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SOME FACTORS AFFECTING MASTITIS OCCURRENCE RATE IN COWS

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The impairment of the udder defense mechanisms causes an increase in the subclinical and clinical mastitis rate in dairy cows. The influence of some factors, that can depress leukocyte activity, on clinical mastitis occurrence rate during the first 8 weeks of lactation was studied on 313 dairy cows in one big farm. Thirty-nine per cent of the animals were affected with clinical mastitis. The predominant mastitis pathogens isolated were streptococci other than-*Streptococcus agalactiae* (41.25%), coagulase – negative staphylococci (11.25%) and coliforms other than *Escherichia coli* (9.37%). Most of mastitis cases appeared during the first 2 weeks of lactation. The cows in fourth and later lactations were more susceptible than younger animals. Also cows with mastitis in previous lactation were more often affected with this disease than animals with healthy udder. Retained placenta and subclinical ketosis did not increase the risk of mastitis.

Key words: mastitis, ketosis, retained placenta.

Mastitis is an inflamatory process of the mammary gland which causes severe losses in milk production and lowers the milk quality. An insufficiency of the mammary gland defence system is considered as the main circumstance of the infection. The natural udder defence mechanism is composed of the phagocytic leukocytes that are effective at ingesting and destroying bacteria. Diapedesis and migration of polymorphonuclear neutrophil leukocytes (PMN) into the mammary tissue and milk are most important to suppress bacterial multiplication and a subsequent infection (29,36).These leukocytes are called milk somatic cells and enter the mammary tissue from the blood. Many genetic and environmental factors affect the number and kinds of leukocytes in milk (19,32,34). Both, the number and activity of PMN can influence the susceptibility of the mammary gland to pathogens. An increased incidence of clinical mastitis and severity of the disease is associated with impairment of cow defence mechanisms (4,5,14,22,43).The resistance to infection is not constant. A depression of important immune functions around the parturition has been reported. Neutrophils obtained during the first week of lactation exhibit an impaired ability to ingest and kill bacteria (4,16,17,18). The degree of periparturient impairment in PMN functions experienced by cows of advanced parity is more severe than it is in younger ones (11).Feeding practices and some health disorders also can influence bovine neutrophil functions at that time (4,28,44).

Bovine ketosis is a common metabolic disease of lactating cows occurring during the first weeks after calving. At that time, dairy cows usually go into negative energy balance what can lead to increased fat mobilisation, increased hepatic

ketogenesis and high concentrations of ketone bodies in blood, milk and other body fluids. (2). Many studies have shown that increased concentrations of ketone bodies reduce the chemotaxis and can inhibit phagocytic and postphagocytic activities of bovine leukocytes in blood and milk *in vitro* and *in vivo* (15,21,25,37).

At the time of parturition, placental tissue becomes a dead foreign body that should be recognised and then rejected in order to complete separation of fetal membranes. A decreased number of leukocytes in the cotyledons at parturition and a decrease in chemotactic activity several days before it were associated with subsequent retained placenta (12,13). In cows with retained placenta a lower ingestion capacity of blood leukocytes was noticed from 2 weeks before to 2 weeks after parturition (16,33). It has been hypothesized that animals who experience a retained placenta due to a decrease in peripheral leukocyte activity are at higher risk of clinical mastitis (35) .

The objective of the study was to evaluate the effects of some factors that impair leukocyte function, such as retained placenta, ketosis, age, stage of lactation on incidence of clinical mastitis in early lactation.

Material and Methods

The investigations were performed during one year on HF cross-bred animals in a commercial dairy herd of 500 cows. Multiparous cows had given an average 5700kg milk in the previous lactation. The cows were machine milked twice daily. A premilking and postmilking teat disinfection was practised during the study with the use of an iodophor product. All multiparous cows received "dry cow" therapy. Animals were housed in a free stall barn and they had access to exercise paddock. Approximately one month before expected parturition they were put in the maternity barn, and usually stayed there 5-7 days after calving or till the end of udder therapy. Feeding of the animals as concerns the energy intake and basic nutritional components, was satisfactory. Puerperal period of 313 cows was analysed. Forty-eight of them (15.3%) did not expel the placenta spontaneously during the first 12 hours after calving. The cows with retained placenta (RP) and 265 cows which expelled the placenta spontaneously (NRP) were examined for mastitis during the first two months of lactation.

Diagnosis of mastitis was based on clinical examination of the udder, macroscopic examination of milk and California Mastitis Test (CTM). Inflamed secretion samples from each quarter that had visible signs of mastitis were collected for bacteriological examination before treatment. The examinations were performed as previously described (27). Only the first cases of mastitis at the study time were included. Four cows with the same bacteria in milk at drying off and after parturition were excluded from analysis. During the study, 6 cows were removed from the herd or died because of disease other than mastitis. Additionally, 37 RP cows and 36 NRP cows which calved at the same time were examined for ketosis. Samples of venous blood taken weekly were analysed for concentrations of ketone bodies (KB), by the method of Göschke. Cows with blood KB level >861μmol/l were recognized as ketotic animals (10). Alimentary ketosis was not found in the course of examination.

