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ORIGINAL ARTICLE

Evaluation of Mastitis Impact on Lameness and Digital Lesions in Dairy Cows

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Lameness;
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Abstract

Objective- This current study was done to find any correlation between clinical mastitis and lameness occurrence and incidence in dairy farms.

Design- This prospective field trial was done on a case control study basis. Cows were divided into two mastitis and control group and lameness recorded and compared in both groups.

Procedures- This current study was done during 9 months in a dairy herd with 800 milking cows. The mastitis scoring system was based on the International Dairy Federation definitions of mastitis severity from one to three. All cows were trimmed two times annually and also high locomotion score, lame and long toe cows referred for possible inspection and treatment. Records of sole ulcer (SU), white line disease (WLD), Toe Ulcer (TU), heel erosion (HE), digital dermatitis (DD) and interdigital necrobacillosis (INB) were assessed in this study. Data of the lesions up to three months after occurrence of mastitis was followed. 543 cows affected with mastitis were allocated to treatment and the same amount of the cows that didn't show any mastitis during past three month allocated to control group.

Results- Occurrence of mastitis reduce incidence of digital dermatitis significantly. Lameness except digital dermatitis were higher in mastitis group than control group ($P<0.05$).

Conclusion and Clinical Relevance- Mastitis can play a role in occurrence of claw horn lesions (CHL) and any control program of lameness in the herds with high incidence of CHL should precede with control program of other predisposing or causative factors of this condition. Mastitis besides other infectious causes as a predisposing factor can play a significant role on lameness.

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1. Introduction

Lameness is one of the most important diseases that make economic losses in dairy farms[1]and up to 79% of the cows reported as lame[2]. Pain is one of the most important causes of lameness that in many cases resulted in gait alterations that finally leads to different locomotion scoring systems[3]. Webster et al explained a useful understanding of lameness in dairy cows. As they proposed base to pathology of the lameness Claw Horn Disruption (CHD) is the main cause of conditions like sole ulcers and white line diseases. Replacing Subclinical Laminitis (SCL) or aseptic pododermatitis with CHD made controversies between the researchers[4]. Mechanical injury to solar corium consider as the main pathogenesis for sole ulceration and CHD. It is proposed that the lesions originate from inside the claw capsule and may not necessarily be associated with primary inflammatory or vascular changes, such as those associated with laminitis and resulting in sinking of the bone[5]. The physiopathology and pathogenesis of SCL were discussed by several researchers and more or less related to nutrition and metabolism[6,7]. In this 'classical' approach, a cascade of events starts with a metabolic problem i.e. ruminal acidosis and then the story develop down to the changes and lesions of the foot. Today much greater importance is attached to inadequate housing, foot care and parturition than to nutrition and feeding.

Claw horn lesions and sole ulcer as the most prevalent lesion in this group, were frequently reported in different studies and are among the most frequent causes of lameness in cattle[8]. Predisposing causes result in mechanical loading and/or metabolic/ enzymatic changes, which directly lead to failure of the suspension system of the third phalanx resulting in vascular injury as result of compression of the corium between P3 and the sole. Disturbances in the microvasculature lead to ischemia and hypoxia. Consequently, cellular proliferation and differentiation in the basal layers of the sole epidermis are interrupted, with subsequent development of a full-thickness horn defect (ulcer) that typically occurs in zone 4 (typical sole ulcer), zone 6 (heel ulcer) or zone 5 (toe ulcer) with exposure of the underlying corium[7].

Mastitis, Lameness and infertility are the most common diseases of dairy cows worldwide and undoubtedly have a significant impact on economic income and comfort of the cows. Mastitis defined as an inflammation of the mammary gland and affects economically by discarded milk costs, decreased milk yield, drug and vet costs, penalties because of increased cell count and higher culling rates. Mastitis is generally classified as clinical or subclinical based on the degree of inflammation, also depending on their pathophysiology can be classified into two groups, contagious mastitis and environmental mastitis. The major pathogens causing contagious mastitis are *Staphylococcus aureus* and *Streptococcus agalactiae*, *Corynebacterium bovis* or *Mycoplasma spp*. These bacteria have poor survival in the environment and generally spread from cow to cow during milking routines by the milkers' hands, towels, or the milking machine. Enviromental mastitis derived from the environment in which the cow lives and is caused by *Coliforms*, Environmental *streptococcus*, *Pseudomonas*, etc.

