

A COMPARATIVE STUDY OF THE HEMATO-BIOCHEMICAL PARAMETERS BETWEEN CLINICALLY HEALTHY COWS AND COWS WITH DISPLACEMENT OF THE ABOMASUM

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The objective of this study was to evaluate hemato-biochemical parameters in cows with left- and right- sided displacement of the abomasum. Comparison of a group of cows with displacement of the abomasum (n = 73) and a control group of cows (n=43) of the same breed, age, physiological period and management showed statistically significant ($p < 0.05$) deviations of the following hematological and biochemical parameters: increased hematocrit (Ht), white blood cell count (WBCC), neutrophils, total bilirubin (Bil-T), glucose, β -hydroxybutyrate, non-esterified fatty acids (NEFA), serum enzymatic activity of aspartate aminotransferase (AST), γ -glutamyl transferase (GGT) and glutamate dehydrogenase (GLDH), together with decreased calcium (Ca), sodium (Na), potassium (K) and chloride (Cl) concentration. Blood analyses in cows with displacement of the abomasum clearly indicated association of the disease with postpartum disease syndromes such as hypocalcaemia, hepatic lipidosis and endotoxemia. Thus, hemoconcentration, leukocytosis with neutrophilia, hypocalcaemia, lipemia, ketonemia, hyperglycemia, slight hypernatremia, hypokalemia, hypochloremia and increased activity of liver enzymes (AST, GGT, GLDH) in blood serum were observed in these cows in comparison to the control group. All established irregularities cause complicated pathophysiological metabolic reactions, which are often clinically manifested as displacement of the abomasum during the periparturient period.

Key words: dairy cows, hemato-biochemical parameters, abomasal displacement, periparturient period

INTRODUCTION

The study of abomasal displacement is dynamic rather than static, as the disease furnishes material for scientific investigations all over the world, wherever dairying is of economic importance.

During the last ten years abomasal displacement in high-yielding Black & White cows has been one of the most common diagnoses among puerperal diseases in Slovenia (Zadnik *et al.*, 2001). The Clinic for Ruminants of Ljubljana has 30 years of experience in dealing with this problem (Skušek *et al.*, 1970). Despite extensive research the etiology and prevention of abomasal displacement have

not been sufficiently clarified (Breukink, 1991; Correra *et al.*, 1993; Geishauser, 1995b; Breukink and Wensing, 1997). Recent research has shown that several interacting pathobiochemical processes are associated with the development of abomasal displacement (Martens, 2000). The severity of clinical signs varies greatly and indicates disturbances of reticulo-abomaso-duodenal function and energy metabolism, as well as the lipoprotein, mineral-electrolyte, cardiopulmonary, thermoregulatory and immune systems (Thomas and Haddock, 1997; Fürell *et al.*, 1998; Vörös and Karsai, 1987; Sustronck, 2000). In addition, a clinician should pay attention to inflammatory and toxemic disorders together with visceral pain and its effect on catabolic metabolic processes in the affected organism (Lascelles, 1996; Thomas and Haddock, 1997). Visceral pain is manifested as depression, inappetence, tachycardia, polypnea, muscle shivering, moaning, grunting and grinding of the teeth (Lascelles, 1996; Obritzhauser *et al.*, 1998). The cause can be attributed to dilatation and distention due to displacement of the organ, twisting and stretching of the mesentery, inflammation of serous surfaces, inflamed abomasal mucosa and abomasal ulcerations. Gastric fluid with a high hydrogen and chloride ion concentration is sequestered in the abomasum or is refluxed by internal vomiting into the forestomach compartments (Vörös and Karsai, 1987; Madison and Troutt, 1988; Geishauser and Seeh, 1996; Sustronck, 2000;). Among clinical-laboratory results that are most significantly associated with the diagnosis of abomasal displacement are abomasal wall atony with abomasal dilatation, delayed abomasal emptying, duodenal-abomasal reflux, dehydration, hypocalcaemia, hyperglycemia, lipemia, hepatic lipidosis, ketonemia, toxemia and hypochloremic, hypokalemic metabolic alkalosis (Muylle *et al.*, 1990; Massey *et al.*, 1993; Fürell *et al.*, 1998; Itoh *et al.*, 1998; Ohtsuka *et al.*, 1997; Delgado-Lecaroz *et al.*, 2000; Ward *et al.*, 1994; Zadnik and Mesarič, 2000).