Effect of parity, retained placenta and effect of mastitis in previous lactation on the rate of clinical mastitis was determined by chi–square analysis.

Results

The prevalence of clinical mastitis during the first weeks after parturition is show in Table 1. The total number of cows with mastitis was 119 (39.3%). The rate of clinical mastitis was greater during the 1st wk of lactation than in the remainder. There were 39.6 % of RP cows and 39.2 of NRP cows affected with mastitis during the 8 weeks observation. The peak incidence rate of clinical mastitis for both, RP and NRP cows occurred just after calving.

Table 1

Prevalence of clinical mastitis in cows with retained placenta and cows with normal puerperium during the first 8 weeks of lactation

Group of cows	No of mastitic cows	Frequency of mastitis in each week							
		1	2	3	4	5	6	7	8
RP cows	19	12	3	2	-	1	1	-	-
(n=48)	(39.6) [1)]	(63.1)	(15.8)	(10.5)	-	(5.3)	(5.3)	-	-
NRP cows	100	63	14	9	6	2	2	3	1
(n=255)	(39.2)	(63.0)	(14.0)	(9.0)	(6.0)	(2.0)	(2.0)	(3.0)	(1.0)
Total	119	75	17	11	6	3	3	3	1
	(39.3)	(63.0	(14.3)	(9.2)	(5.0)	(2.5)	(2.5)	(2.5)	(0.8)

Explanation: RP – retained placenta, NRP – not retained placenta, [1)] per cent of cases in brackets.

Table 2 contains the results of bacteriological examinations of secretion from clinically inflamed quarters. The predominant mastitis pathogens isolated were streptococci other than *Streptococcus agalactiae* (41.25%), coagulase-negative staphylococci (11.25%) and coliforms other than *Escherichia coli* (9.37%).

Table 2

Bacteriological findings in secretion from mammary quarters at time of CM diagnosis

Bacteria	No of infected quarters		
	NRP cows	RP cows	Total
Streptococcus agalactiae	11 (7.9)[2)]	3 (14.3)	14 (8.7)
Other streptococci	58 (41.7)	8 (38.1)	66 (41.2)
Staphylococcus aureus	6 (4.3)	-	6 (3.7)
CNS	15 (10.9)	3 (14.3)	18 (11.2)
E. coli	4 (2.9)	3 (14.3)	7 (4.4)
Other coliforms	11 (7.9)	4 (19.0)	15 (9.4)
Corynebacterium bovis	5 (3.6)	-	5 (3.1)
Arcanobacterium pyogenes	2 (1.4)	-	2 (1.2)
Bacillus spp.	5 (3.6)	-	5 (3.1)
Micrococcus sp.	2 (1.4)	-	2 (1.2)
No growth or mixed culture[1)]	20 (14.4)	-	20 (12.5)
Total	139 (100)	21 (100)	160 (100)

Explanation: RP, NRP as in Table 1, [1)]bacteriologically negative samples and samples containing a mixed culture of environmental organisms, [2)]per cent of cases in brackets.

The most RP cows with the blood KB concentration greater than 861μmol/l (60%) was found in the first postpartum week. In the NRP group, most of the ketotic cows were found in the third (48.6%) and sixth (45.9%) weeks after parturition (Table 3). None of the cows developed clinical ketosis throughout the study.

Table 3

Per cent of cows with retained placenta and cows with normal puerperium with subclinical mastitis in the first 8 weeks of lactation

Group of cows		Weeks after parturition							
		1	2	3	4	5	6	7	8
RP cows (n=38)	%	60.5	34.2	18.4	13.2	15.8	5.2	5.2	7.9
NRP cows (n=37)	%	8.1	43.2	48.6	29.7	27.0	45.9	27.0	21.6

Explanation: RP, NRP as in Table 1.

Influence of parity on the prevalence of clinical mastitis in NRP group is demonstrated in Table 4. The cows with four and more than four lactations were affected nearly 20% more often than the younger ones (55.4 and 32.6 per cent, respectively).

Table 4

Effect of lactation number on mastitis occurrence rate (cows with normal puerperium)

Number of lactation	1	2	3	4	5	6 - 8	Total
Examined cows	81	60	40	30	18	26	255
No of cases	26	20	13	17	10	14	100
% mastitic cows	32.1	33.23	32.5	56.7	55.5	54.8	39.2

As Table 5 indicates, cows with history of mastitis in the previous lactation had an increased risk of mastitis (59.3%) during the next lactation as compared with healthy cows in the previous lactation (37.1%) and heifers (35.5%).

Table 5

Prevalence of mastitis in cows with and without a mastitis history in the previous lactation

Group of cows	No of examined cows	Cows with mastitis	
		number	%
Cows with mastitis in the previous lactation	59	47	57.3
Cows without mastitis in the previous lactation	97	44	33.8
Primiparous cows	62	30	32.9

Discussion

In the present study, most of clinical mastitis cases occurred during the first two weeks of lactation. Environmental mastitis pathogens caused a high proportion of clinical mastitis at that time. Contagious pathogens (*Staphylococcus aureus,*

Streptococcus agalactiae) were found in samples from only 12.5 per cent of clinically affected quarters.

