

Observations on Teat Lesions

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Veterinarians and dairy producers have been required to deal with teat lesions for many years. Teats traumatized by being stepped on, barbed wire cuts, etc., have always been a problem. Virus lesions from pseudo cow pox have also been present for many years. The advent of machine milking added another possible source of teat trauma, due to the mechanical effects of the milking machine. In about the last twenty-five years, virus lesions due to herpes viruses have also come into play as a major problem. The widespread use of teat dipping opened another area -- the possibility of chemical burns and injuries when products were improperly compounded, mixed or applied.

Lesions involving the teat barrel do not generally directly cause mastitis, but do interfere with the milking process and may cause secondary problems. Lesions which affect the teat end and orifice consistently result in increased mastitis problems because of interference with the protective effect of the teat orifice, which is a major barrier preventing bacteria from entering the gland.

Significance

The type and extent of lesion that is necessary to produce an increased intramammary infection is an important consideration. For many years, cows' teats were described as having "pulled-out ends" or prolapsed sphincters. It was generally considered that these lesions were due to milking machine trauma, primarily high vacuum, and progressed to ulcers and scab formation. This phenomenon was researched by Sieber and Farnsworth in the late '70s. An important aspect of this phenomenon was discovered almost by accident. Sieber was collecting teats at a local packing plant for histological section when they began to process beef cows. Out of curiosity, he began to examine teat ends on beef cows and discovered that beef cows had the same everted, pulled-out, prolapsed teat ends that were being attributed to the milking machine in dairy cows. Subsequently, cows in hand-milked herds with no chemical use were examined and also shown to have this same type of lesion. Histopathology of these teat ends showed

these rings to be excess keratin (one medical school pathologist referred to them as callouses). This finding raises a fundamental question. A callous may not be normal, but does it increase bacterial infection rates in quarters where it is present? It can be theorized that this may have some effect on teat closure, or that some form of callouses (particularly on pointed teats) are quite rough and may have spiculite projections that make it hard to clean the teat orifices at milking time resulting in greater bacterial contamination. A system of classification of different types of teat end lesions was developed. A study was conducted to examine the correlation of various types of teat lesions with the prevalence of intramammary infections, as well as the milking machine and milking practice factors, in about 5,000 cows. Findings of this study showed no statistical difference in the prevalence of infection among normal teats with the various hyperkeratotic lesions, regardless of type (rough or smooth) or degree. Teats with erosions or scabs on the teat end did show a significantly higher prevalence of infection. It was also observed that the callous-type lesions did not progress from mild to severe to erosions or scabs as a natural process. Heifers seem to develop a degree of callous within the first 6-8 weeks of lactation, which then persisted at that level, regressed to some degree during the dry period, and returned to about the same level the next lactation.

The correlation of lesions with other factors was also examined. Milking machine factors such as vacuum level and pulsation ratio, that were varying but within what are considered the normal ranges, did not appear to affect the degree of lesions. Milking practices that resulted in extended milking time -- such as no preparation, no pre-milking stimulation or over-milking -- did increase the degree of lesions. One of the most significant correlations was with milk production, which also tends to extend milking time. Later work conducted by Graeme Mein at Wisconsin showed an increase in what he termed hyper-keratosis related to the compressive load of the milking machine liner.

There has been for many years a general assumption that any changes in teat end condition result in an increase in infection. However, there does not appear to

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be any significant data that callouses or hyper-keratosis of teat ends alone (without erosions or additional lesions) result in increased intramammary infection.

Milking Equipment

Milking equipment malfunction has traditionally been considered a cause of teat lesions. There is some evidence that hyper-keratosis and transient edema may result from this source, however, severe lesions such as abrasions and scabbing of the teat orifice do not appear to be easily produced by milking machines unless the malfunctions are extremely severe, such as extremely high vacuum or nearly total pulsation malfunction.

Teat Dipping

The widespread adaptation of the practice of post-milking teat dipping has also resulted in a significant additional source of teat irritation. A variety of problems can exist. The incorrect use of concentrated products such as udder washes or cleaners for teat dip can result in severe lesions within one or two applications. Improper mixing, freezing or simply production accidents with these products can also produce a variety of lesions. More frequently, however, problems with teat dips are associated with drying of the teat skin rather than ulcers and erosions. When a teat dip is a source of a problem, changing dips will result in rapid resolution of the situation. However, many situations involving lesions, particularly in viral infections, tend to resolve themselves with time -- so an improvement resulting from using a new teat dip may be coincidental. Unless the dip can be shown to have chemical or pH abnormality or shown to produce lesions when it is used again at a later time, it is difficult to be sure of a cause-and-effect relationship.

Climatic Factors

Teat lesions can also be produced by weather factors. Teat ends or entire teats can be affected by freezing or frostbite. Animals with edema of the teats and udder are more subject to this effect and need more protection than normal animals. Teats and teat ends are also more subject to freezing when wet with such things as teat dip or a drop of milk remaining on the teat end after milking. Problems occur primarily when animals are exposed to both wind and cold temperatures right after milking, such as in milking parlors where cows exit directly outside and must travel to other housing. The practice of eliminating post-milking teat dipping during cold weather is often used to avoid cold damage. But if this practice continues for any length of time, an increase in infections of contagious pathogens such as *Staph aureus* can result. Numerous methods are used for managing teat dipping in cold weather, but dipping the teats and then blotting off the excess dip about 15

or 20 seconds before the animals are turned outside appear to be a practical solution to this problem.