The aim of this report is to present and discuss our clinical and laboratory data from cows with abomasal displacement, which might be of interest in bovine husbandry.

MATERIALS AND METHODS

This study included 73 Black&White cows with abomasal displacement that were treated at the Clinic for Ruminants in Ljubljana from 1999 to 2002. Cows from 32 farms were sent for clinical treatment by field veterinarians with practices within 40 km around Ljubljana.

The type of displacement was diagnosed by routine clinical examination including inspection, palpation, auscultation, simultaneous percussion and auscultation, rectal examination, ballottement and succussion of the left and right side of the abdomen (Zadnik and Modic, 1995; Radostits *et al.*, 2000). Fundamental diagnosis was based on identification of a characteristic »ping and pung effect« in the area of the left and right abdomen. All clinical diagnoses were surgically verified and confirmed.

Prior to surgical intervention blood samples were taken from v. coccygicae via the vacuum method into three different tubes: VenoJect® tubes (3 ml) with anticoagulant EDTA (K₃) for hematological analyses, BD Vacutainer™ (Plymouth,

UK) tubes (3 ml) with LH (60 I.U.) and L Iodoacetate (1.5 mg) for measurement of glucose content and VenoJect® Terumo Europe (Belgium) tubes (10 ml) for biochemical analyses of blood serum.

The hematological status of the cows was determined by blood analysis on a Vet abc™ electronic counter (Animal Blood Counter, France).

Biochemical analyses of blood serum were performed using a Cobas Mira biochemical analyzer (La Roche Diagnostics).

Energy and lipid metabolic status of the cows was evaluated by determination of BHB, NEFA, glucose and total cholesterol concentrations in blood serum.

Liver health was evaluated by determination of AST, GGT and GLDH activities and measurement of total bilirubin content in blood serum.

Mineral-electrolyte status was determined by measurement of Ca, Na, K, and Cl content.

The results of hematological-biochemical analyses of cows with abomasal displacement were compared with the results of the analyses of the control group. The control group included 43 clinically normal Black&White cows, which originated from the same area as the patients. The cows were of the same age, breed and post partum period in the same season (October – May). They were kept under similar feeding and management conditions.

All data were processed via the Statistical Package for Social Science, (SPSS version 10.0 for Windows). After analysis of mean value variance of hemato-biochemical parameters the subprogram Anova was used.

RESULTS

Mean age and mean post partum period of cows with abomasal displacement and the control group did not significantly deviate ($p > 0.05$). In 36% of cows with displacement obvious signs of toxemia were detected. Metritis was found in 15.2% and mastitis in 13.8% of the affected cows.

Table 1. Mean age, mean post parturient period, SD and P value in the abomasal displacement and control groups

Group of cows	Number of cows	Mean age \pm SD (year)	Mean post parturient time \pm SD (day)
Left abomasal displacement	57	4.03 \pm 1.42	13.2 \pm 8.5
Right abomasal displacement	16	4.62 \pm 1.81	21.1 \pm 15.7
Left + right abomasal displacement	73	4.16 \pm 1.52	14.9 \pm 10.9
Control	43	4.59 \pm 1.36	18.4 \pm 5.7

P > 0.05

Table 2. Mean values \pm SD of hemato-biochemical parameters in the abomasal displacement and control groups and P values obtained for differences between mean values for sick and healthy cows