The most common metabolic conditions predisposing to claw lesions include rumen acidosis, laminitis, and conditions caused by coliform bacteria such as coliform mastitis[9,10]. Endotoxin release associated with these conditions can result in the formation of vasoactive cytokines, causing vascular changes as well as activation of metalloproteinases responsible for the breakdown of collagen, a major component of the suspension system of P3 [11]. that leads to sinking and rotation of the P3 and compressive damage of the solar and perioplic vasculature in the corium that lie beneath [11,12]. Displacement of P3 and subsequent damage to the underlying corium is compounded by mechanical loading secondary to claw horn overgrowth and unbalanced weight bearing.

Normal biomechanics of weight bearing may also play a role in the pathogenesis of sole ulcer formation. During normal locomotion, the lateral claws of the rear legs carry more weight as opposed to the medial claws, which results in accelerated horn growth, particularly at the heel. This overgrowth of the hind lateral claw leads to concussion of the solar corium and contributes to sole ulcer formation. Mechanical injury without primary inflammatory changes also occurs in thin-soled dairy cattle where the protective function of sole horn becomes inadequate, particularly on hard walking surfaces. The sole becomes thin and eventually results in a full-thickness defect [8].

In isolated perfused equine limbs, endotoxin at a clinically relevant concentration induced a distinct inflammatory reaction with intravascular and extravascular accumulation of leukocytes in the laminar tissue, similar to that seen during the developmental phase of laminitis. Therefore, endotoxin should be considered as a causative factor for some types of laminitis [13].

However presence of back arch, reported in bovine affected by lameness and metritis, had been observed to

occur in clinical mastitis cases and was therefore included in the scoring system[14].

The aim of this current study was to evaluate any possible lameness and foot lesion following clinical mastitis based on mastitis scoring system.

2. Materials and Methods

Dairy farm

This current study was done during 9 month (three seasons) in a dairy herd with 800 milking cows. The average daily milk production of the farm recorded as 36 liter during study. Cows kept in loose stalls with dry feces bedding and fed by a total mixed ratio (TMR). Cows separated by milk production level and parity in deferent barns. Mixed quarter somatic cell count (SCC) of the cows measured on a monthly basis.

Mastitis detection and scoring

The mastitis scoring system was based on the International Dairy Federation definitions of mastitis severity. Score one or mild was assigned if the milk was grossly abnormal but the affected gland was grossly normal and there were no systemic signs of disease; Score two or moderate was assigned if the milk was grossly abnormal and the affected gland was inflamed but there were no systemic signs of disease; and Score three or severe was assigned if the milk was grossly abnormal, the affected gland was inflamed, and there were systemic signs of disease[15]. Parity, Days in milk (DIM), affected quarter, mastitis score, treatments, response to treatment, period of treatment and possible recurrence of mastitis were recorded in this farm and needed information were extracted from farm data. Data of the cows that treated with antibiotics or anti-inflammatory during a month before study were excluded.

Lameness detection and scoring

Hoof care was done by professional hoof trimmers and veterinarians. All cows were trimmed two times annually and also high locomotion score, lame and long toe cows referred for possible inspection and treatment. Data recorded in hoof trimming unit and new cases recorded if the cow didn't have any lesion in the same digit or topographic region of the hoof during past three month. Records of sole ulcer (SU), white line disease (WLD), Toe Ulcer (TU), heel erosion (HE), digital dermatitis (DD) and interdigital necrobacillosis (INB) were assessed in this study. Data of the lesions up to three month after occurrence of mastitis was followed. Lesions in different zones of the hoof as previously reported were considered in this current study [16]. A gross lesion in zone three and two of the hoof considered as a white line disease (WLD), lesion on zone 4 of the hoof considered as sole ulcer (SU) and a lesion in zone 5 and one of the hoof considered as a toe ulcer (TU). A gross lesion in zone six considered as heel erosion (HE), a lesion in zone 10 considered as digital dermatitis (DD) and a lesion in zone 0 considered as interdigital necrobacillosis (INB).

