siemens Healthcare, Zürich). Urine samples were tested for ketone bodies before the start of treatment.

Statistical analysis
Data were analysed using Stata (StataCorp LP, College Station, Texas, USA). Frequencies, means and standard deviations were calculated. Normal distribution of continuous data was examined using the Wilk-Shapiro test. Normal data were reported as mean ± standard deviation and non-normal data as median and range. Differences between continuous data were analysed using a two-sided paired or unpaired t-test and differences between categorical data were analysed using a chi-square test for association. P < 0.05 was considered significant.

Results

Clinical findings before treatment
Cows of group A were anorexic (n = 15) or had a reduced appetite (n = 5) and in group B all cows were anorexic (Chi² test, P < 0.05). Other clinical variables did not differ between the groups and therefore are presented for both groups combined. Thirteen cows had a normal demeanour, 15 were obtunded, 10 were somnolent and 2 were comatose. The latter 2 cows were in lateral recumbency and the remaining 38 were in sternal recumbency. The heart rate ranged from 40 to 120 beats per min (median, 68 bpm) and the rectal temperature ranged from 36.4°C to 39.4°C (38.1 ± 0.73°C). The superficial body temperature was lower than normal in 34 cows. Rumen motility was absent in 35 cows and reduced in the remaining 5. Intestinal motility was absent in 27 cows and 6 cows passed small amounts of dry faeces. Low levels of ketone bodies were detected in the urine of one cow. The clinical findings have been described in detail (Grob, 2015).

Clinical findings after treatment and response to treatment
The course of the disease did not differ significantly between the two groups. The demeanour normalised after the start of treatment in all but one cow and the mean rectal temperature returned to the reference interval within 3 hours. Appetite returned to normal in 30 cows within 7 hours and all but 2 cows had rumen motility 8 hours after the start of treatment. Thirty-four cows defecated at least once during the eight-hour observation period.

Thirty-two cows stood within 8 hours after the start of treatment and 8 did not. Eighteen of the responding cows belonged to group B and 14 belonged to group A (90% versus 70%, P = 0.23). Seven of the initially responding cows relapsed (three from group A and four from group B). Of the 7 cows that relapsed and the 8 that did not respond to the initial treatment, 10 stood after a second calcium infusion. The other 5 became downer cows, and 3 of these recovered. The overall recovery rate did not differ between groups (group A, 18/20 [90%]; group B 19/20 [95%]). Twelve cows (60%) of group A and 14 cows (70%) of group B responded to the initial treatment (Tab. 1). Of the remaining 14 cows, 11 (group A, 6; group B, 5) stood after two or more treatments. Two cows (group A) failed to respond to treatment and were euthanized and one cow of group B died during a follow-up treatment of acute heart failure.
Treatment of cows with parturient paresis using intravenous calcium and oral sodium phosphate

U. Braun, D. Grob, M. Hässig

Serum concentrations of electrolytes before treatment
The serum concentrations of electrolytes before treatment did not differ between groups. All cows had hypocalcaemia with total calcium less than 2.0 mmol/l (0.54 to 1.97 mmol/l; 1.07 ± 0.39 mmol/l) and hypophosphataemia with inorganic phosphate less than 1.30 mmol/l (0.16 to 1.18 mmol/l; 0.50 ± 0.26 mmol/l). Ionised calcium ranged from 0.20 to 0.98 mmol/l (0.51 ± 0.22 mmol/l) and magnesium, which was decreased in one cow, ranged from 0.59 to 1.77 mmol/l (1.24 ± 0.25 mmol/l). Total calcium and ionised calcium concentrations (r = 0.97, P < 0.01) and total calcium and inorganic phosphate concentrations (r = 0.46, P < 0.01) were significantly correlated.