Hemato-biochemical parameters	Left abomasal displacement (n = 57)	Right abomasal displacement (n = 16)	Left + right abomasal displacement (n = 73)	Control healthy cows (n = 43)
Ht (L/L)	0.334 \pm 0.056	0.340 \pm 0.039	0.335 \pm 0.052	0.292 \pm 0.019***
WBCC ($\times 10^9$ /L)	7.68 \pm 3.05	10.98 \pm 6.36	8.41 \pm 4.21	8.03 \pm 1.43 *
Neutrophils (%)	50.5 \pm 17.2	57.6 \pm 14.2	52.1 \pm 16.1	35.1 \pm 9.2 ***
Ca (mmol/L)	2.20 \pm 0.21	2.15 \pm 0.25	2.19 \pm 0.22	2.36 \pm 0.18 ***
Na (mmol/L)	142.9 \pm 5.1	140.5 \pm 3.8	142.0 \pm 4.9	146.8 \pm 2.7 ***
K (mmol/L)	3.9 \pm 0.5	4.1 \pm 0.7	3.9 \pm 0.6	5.1 \pm 0.4 ***
Cl (mmol/L)	99.3 \pm 7.5	93.7 \pm 12.0	98.0 \pm 8.9	105.2 \pm 3.0 ***
Bili-T (μ mol/L)	22.6 \pm 8.0	17.7 \pm 8.9	21.5 \pm 8.4	4.6 \pm 1.9***
AST (IU/L)	155.0 \pm 96.9	180.0 \pm 126.0	161.1 \pm 103.4	60.2 \pm 12.1 ***
GGT (IU/L)	35.5 \pm 39.8	65.5 \pm 42.2	42.3 \pm 41.9	20.9 \pm 6.7 **
GLDH (IU/L)	40.4 \pm 83.3	101.5 \pm 117.2	54.7 \pm 41.9	6.7 \pm 2.5 ***
Glucose (mmol/L)	4.74 \pm 1.84	6.48 \pm 4.54	5.14 \pm 2.75	3.17 \pm 0.48 ***
Cholesterol (mmol/L)	2.68 \pm 1.86	2.75 \pm 1.50	2.69 \pm 1.77	3.03 \pm 1.05 NS
BHB (mmol/L)	1.77 \pm 1.37	1.07 \pm 1.21	1.62 \pm 1.36	0.71 \pm 0.22 ***
NEFA (mmol/L)	2.67 \pm 0.45	1.01 \pm 0.71	2.31 \pm 0.41	0.35 \pm 0.21 **

p<0.05 ; *p<0.001; NS=non significant

DISCUSSION

In 73 cows with abomasal displacement, from 1999 to 2002, we found 56 (77.7%) to the left and 16 (22.3%) to the right. In 36% of the cows severe toxemia had to be dealt with. Displacement of the abomasum was sometimes accompanied by marked metritis (15.2%) and mastitis (13.8%). Periparturient dairy cows are quite susceptible to intramammary infections and clinical mastitis (Marcus and Goff, 1989). Moreover, Markusfeld (1986), Corraera *et al.* (1993) and Grohn *et al.* (1995) reported the same, frequent occurrence of complications due to abomasal displacement.

The significantly elevated mean hematocrit in cows with displacement in comparison to control cows indicated moderate dehydration. Hemoconcentration was more pronounced in right-sided displacements. Hemoconcentration with clinically manifested dehydration was identified in sheep and cows via experimen-

tally induced blockage of the cranial part of the duodenum just behind the pylorus (Fubini *et al.*, 1991; Ward *et al.*, 1994). In dislocation of the abomasum very similar pathological events may be observed because emptying of the contents from the organ is obstructed. It is known that cows with displacement defecate less frequently and the feces are usually scanty (Radostits *et al.*, 2000). This was confirmed by our clinical findings (Zadnik and Modic, 1995). All cows included in the experiment exhibited signs of dehydration, as the mean hematocrit increased by 30%, that is from $30.5 \pm 0.9\%$ to $41.5 \pm 2.1\%$ (Ward *et al.*, 1994). Hemoconcentration is also associated with insufficient abomasal emptying due to duodenal-abomasal reflux, as well as decreased food and water intake (Rings *et al.*, 1984; Ward *et al.*, 1994; Geishauser and Seeh, 1996).

Both disturbances can be clinically recognized. In cattle two hours after i/v $O_{26}B_6$ E. coli endotoxin infusion the abomasal emptying rate was reduced to 50%. Before endotoxin infusion the animals showed a normal abomasal emptying rate of 4.400 ± 2.000 ml/hour (Sustronck, 2000). After increased endotoxin application cessation of rumen contractions was observed for several hours (Sustronck, 2000).