Walking of the cows were scored on a monthly basis with a 5 point scale locomotion scoring system (LSS) as the score one refer to the most normal cow and severely lame cow got score five in this system[17]. Data of LSS were included a month before occurrence of mastitis till three month after its occurrence.

Design of study and Statistical analysis

Cow base to mastitis status allocated in two groups (mastitis and control groups). During 9 month study cows with one of the above mentioned grade of mastitis allocated to mastitis group and the same number of the cows that didn't show any record of mastitis at least three month before inclusion in the study allocated to control group. All data were analyzed by SPSS software version 21. Relationship between mastitis and occurrence of each lesion evaluated by Chi-square test. Effect of mastitis occurrence on locomotion scores were analyzed by Friedman test P values of 0.05 and less considered as significant.

3. Results

Total of 543 cows affected with different stages of mastitis and 543 normal cows included in this current study. Basic findings in mastitis cows appeared in table 1.

Table 1. Descriptive data of cows with clinical mastitis in this study

Mastitis score			Number of affected quarters			Parity		
Grade	**No**	**%**	**Quarters**	**No**	**%**	**Parity**	**No**	**%**
1	42	7.7	1	521	95.4	1	118	21.7
2	493	90.8	2	16	2.9	2,3	210	38.7
3	8	1.5	3	1	0.2	4,5	92	16.9
			4	4	0.7	>5	25	4.6

Incidence rate of clinical mastitis was 4 to 12% during the study. Incidence of SU, WLD, Toe Ulcer (table 2) HE, DD and INB (table 3) and lameness (Table 4) were recorded in Mastitis and Control groups.

As it appeared in the table 1 most recorded mastitis cases were in grade 2 (90.8%). Following these findings all grades of mastitis considered as mastitis positive cases and moved in other statistical and descriptive calculations.

Iincidence of WLD and SU in mastitis group (3.5, 2.9 percent) were higher than control (2.9 and 1.3 percent). However none of these differences were significant.

Table 2. Distribution of non-infectious lesions (Claw horn Lesions) SU, WLD and TU in Mastitis and Control group.

			WLD		SU		TU	
			-	+	-	+	-	+
Mastitis	-	No	530	16	539	7	543	**3**
		%	97.1	2.9	98.7	1.3	99.5	**0.5**
	+	No	527	19	530	16	543	**3**
		%	96.5	3.5	97.1	2.9	99.5	**0.5**

The size of the lesions was omitted and the presence of the lesions just was criteria for inclusion it in the study. As it appeared in table 3, DD significantly reduced in mastitis group (P<0.05) compared to control group however, HE and INB were higher in mastitis group (P>0.05) compared to control group.

Table 3. Distribution of infectious lesions (DD, HE, IBN) in Mastitis and Control group.

			HE		DD*		IBN	
			-	+	-	+	-	+
Mastitis	-	No	546	0	532	14	542	**0**
		%	100	0	97.4	2.6	100	**0**
	+	No	542	4	541	5	545	**1**
		%	99.3	0.7	99.1	0.9	99.8	**0.2**

Lameness data in table 4 are the sum of the infectious and noninfectious lesions in mastitis and control group. Because of more contagious nature of digital dermatitis, its data were excluded from sum of lesions and results show a significant increase of lameness in DD excluded lameness (Table 4).

Table 4. Distribution of lameness in Mastitis and Control group (DD excluded).

			Lameness		Lameness (DD excluded)*	
			-	+	-	+
Mastitis	-	No	508	40	508	**26**
		%	93	7.8	93	**5.1**
	+	No	498	48	498	**44**
		%	91.2	8.8	91.2	**8.1**

***: Significantly higher in Mastitis group (P=0.015)**

The average LSS in both groups recorded in figure 1. This average shows locomotion scores of all cows in all barns.