Serum concentrations of electrolytes after the start of treatment
The profiles of total calcium differed significantly between the groups (Fig. 1). Total calcium increased sharply and significantly after the start of treatment, and within 10 minutes, the cows of both groups had transient hypercalcaemia. The peak at 10 minutes was followed by a gradual and slow decrease in calcium concentration. Cows of group A were hypercalcaemic for 2 hours, normocalcaemic between 2 and 7 hours and hypocalcaemic at 24 hours. Cows of group B were hypocalcaemic for 7 hours, normocalcaemic at 8 hours and hypocalcaemic from 24 to 72 hours. The calcium concentration was significantly lower (P < 0.05) in group B than in group A from 24 to 72 hours. The number of cows with hypocalcaemia at eight, 24, 48 and 72 hours differed significantly between the two groups (Tab. 2). At 8 hours, there were more hypocalcaemic cows in group A (7 vs. 1, P < 0.05) and at 72 hours, there were more hypocalcaemic cows in group B (3 vs. 12, P < 0.01). The concentrations of ionised calcium were analogous to those of total calcium (Fig. 2).

As expected, inorganic phosphate was not affected by the treatment in group A and remained below the reference interval for 24 hours (Fig. 3). Cows of group B had normophosphataemia from 90 minutes until 72 hours. There were significantly more hypophosphataemic cows at eight and 24 hours in group A than in group B (8 hours, 14 vs. 5; 24 hours, 14 vs. 4; P < 0.05) (Tab. 2).

The magnesium profiles were similar in both groups (Fig. 4), but the concentrations were significantly higher in group B than in group A from 40 minutes to 7 hours (P < 0.05). The concentration increased sharply and significantly (P < 0.01) within 10 minutes, after which time it decreased gradually.

Table 1: Response to treatment in 40 cows with parturient paresis.

<table>
<thead>
<tr>
<th>Response to treatment</th>
<th>Group A</th>
<th>Group B</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cured after single treatment</td>
<td>12 (60.0%)</td>
<td>14 (70.0%)</td>
<td>26 (65.0%)</td>
</tr>
<tr>
<td>Cured after more than one treatment</td>
<td>6 (30.0%)</td>
<td>5 (25.0%)</td>
<td>11 (27.5%)</td>
</tr>
<tr>
<td>Not cured</td>
<td>2 (10.0%)</td>
<td>1 (5.0%)*</td>
<td>3 (7.5%)</td>
</tr>
</tbody>
</table>

* Heart failure and death during relapse treatment with calcium borogluconate

Figure 1: Serum concentrations (means ± standard deviations) of total calcium after intravenous infusion of calcium borogluconate (group A) and after intravenous infusion of calcium borogluconate combined with oral administration of sodium phosphate (group B). The yellow shaded area indicates the reference interval of 2.00 to 2.80 mmol/l for total calcium.

Figure 2: Serum concentrations (means ± standard deviations) of ionised calcium after intravenous infusion of calcium borogluconate (group A) and after intravenous infusion of calcium borogluconate combined with oral administration of sodium phosphate (group B). The yellow shaded area indicates the reference interval of 1.06 to 1.26 mmol/l for ionised calcium.
Relationship between electrolyte concentrations and response to treatment

The pretreatment electrolyte concentrations and the profiles of total and ionised calcium, inorganic phosphate and magnesium after treatment did not differ significantly between the 32 cows that stood within eight hours of the start of treatment and the eight cows that did not.

Discussion

The clinical findings of the cows with parturient paresis were in agreement with those of other studies (Salis, 2002; Jehle, 2004; Dumelin, 2005; Zulliger, 2008; Blatter, 2012) and are not discussed further. Pretreatment calcium, inorganic phosphate and magnesium were 1.2 ± 0.45, 0.5 ± 0.26 and 1.2 ± 0.25 mmol/l, respectively, which were in agreement with previously reported means of 1.0 to 1.2 mmol/l for calcium, 0.4 to 0.6 mmol/l for inorganic phosphate and 1.2 to 1.3 mmol/l for magnesium (Salis, 2002; Jehle, 2004; Dumelin, 2005; Zulliger, 2008; Blatter, 2012). The mean concentration of ionised calcium in the present study was 0.5 ± 0.22 mmol/l, which was similar to that of the previous reports (0.6 to 0.7 mmol/l). Based on the classification system of Kvart et al. (1982), 57.5% of the cows had severe hypocalcaemia, which was higher than the range of 26.7 to 50.0% observed in earlier studies (Kvart et al., 1982; Salis, 2002; Jehle, 2004; Dumelin, 2005; Zulliger, 2008; Blatter, 2012).