Leukocytosis with neutrophilia occurred in our cows, especially in right-sided dislocation, is an immunological response to endotoxemia, abomasitis and peritonitis. Other authors also detected leukocytosis, neutrophilia and hypergamma-globinemia in 75% of animals with displacement due to accompanying inflammatory disorders (peritonitis, metritis, mastitis) and circulating bacterial and metabolic toxins (Cullor, 1992; Edas, 1993; Geishauser, 1995b; Sustronck, 2000). Subacute endotoxemia may be the cause for leukocytosis, neutrophilia and hemoconcentration (Edas, 1993; Sustronck, 2000). Endotoxemia results in systemic hypotension, capillary permeability is increased and diffuse formation of microvascular obstructions is seen. Due to increased vascular permeability a transmural shift of albumin and other colloids occurs, which carries water to the interstitial space (Cullor, 1992).

The majority of our cows with abomasal displacement showed mild hypocalcaemia, namely, the mean value significantly ($p < 0.001$) deviated from that for control cows. Similar results were reported by Delgado-Lecaroz *et al.* (2000), who found that as much as 70% of affected cows showed serum Ca values below 2.08 mmol/L. Moderate hypocalcaemia in cows within the first week post partum to a certain degree is a physiological process. However, obviously disturbed abomasal motility occurred when the Ca concentration dropped to 50% or 1.2 mmol/L (Daniel, 1983; Madison and Trout, 1988). Puerperal hypocalcaemia (< 1.97 mmol/L) represents a significant risk factor for the development of abomasal displacement (Massey *et al.*, 1993; Østergaard and Gröhn, 1999; Houe *et al.*, 2001). In cows suffering from hypocalcaemia two weeks after parturition the risk for the development of left-sided dislocation was great (Massey *et al.*, 1993; Geishauser *et al.*, 1998b; Geishauser *et al.*, 1999). Hypocalcaemia adversely affects the tone of the abomasal and ruminoreticular wall (Huber *et al.*, 1981; Daniel, 1983; Madison and Trout, 1988; Delgado-Lecaroz *et al.*, 2000). Emptying of contents from the abomasum was even more obstructed if the organism suffered from metabolic alkalosis (Poulsen and Jones, 1974). Ward *et al.* (1994) reported that plasma cal-

cium concentration showed a substantial decrease during the first 6 h of diversion of abomasal outflow and a peak deviation from baseline values. At the end of diversion all cows were significantly hypocalcaemic (1.6 ± 0.15 mmol/L; $p < 0.01$). When hypocalcaemia followed metabolic alkalosis, cows responded with an increased incidence of dislocations (Vörös and Karsai, 1987; Geishauser and Okenotrop, 1997). Adding anion salts to the diet of dry cows to achieve mild metabolic acidosis (acidification) is currently used for prevention of post parturient hypocalcaemia and puerperal paresis (Block, 1994; Gašperlin and Zadnik, 2001). However, according to Sustronck (2000) further application of calcium drugs (i/v, p/os) is not effective; the relapse of abomasal displacement was significant (65% – 70%).

Because the ruminant forestomach compartments have the capacity to absorb Na^+ and Cl^- , the reflux and sequestration of abomasal secretions that occur in an obstruction/ligation model or naturally-occurring obstructions may make these ions, to some degree, accessible to the plasma by this route (Gable and Martens, 1991; Ward *et al.*, 1994). Cows with displacement, especially to the right-side, showed a significantly ($p < 0.001$) lower mean level of sodium electrolyte in serum compared to control cows. Barely recognized hyponatremia and hypokalemia and pronounced hypochloremia resulted mainly from metabolic acid-base imbalance in the affected cows due to the duodeno-abomasal reflux of contents and endotoxemia (Fubini *et al.*, 1991; Edas, 1993; Ward *et al.*, 1994; Geishauser and Seeh, 1996; Ohtsuka *et al.*, 1997a). Ohtsuka *et al.* (1997a) reported that 12 to 24 hours after experimentally induced toxemia via $\text{O}_{26} \text{B}_6$ E.coli toxin, the level of sodium electrolyte was significantly ($p < 0.05$) elevated to 146.0 mmol/L, while the level of chloride dropped to 96.0 mmol/L and of potassium to 3.2 mmol/L. After 24 hours the concentrations stabilized and were similar to those of the control group. Hypochloremia (94.67 ± 2.54 mmol/L), hypokalemia (3.49 ± 0.33 mmol/L) and pronounced metabolic alkalosis with respect to the control group of cows were established also in cows with toxemic mastitis (Ohtsuka *et al.*, 1997b). The duodeno-abomasal reflux is extensively increased predominantly in cows with right-sided displacement, namely, a 2- to 3- fold increased concentration of bile acids was found in the reticulo-ruminal fluid in cows with dislocation (Geishauser and Seeh, 1996).