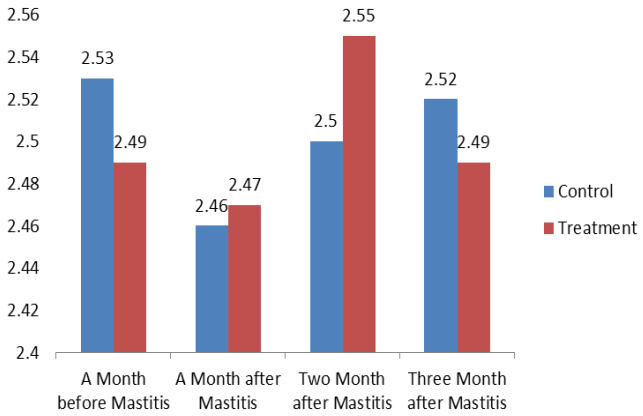

Figure 1. LSS variations during the study in Mastitis and Control groups

4. Discussion

The aim of this study was to evaluate possible correlation between clinical mastitis and digital lesions in cows. Lameness usually lasted for a long time and finding new cases always is very important for having a correct estimation of what happens in a herd. In current study just new cases 90 days after occurrence of a lesion were selected. Sole ulcer usually cure in 22-98 days. Selecting a 90 days period consider as cure time for old lesions and after this time cases recorded as new case[18].

All cows were observed up to three month after mastitis occurrence. Normal sole thickness and normal growth rate reported as (5-7 mm) and (5 mm/ month) respectively[19]. In this situation any lesions due to any ischemic pathogenesis will needs 30-45 days to be completely apparent in solar region after its occurrence and as previously mentioned its

average cure rate estimated as 50 days[18], this is why a 90 day after occurrence of mastitis consider for detection and follow up any lesion in the sole.

One of the common protocol in mastitis treatment is using antibiotics. DD and INB have an infectious entity and they could not affect occurrences of mastitis. it seems that occurrence of mastitis also basically doesn't have any effect on occurrence of these diseases. However, DD was reported lower in mastitis group compared to control group in this current study (table 3) that may be a result of possible antibiotic therapy in mastitis group for treatment of mastitis

However after omitting information of DD from total numbers of new cases, incidence of lameness recorded higher in mastitis group ($P<0.05$)(table 4). The most important reason for claw horn lesions known as ischemia in microcirculation of the lamellar region [9,10] that may occur following mastitis. Also changing in lying behavior of the cows following mastitis[19,20] maybe another cause of increasing claw horn lesions. However, in other studies herd size, management, breed, parity, season, reproduction diseases, metabolic disorders and diseases related to the udders are evaluated in lameness occurrence and reportedly breed, parity, season and herd size are effective on occurrence of lameness[21]. Intramammary injection of echerschia coli LPS (lipopolysaccharides) resulted in increase of inflammatory factors including IL-1β, IL-8 and TNFα in dairy cows[22]. Endotoxemia usually occurred with general inflammation and it seems that is important in many diseases that finally make laminitis in horses[23]. Endotoxins play an important role in many diseases including laminitis and abomasal displacements[24]. Cows with experimental endotoxemia didn't show any signs of laminitis[25].Intramammary infusion of LPS caused changes in both behavioral and physiologic variables in lactating dairy cows. Time spent lying, eating, and chewing cud were negatively correlated with physiologic responses in cows. Evaluation of behavior patterns may provide an ancillary measure, along with evaluation of physiologic variables, for monitoring well-being, clinical responses, and recovery from acute clinical mastitis[26]. LPS has no cytotoxic effect on epidermal and dermal cells isolated from hoof tissue, but impairs integrity of hoof explants. In addition, LPS led to an alteration of the lactic acid production in the lamellar tissue. However LPS can affect the integrity of the equine hoof tissue in vitro, endotoxins should be further explored for their contribution to facilitate the development of laminitis [27]. The DEPs (Differentially expressed proteins) were closely related to the occurrence and development of laminitis and the lipid metabolic disturbance may be a new pathway to cause laminitis in dairy cows. The results provide the theory foundation for further revealing the mechanism of laminitis and screening the early diagnostic proteins and therapeutic target[28]. Mechanical overload and tissue compression interfere with the perfusion of fluids and with nutrient supplies to horn producing tissue. Bioactive molecules derived from metabolic activity or systemic disease will impact on vascular walls and perfusion. These factors have the potential to change the diameter of the dermal vessels or to damage the endothelial wall. Of particular relevance is metabolic stress related to parturition, lactation or dietary problems resulting in metabolic disorders like ketosis or acidosis. Some factors such as histamine, lactate, endotoxin can directly damage the endothelial lining of the vessels and increase transvascular movement. Vasoactive factors such as serotonin or bradykinine will cause constriction of vascular walls with the result of reduced perfusion or reduced drainage form the capillary bed. The latter will result in increased transvascular movement and increased pressure inside the claw capsule [29]. Both reduced perfusion and alterations in the vessel themselves will impair horn production and finally provoke horn of inferior quality. A significant weakening of the horn capsule is a central result of subclinical laminitis. The consequence is an increased susceptibility of the claw to damage and lesions secondary to laminitis. [30].