The calcium profile of group A was very similar to those of previous studies, in which the control cows received the same treatment as in the present study (Salis, 2002; Dumelin, 2005; Blatter, 2012). After rapid intravenous calcium administration, cows were hypercalcaemic for about 2 hours, normocalcaemic for another five hours and then hypocalcaemic again. The calcium profile of group B was very similar to that seen in another study.

Table 2: Number of cows with hypocalcaemia and hypophosphataemia at different time points after the start of treatment.

<table>
<thead>
<tr>
<th>Hours after start of treatment</th>
<th>Hypocalcaemia (calcium &lt; 2.0 mmol/l)</th>
<th>Hypophosphataemia (inorganic phosphate &lt; 1.3 mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A 20 0 20 20 7 1* 14 5*</td>
<td>Group A 20 20 13 18 14 4*</td>
</tr>
<tr>
<td></td>
<td>Group B 7 13 9 12** 6 2</td>
<td>Group B 0 14 13 3 7 3</td>
</tr>
</tbody>
</table>

* Difference to group A: $P < 0.05$, chi² test  
** Difference to group A: $P < 0.01$, chi² test  
† Calcium and inorganic phosphate differ between groups ($P < 0.05$, chi²-test)  
‡ Inorganic phosphate differs between groups ($P < 0.05$, chi²-test)  
§ Calcium differs between groups ($P < 0.01$, chi²-test)
Treatment of cows with parturient paresis using intravenous calcium and oral sodium phosphate
U. Braun, D. Grob, M. Hässig

in which cows also received 1000 ml calcium borogluconate and were hypercalcaemic for 7 hours, then normocalcaemic for a short period of time and hypocalcaemic at 24 and 48 hours (Jehle 2004). Similar to previous findings (Jehle, 2004), cows of group B had lower calcium concentrations at 24, 48 and 72 hours than cows of group A, possibly because of longer-lasting hypercale mia in the former. It has been shown that within minutes, hypercalcaemia results in a drastic decrease in parathormone concentration and increased renal excretion of calcium (Goff, 1999). Parathormone concentration decreased sharply within 10 minutes after calcium infusion and remained at a low level for a few hours (Zulliger, 2008) or days (Dumelin, 2002). Hypercalcae mia also causes calcitonin secretion from the thyroid (Pearse, 1966; Care et al., 1970) and in turn inhibits bone resorption and stimulates urinary excretion of calcium. These mechanisms may have accounted for the lower calcium concentrations from 24 to 72 hours in group B.