Ten to twelve hours after experimentally induced reduction of abomasal emptying into the duodenum, cows began to show signs of muscular weakness, depression and dehydration (Ward *et al.*, 1994). Eight hours after duodenal obstruction hypochloremia (87.2 mmol/L), hypokalemia (2.9 mmol/L), hypocalcaemia (1.62 mmol/L) and metabolic alkalosis were noted in blood plasma of the affected cows. No significant differences occurred in sodium electrolyte concentration (Ward *et al.*, 1994). In three sheep suffering from abomasal emptying defects chloride electrolyte content in the abomasal fluid increased in the first sheep to 35.0 mmol/L, in the second to 50.0 mmol/L and in the third one even to 130.0 mmol/L (Rings *et al.*, 1984). The physiologically optimal chloride concentration in abomasal fluid is considered to range between 8.0 and 15.0 mmol/L (Rings *et al.*, 1984).

In our studies on cows with abomasal displacement a marked hyperbilirubinemia was observed. We are of the opinion that hyperbilirubinemia, especially in cows with left-sided displacement, resulted from bile duct obstruction because of progressive anatomic changes in the positions of the abomasum and omentum. Simultaneously with displacement of the abomasum the duodenum is also grossly stretched and extended. Ductus choledochus enters the duodenum 50 to 70 cm caudally from the pylorus. In left-sided displacement a smaller duodeno-abomasal reflux was detected compared to that in right-sided displacement and torsions (Geishauser *et al.*, 1997a). According to our own experience and the observations of Geishauser and Seeh (1996), an enlarged gallbladder is always found during reposition of a displaced abomasum. On the first day after reposition of a dislocated abomasum an increased concentration of bile acids ($3.37 \pm 1.84 \mu\text{mol/L}$) was found in abomasal fluid. However, the concentration dropped to $1.66 \pm 1.53 \mu\text{mol/L}$ on the second and third day after reposition of the abomasum. The normal bile acid concentration in cows on a hay-concentrate diet was $1.30 \pm 1.01 \mu\text{mol/L}$ (Geishauser and Seeh, 1996).

In our case significantly ($p < 0.001$) increased mean enzyme activities of AST, GGT and GLDH confirmed the association of dislocation with hepatic lipodosis, endotoxemia and hepatocyte damage. We are of the opinion that significantly increased GGT activity resulted from disturbed outflow into the duodenum. Increased AST activity from 100.0 to 180.0 U/L in the blood of cows 1 to 2 weeks post parturition provides a good diagnostic aid in predicting the development of abomasal dislocation (Geishauser *et al.*, 1997b; Geishauser and Okentrop, 1997). Itoh *et al.* (1998) reported that cows with displacement of the abomasum showed significantly ($p < 0.01$) increased mean enzyme activities of AST (280.0 ± 155.0 IU/L) and GGT (43.2 ± 27.4 IU/L) compared to control group. Moreover, the enzyme activities significantly deviated ($p < 0.05$) from those for the group of cows with primary ketosis.