For time spent lying contrasting results have been previously reported in cows affected by LPS-induced mastitis; while in some studies a clear reduction of the time spent lying was detected after the intramammary challenge[19,20], in others no differences were found[26]. However LSS recordings that may play a reflex of cow comfort and lying behavior didn't show any change in this current study ($P>0.05$) (Figure 1).

5. Conclusion

Result of this study shows that mastitis can play a role in occurrence of CHL in the herd and any control program of lameness in the herds with high incidence of CHL should precede with control program of other predisposing or causative factors of this condition. Mastitis besides other infectious causes as a predisposing factor can play a significant role on this condition. However further studies including other risk factors for mastitis and possible LPS follow up in larger populations may show more precise information.

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Conflict of interests

None

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چکیده

ارزیابی اثر ورم پستان بر رخداد لنگش در گاوهای شیری

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هدف: مطالعه حاضر به منظور ارزیابی اثر احتمالی ابتلا به اورام پستان برلنگش گاو شیری انجام شد.
طرح مطالعه: مطالعه بالینی به شکل آینده نگر بر اساس گروه های کنترل و درمان انجام شد.
روش کار: مطالعه در یک دوره ۹ ماهه در یک گاوداری شیری با ۸۰۰ راس گاو دوشا انجام شد. تشخیص اورام پستان به بکار گیری روش سه نقطه ای اسکورینگ ورم پستان حاصل از فدراسیون بین المللی اورام پستان انجام شد. تمامی گاوهای مورد مطالعه حداقل دو بار در سال سم چینی شدندو گاوهای با اسکور بالا، سم بلند و لنگ نیز برای درمان های احتمالی به باکس سم چینی ارجاع گردیدند. اطلاعات مربوط به زخم کف سم (SU)، بیماری خط سفید (WLD)، زخم پنجه (TU)، ساییدگی پاشنه (HE)، درماتیت انگشتی (DD)، و نکروباسیلوز بین انگشتی (INB) در این مطالعه وارد شدند. اطلاعات مربوط به جراحات تا سه ماه بعد از رخداد اورام پستان دنبال شد. تعداد ۵۴۳ گاو مبتلا به اورام پستان در طول مطالعه شناسایی شدند و در گروه درمان قرار گرفتند، تعداد مشابهی گاو که در طول سه ماه گذشته مبتلا به اورام پستان نبودند نیز شناسایی شدند و در گروه کنترل قرار گرفتند.
نتایج: رخداد اورام پستان منجر به کاهش موارد بالینی درماتیت انگشتی در گروه درمان شد و سایر موارد لنگش بدون در نظر گرفتن درماتیت انگشتی در گروه درمان بیشتر رخ دادند ($P<0.05$).
اهمیت بالینی: اورام پستان می تواند نقش کلیدی در رخداد جراحات بافت شاخی سم داشته باشد و هر برنامه کنترلی در مورد این جراحات باید دربرگیرنده عوامل مستعد کننده به آنها از جمله اورام پستان باشد. اورام پستان در کنار سایر عوامل مستعد کننده می تواند نقش معنی داری در رخداد لنگش در گله های شیری داشته باشد.

کلمات کلیدی: لنگش، گاوشیری، اورام پستان، جراحات بافت شاخی