The serum inorganic phosphate concentration increased markedly within 90 minutes after oral administration of 500 g sodium phosphate to cows of group B. A dose of 350 g gave similar but slightly lower inorganic phosphate concentrations (Dumelin, 2005; Braun et al., 2007b); 80% of cows had normo- or hyperphosphataemia 24 hours after the administration of 500 g sodium phosphate compared with 53% after the administration of 350 g (Dumelin, 2005; Braun et al., 2007). Several studies have shown that oral administration of sodium phosphate is effective in increasing the serum inorganic phosphate concentration in hypophosphataemic cows (Cheng et al., 1998; Dumelin, 2005; Zulliger, 2008). A recent study showed that monosodium and monopotassium phosphate are equally effective, whereas monocalcium phosphate has a less pronounced effect on the serum inorganic phosphate concentration (Idink and Grünberg, 2015). Sixty-five per cent of all cows were cured after a single treatment, which was considerably higher than the cure rates of 37% (Blatter, 2011), 46.7% (Salis, 2002; Jehle, 2004; Dumelin, 2005) and 56.7% (Zulliger, 2008) obtained previously at our clinic, but similar to rates observed by other authors (66.0%, Bostedt et al., 1979; 66.5%, Lesch and Gelfert, 2006; 73.5%, Siegwart and Niederer, 2005). Fourteen cows (70%) of group A and 18 cows (90%) of group B stood within 8 hours after the start of treatment. Increasing the dose of sodium phosphate from 350 g (Dumelin, 2005; Braun et al., 2007a) to 500 g increased the recovery rate numerically from 80 to 90%. Although cure rates did not differ significantly between groups A and B, these results and those by Dumelin (2005) suggest that adding oral sodium phosphate to the standard intravenous treatment of 500 ml calcium borogluconate improves the treatment outcome in cows with parturient paresis. They also corroborate the role of hypophosphataemia in the pathogenesis of parturient paresis, which recently has been studied experimentally in dairy cows (Grünberg et al., 2015). Within 9 days of dietary phosphorus deprivation, the plasma inorganic phosphate concentration decreased on average by 60%, and by 2 weeks, electromyographic examination showed increased occurrence of pathological spontaneous activity in striated muscle in several cows (Grünberg et al., 2015). This spontaneous muscle activity is suggestive of neuromuscular membrane instability and of the involvement of hypophosphataemia in the pathogenesis and perpetuation of parturient paresis. From a clinical point of view, the rapid resolution of the recumbent state is critical to prevent downer cow syndrome. A relapse after an initial response to treatment is considerably less detrimental than a total lack of response. Experimentally-induced downer cow syndrome (healthy anaesthetised cows maintained in sternal recumbency) resulted in ischaemic muscle necrosis of the pelvic limb that was positioned under the body after 6 to 12 hours of recumbency (Cox et al., 1982). In the current study, 2 cows of group A but none of group B had to be euthanized because of downer cow syndrome; in the study by Dumelin (2005), 2 cows of the control group and one cow that received oral phosphorus had downer cow syndrome. The relapse rate did not differ between the two groups and the overall rate was 17.5%. This was higher than in some studies (6.6%, Zulliger, 2008; 13.0%, Salis, 2002) and lower than in others (20.0%, Dumelin, 2005); 23.3%, Jehle, 2004; 33.3%, Blatter, 2012). Other authors reported relapse rates of 23 to 40% (Oetzel, 1988; Martig, 2002). The proportion of downer cows was 17.5%, which was similar to other reports (Kvart et al., 1982, 10.1%; Salis, 2002, 13.3%; Jehle, 2004, 13.3%; Zulliger, 2008, 13.3%; Blatter, 2012, 16.6%). There were slightly more downer cows in group A than in group B (4 vs. 1, P = 0.09).

Conclusion
Supplementation of the standard intravenous treatment with 500 g sodium phosphate administered orally to cows with parturient paresis caused normal or elevated serum inorganic phosphate concentration in 80% of cows. The response rate to the initial treatment was 90% in cows that received oral sodium phosphate in addition to intravenous calcium and 70% in cows treated with intravenous calcium alone. These findings, although not statistically significant, are promising and should be verified using a larger number of cows.
Traitément de la parésie post-partum des vaches au moyen de calcium intraveineux et de phosphate de sodium oral