In our study hyperglycemia was observed in the group of cows with abomasal displacement. Other authors also reported a similarly increased mean glucose concentration 4.11 ± 1.49 mmol/L in blood of cows with abomasal displacement (Van Meirhaege *et al.*, 1988; Itoh *et al.*, 1998). Obvious hyperglycemia was found in cows with right-sided dislocation probably due to rapidly developing endotoxemic shock. Hyperglycemia is also likely to occur when tissues fail to respond to insulin. Insulin plays the central role in moving the cow's metabolism to words lipogenesis in the early stage of pregnancy and lipolysis in late pregnancy and at the beginning of lactation (McNamara, 1991). It has been confirmed that diminished tissue sensitivity to insulin stimulation in late pregnancy and early lactation is also associated with impaired abomasal emptying and reduced contraction of the abomasal wall. Sustronck (2000) reported a clear correlation between hyperinsulinemia and delayed emptying of abomasal contents. In his experiment the inhibitory effect of insulin was independent of the glucose concentration in blood. However, it is interesting that he did not establish any association between hyperglycemia and abomasal emptying rate (Sustronck, 2000). A hyperglycemic form of ketosis (type II) is associated with decreased absorption of glucose because of impaired regulation of metabolic energy (Holtenius and Holtenius, 1996;

Sustronck, 2000). This finding and the often prolonged post parturition hypocalcaemia are important pathogenetic factors in the development of abomasal dislocation (Klucinski *et al.*, 1988; Muylle *et al.*, 1990; Kerli and Goff, 1992). On the basis of our results we are of the opinion that hyperglycemia, especially in right-sided dislocation, may be associated with impaired outflow of pancreatic juice and disturbed blood circulation in the pancreatic parenchyma because of changes of duodenal and omental position due to dislocation of the abomasum. In cattle there is no main pancreatic duct while the accessory one is 80 - 90 cm away from the pylorus and 30 - 40 cm from ductus choledochus (Zietzschmann *et al.*, 1974). Geishauser *et al.* (1998) are of the opinion that glucose concentration does not have diagnostic significance for left-sided dislocation of the abomasum.

Increased mean levels of BHB and NEFA confirmed that cows with dislocation suffered from obvious and prolonged disturbance in glucose and lipid metabolism due to the combined pathological influences of endotoxemia, energy imbalance (ketosis) and hepatic lipidosis. Similar results were reported by Itoh *et al.* (1998). The optimal BHB concentration in dairy cows is below 1.0 mmol/L (Whitaker *et al.*, 1983). The concentration begins to increase when the animal is submitted to enhanced energy stress (Whitaker, 1997). The optimal NEFA concentration in blood serum is under 0.7 mmol/L (Whitaker, 1997). NEFA is a better indicator of lipid metabolism than BHB (Whitaker, 1997). In cows which suffered simultaneously from fatty liver, ketosis and left-sided dislocation less apolipoprotein B-100 and apolipoprotein A-1 was detected (Oikawa *et al.*, 1997). Oikawa *et al.* (1997) reported that cows with ketosis and left-sided dislocation showed the highest mean BHB (3.65 ± 1.75 mmol/L) and NEFA (1.65 ± 0.55 mmol/L) concentration. Cows with left-sided abomasal displacement but without ketosis showed significantly ($p < 0.01$) lower BHB (0.657 ± 0.244 mmol/l) and NEFA (0.957 ± 0.313 mmol/L) concentrations. The significantly lowest mean BHB (0.852 ± 0.319 mmol/L) and NEFA (0.336 ± 0.147 mmol/L) concentrations were found in control cows in early lactation (Oikawa *et al.*, 1997). BHB levels (from 1.0 to 1.6 mmol/L) in cows 1 to 2 weeks post parturition were fairly reliable indicators of left - sided abomasal dislocation (Geishauser *et al.*, 1997a; Geishauser *et al.*, 1997b).

In our study mean total cholesterol concentration in cows with dislocation did not significantly deviate ($P = 0.262$) from the value for the control group, although a decrease was observed. A significant decrease of mean cholesterol concentration (1.89 ± 0.56 mmol/L) in cows with dislocation was reported by Itoh *et al.* (1998). In blood serum of cattle highly saturated lipoprotein is the predominant lipoprotein species. Cholesterol is the dominant lipid in this fraction (Raphael *et al.*, 1973). In primary ketosis cholesterol concentration was significantly increased (Itoh *et al.*, 1998). In fatty liver degeneration a very low cholesterol concentration was found while NEFA (FFA) and phospholipid concentration were very high (Holtenius, 1989). This finding illustrates that lipoprotein synthesis was reduced in the liver by fatty infiltration due to NEFA entering and cholesterol leaving the liver (Holtenius, 1989). Cows with primary ketosis usually show a high NEFA level and fairly normal cholesterol and phospholipid concentrations (Holtenius, 1989; Itoh *et al.*, 1998).