The percentage of mastitic cows was similar to the prevalence reported by Whitaker *et al.* (40) and Wilson *et al.* (41). In the first 2 weeks of lactation, the cows appeared to be most susceptible to the disease. At that time, the highest incidence rate of intramammary infections (IMI) and clinical mastitis were observed by many authors (1,3,26,30,39). In *post partum* period mastitis could be the result of new IMI which occurred after calving or persisted from the dry period (30). The persistence of *Streptococcus dysgalactiae* from the dry period was confirmed using DNA typing (1).

In the present study, age of the cows influenced the rate of clinical mastitis. We observed that proportion of cases of mastitis in fourth and later lactations was 22.8 per cent greater than that found in the first 3 lactations (df=1, χ^2=4.23, P=0.039). However, cows in the first to third lactations appeared to be equally susceptible to mastitis. There was also no differences between cows in the fourth to eighth lactations. Todhunter (39) reported that the rate of environmetal streptococcal intramammary infection was significantly higher during the first half of the dry period for cows in a dry period 4 or further, and the rate of IMI was higher during lactation for cows with>4 lactations. Gilbert *et. al.* (11) reported that the degree of periparturient impairment in neutrophil function experienced by cows of advanced parity (4 or more) is more severe than it is in younger cows and that this may be a factor mediating their increased susceptibility to some *post partum* complications. In many studies (3,7,8,9,30,39), older cows were found to be at increased risk of clinical mastitis. According to Emanuelson *et al.* (8) older cows are at higher risk not only of mastitis, but also of retained placenta and ketosis. In our study, cows in parity greater than 4 had higher incidence rate of retained placenta as compared with younger animals (df=1, χ^2=3.49, P=0.06), data not published.

Cows with mastitic history in the previous lactation had a greater chance of being treated for mastitis in the next lactation as compared with the cows with healthy udder in the previous lactation and heifers, which remained more often healthy throughout the study. The differences were statistically significant (df=1, χ^2=4,38, P=0.036 for multiparous and df=1, χ^2=3.97, P=0.046 for primiparous cows). There are evidences that mastitis tends to recur in the same cow and suggestions that under the same conditions, some cows are more susceptible to infection than others.

The retained placenta did not increase incidence of mastitis in this study. Percentage of mastitic cows was similar both in RP and NRP groups, and the majority of cases (almost 80%) occurred in the first two weeks after calving. Erb *et al.*(9) and Correa *et al.* (6) did not find the association between retained placenta and mastitis. Zduńczyk *et al.* (42) reported that after induction of calving with PGF 2α, number of cows with retained placenta was much higher compared with cows after natural parturition (80 and 7.5% respectively), but mastitis occurrence rate was similar in the two groups. According to Emanuelson *et al.* (8) retained placenta increased risk of mastitis only in Swedish Red and White breeds but not in Swedish Friesian breed. Schukken *et al.* (35) and Suriyasathaporn *et al.* (38) inform that cows with retained placenta are more likely to develop mastitis than other animals. In our present study, some effect of retained placenta therapy with antibiotic on incidence of IMI could not be excluded. It is also possible that coliform mastitis can be more frequent in cows with retained fetal membranes.

This study found that cows with retained placenta were at 2.7 times higher risk of subclinical ketosis within the first week after calving but cows with normal puerperium were at higher risk of ketosis within the next weeks of the study. Similar proportions were observed in our previous study (24). Despite the fact that the number of cases of subclinical ketosis differed, there was no difference in the incidence of clinical mastitis between the two groups. The rate of mastitis was the same in both RP and NRP groups, and the highest incidence was recorded in the two groups during the first two weeks after parturition. This result suggests that subclinical ketosis does not increase the incidence of mastitis. Curtis *et al.* (7), Erb *et al.* (9), and Correa *et al.* (6) did not find a relation between ketosis and mastitis. According to Oltenacu and Ekesbo (31), ketosis increases incidence of mastitis ocurring in d 51-250 of lactation, but not in d 1–50 of lactation. Removal of the mammary gland, which prevented lactogenesis and attendant metabolic disease problems associated with lactogenesis, does not prevent a decline in neutrophil function in periparturient cows. However, neutrophil activity recovered more quickly following parturition in mastectomized cows as compared with intact cows (20). Kremer *et al.* (23) reported that experimental *Escherichia coli* mastitis in ketonemic cows was relatively much more severe as compared to nonketonemic ones. In the present study severity of the disease was not evaluated.

As conclusion it can be stated that under the condition of one herd and under the same management practices, susceptibility to mastitis depends on cow age, stage of lactation, and previous mastitis history. By taking appropriate actions, it might be possible to prevent the occurrence of the disease, or it may be economically advantageous in certain situations to cull the high risk animal.

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