On a étudié si on pouvait obtenir plus souvent, chez les vaches souffrant de parésie post-partum auxquelles on administrait par voie intraveineuse 1000 ml d’une solution à 40% de borogluconate de calcium et par voie orale 500 g de phosphate de sodium, une normo-calcémie et normo-phosphatémie durable et un meilleur résultat thérapeutique que chez les animaux recevant uniquement 500 ml de la solution de borogluconate de calcium. Les essais ont été faits sur 40 vaches laitières souffrant de parésie post-partum qui présentaient toutes initialement une hypocalcémie et une hypophosphatémie. Les animaux ont été répartis alternativement en deux groupes de traitement A et B, comprenant chacun 20 vaches. Les animaux des deux groupes ont été traités avec 500 ml d’une solution à 40% de borogluconate de calcium par voie intraveineuse. Ceux du groupe B ont reçu en outre 500 ml de borogluconate de calcium en perfusion continue et 500 g de phosphate de sodium par sonde naso-oesophagienne. En l’espace de 8 heures, 32 vaches se sont levées et 8 pas. Parmi les 32 vaches qui se sont levées, 18 appartenaient au groupe B (90%) et 14 au groupe A (70%) (P = 0.23). Chez 7 des 32 vaches qui se sont levées, on a observé une récidive. Parmi les 7 cas de récidive et les 8 vaches qui ne s’étaient pas levées après 8 heures, 10 animaux se sont levés après une deuxième perfusion de calcium. Cinq vaches ont développé un Downer-Cow-Syndrom, dont trois purent être guéris après un traitement intensif. Le succès thérapeutique global ne diffère pas significativement d’un groupe à l’autre. Dans le groupe A, 12 animaux (60%) ont été guéris après un seul traitement et, dans le groupe B 14 (70%). Sur le solde de 14 vaches, 11 se sont relevées après deux ou plusieurs traitements. Deux vaches ont dû être euthanasiées pour un Downer-Cow-Syndrom et une a péri d’une insuffisance cardiaque. Le traitement à conduit, chez les vaches du groupe B à des concentrations sanguines de calcium significativement plus élevées dans les premières 8 heures que chez celles du groupe A. D’autre part, l’administration, dans le groupe B, de phosphate de sodium a amené une élévation significative et durable de la concentration de phosphate inorganique. Le pourcentage des animaux qui se sont levés dans les 8 heures et étaient guéris après un seul traitement était plus élevé dans le groupe B que dans le groupe A (P >0.05). D’autres études, avec des nombres d’animaux plus importants, sont nécessaires pour conforter ces résultats.

Traitément con calcio per via endovenosa e fosfato di sodio per via orale per le mucche affette da collasso puerperale

Si è esaminato se la somministrazione, nelle mucche affette da collasso puerperale, di 1000 ml di una soluzione al 40% di calcio borogluconato per via endovenosa e 500 g di fosfato di sodio per via orale, possa spesso mantenere una normocalcemia, una normofosfatemia e dare migliori risultati che la sola somministrazione di 500 ml di calcio borogluconato. Gli esami sono stati eseguiti su 40 mucche da latte affette da collasso puerperale. Tutte le mucche avevano inizialmente una ipocalcemia e una ipofosfatemia. Le mucche sono state alternativamente suddivise in due gruppi di trattamento A e B di ciascuno 20 mucche. Le mucche di entrambi i gruppi sono state trattate per via endovenosa con 500 ml di una soluzione al 40% di calcio borogluconato. Le mucche nel gruppo B hanno ricevuto inoltre 500 ml di calcio borogluconato via infusione endovenosa continua e 500 g di fosfato di sodio per sondino nasogastrico. Entro 8 ore, 32 mucche si erano alzate mentre 8 no. Delle 32 mucche alzate, 18 (90%) animali erano del gruppo B e 14 (70%) del gruppo A (p = 0.23). Purtroppo, 7 delle 32 mucche hanno subito una ricaduta. Delle 7 mucche ricadive e delle 8 che non si erano alzate entro le 8 ore, dopo una rinnovata infusione di calcio, 10 si sono levate. Cinque mucche hanno sviluppato la sindrome della “downer cow”, tra queste 3 erano guarite dopo una terapia intensiva. Il successo del trattamento globale non si differenzia tra i due gruppi in modo significativo. Nel gruppo A, 12 (60%) mucche e nel gruppo B, 14 (70%) erano guarite dopo un unico trattamento. Delle restanti 14 mucche, 11 si sono alzate dopo due o più trattamenti. Due mucche hanno dovuto essere soppresse a causa della sindrome della “downer cow”, una invece è morta di insufficienza cardiaca. Il trattamento ha provocato nel gruppo B un aumento significativo della concentrazione di calcio nel sangue nelle prime 8 ore cosa non avvenuta nel gruppo A. In seguito, nel gruppo B si è rilevato mediante la somministrazione di fosfato di sodio un aumento significativo e costante della concentrazione di fosfato inorganico nel sangue. La percentuale di mucche che si sono alzate entro 8 ore e guarite dopo un solo trattamento, era superiore nel gruppo B che nel gruppo A (P>0.05). Per convalidare i risultati sono necessarie ulteriori analisi con un maggior numero di animali.
References


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