During the puerperal period subclinical endotoxemia due to more or less developed hepatic lipidosis needs to be taken into account in cows with signs of inappetence, anorexia, indigestion, ketosis, abomasal dislocation, metritis and mastitis. Such endotoxemia was experimentally produced by administration of toxins from the group of eicosanoids e.g. thromboxane, prostacyclin, prostaglandin E (Andersen, 1988; Jarlov *et al.*, 1988; Sustronck, 2000). Eicosanoid endotoxins affect the motility of smooth muscles of the forestomach and the abomasum by inhibiting neurally produced responses. Toxins are instrumental in the development of abomasal dislocation. Recently some *in vitro* and pharmacological studies proven that inhibition of motility was associated with malfunction of the intrinsic nervous system - longitudinal muscle myenteric plexus from the abomasal antrum - in combination with the loss of cholinergic muscle responses because of which nitrergic inhibitory abomasal wall tone increased (Ooms and Oyaert, 1978; Geishauser, 1995a; Nelson *et al.*, 1995; Füll *et al.*, 1998; Geishauser and Reiche, 1998; Reiche *et al.*, 2000).

CONCLUSION

On the basis of our results and reports from the literature it can be concluded that abomasal dislocation is accompanied by moderate and/or severe hepatic lipidosis, negative energy balance, endotoxemia and subclinical hypocalcaemia. Metritis and mastitis are frequent clinically established complications indicating the immunosuppressive effect of bacterial and metabolic endotoxins and ketone compounds with resulting immune deficiency in cows with dislocation. The results obtained for hemato-biochemical analyses indicate a multifactorial etiology with predominant metabolic and hormonal imbalance in the critical three weeks before and three weeks after parturition.

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KOMPARATIVNA STUDIJA HEMATOLOŠKO-BIOHEMIJSKIH PARAMETARA KOD KLINIČKI ZDRAVIH KRAVA I KRAVA SA DISLOKACIJOM SIRIŠTA

ZADNIK T

SADRŽAJ

U ovom radu smo izneli rezultate hematoloških i bihemijskih parametre kod krava sa levom i desnom dislokacijom sirišta. Rezultati analize krvi i statistička ocjena rezultata u grupi krava (n = 73) sa dislokacijom sirišta i kontrolnoj grupi zdravih krava (n = 43) koje su bile iste rase, starosti, u istom fiziološkom periodu i sa istog područja/stada, ukazuju na statističku signifikantnu ($p < 0.05$) devijaciju sledećih hematoloških i biokemijskih parametara: povećana vrijednost hematokrita (Ht), bijele krvne slike (WBCC), neutrofila, totalnog bilirubina (Bil-T), glikoze, β -hydroxybutirata (BHB), nezasićenih masnih kiselina (NEFFA), povećana aktivnost enzima aspartat aminotransferase (AST), γ -glutamil transferase (GGT) i glutamat dehidrogenase (GLDH) te pad u koncentraciji kalcija (Ca), natrija (Na), kalija (K) i klorida (Cl). Krvne analize kod krava sa dislokacijom sirišta jasno ukazuju na povezanost dislokacije sa puerperalnim sindromom "hipokalcemija - hepatička lipidoza – endotoksemija". Kod naših pacijenata u odnosu na kontrolnu grupu zdravih krava utvrđene su hemokoncentracija, leukocitoza sa neutrofilijom, hipokalcemija, lipemija, ketonemija, hiperglikemija, umerena hipernatremija, hipokalemija, hipokloremija i povećana aktivnost jetrenih enzima AST, GGT i GLDH. Utvrđena odstupanja od normale prouzrokuju brojne i komplikovane patofiziološke metaboličke reakcije, koje se u puerperiju često klinički jasno odražavaju kao dislokacija sirišta.