Pseudo Cow Pox

Viral infections have also been a source of teat lesions. Pseudo cow pox virus, recognized for many years, typically produces vesicles that rupture and form circular lesions which heal in a week or two. Lesions of the teat barrel are most common, resulting in difficulty in milking due to discomfort of the cow. They may also affect milk let-down. Lesions may also affect the teat end, sometimes causing secondary intramammary infections. Herds that experience an outbreak of this virus usually exhibit a significant portion of the herd showing lesions over a short period of time and the problem is rather rapidly eliminated. In some instances the lesions may not be typical and may affect only teat ends, making diagnosis somewhat more difficult. An occasional herd also experiences a slow spread of the virus. There have been instances in large herds where this virus has resulted in a continuous problem, with a few animals infected at all times over a long period of time as animals began to lose immunity after the initial infection. However, most herds which experience an outbreak one year do not have a problem again the following year.

Diagnosis of pseudo cow pox can be made by examining fresh scab material from lesions with electronmicroscopy or looking for inclusion bodies. Serology has also recently become available and may be a more practical method of making a diagnosis when adequate lesions for examination are not available.

Herpes Virus Lesions

There are two herpes viruses which have been involved in teat lesions. **The herpes mammalitis virus, or herpes II, has been recognized in Minnesota since the early '60s.** The fact that the herpes IV or DN599 virus also produces similar lesions was recognized in the mid '70s. These viruses appear to have similar epidemiology and produce clinically similar lesions, although they are serologically distinct. These viruses have the reputation of producing very severe erosive lesions. Some cases may result in severe ulceration of most of the skin of the teat. Lesions are very slow to heal, frequently resulting in secondary intramammary infections. A syndrome is also seen, primarily in heifers, where the skin of much of the teat becomes hard and is sloughed. This is usually associated with edema around the time of parturition. Normally these heifers cannot be successfully milked, develop secondary mastitis and end up being eliminated from the herd very quickly.

The range of lesions due to these viruses, however, is quite wide -- from animals that simply seroconvert with no lesions, to cows that become hard milkers but

show no physical lesions, to animals that show mild lesions that eventually heal, to animals which show very severe lesions.

Recent histological studies have discovered virus inclusion bodies in areas near the teat canals in cows that have simply become hard milkers with no evidence of lesions.

In most herds when a problem is first noticed, a substantial number of animals, particularly heifers, may be involved. But, in subsequent years the number of animals involved and the severity tends to decrease to the point where only an occasional animal is involved. However, a few herds experience an ongoing problem with heifers and may also have cows involved year after year. Some of these herds may have 5-10% of the animals with lesions most of the time. There have never been any factors identified which appear to be responsible for these differences in herd response.

There appears to be a seasonal relationship to the lesions in the herpes herds. Problems with lesions are primarily seen between November and April in the Minnesota and Wisconsin area. Some herds have experienced animals with problems in warm weather, but in most cases the seasonal pattern is rather distinct. There has been speculation that due to the better skin condition in the warm months, the virus is less able to infect. There is also some evidence that herpes has an affinity for body parts with a lower temperature, such as the teat. It can be noted in general that fewer teat lesions from most causes appear to be seen during the warm weather period.

The method of viral transmission has not been established. When one considers the general biology of herpes virus along with the epidemiology of the disease in herds, it appears likely that carrier animals exist. The method of cow-to-cow spread is also not clear. British literature suggests insect transmission, but the evidence that the problems are seen primarily during the times of insect absence in the upper Midwest suggests that other methods may be more important in this area. The fact that a heifer usually shows a problem one or two weeks after calving suggests spread during the milking process. However, blood samples taken at breeding time have shown rather high titers in heifers at that age. Whether the recrudescence that occurs with the human herpes simplex occurs with these viruses also has not been determined. As previously noted, titers in heifers at breeding time suggests early exposure. Instances have been observed when heifers showed titers at an early age that then decreased or disappeared, but the animal later showed lesions with a high titer present at the time of calving. This could simply be early exposure from which the animal did not develop lasting immunity, or it could be a latent herpes infection that was reactivated with the stress of calving.

Diagnosis of herpes infection was first attempted by virus isolation. However, these attempts are usually unsuccessful (although isolation from experimental infections appears to be quite successful). The use of serum neutralization titers appears to be more successful and practical. There is no solid data to determine significance of titer levels, but based on herd epidemiology, herpes II titers of 1:16 or higher and herpes IV titers of 1:20 or higher are an indication of exposure to the virus. Herpes II titers of greater than 1:256 are seldom seen. The herpes IV titers often exceed 1:750 and may reach 1:1250. Classically, viral diseases are diagnosed by demonstrating an increase in titers from paired serum samples, one taken early in the course of the disease and the other one to two months later. However, the most practical way to use serum titers for diagnostic purposes in this area appears to be to obtain samples from several (four or five) animals showing lesions of various durations, being sure that some cases are at least two weeks or more in duration. At this time it does not appear practical to make specific animal diagnoses with this method, but the fact that the virus is in the herd can be established. The two herpes viruses do not appear to cross-react to any extent, so serology testing for both should be done for diagnostic purposes.

The prevention and treatment of these lesions has been an area of major frustration for veterinarians and producers. It is difficult to determine the effectiveness of various procedures without the use of controlled studies because of the variability in numbers and severity of animals infected from year to year. The use of serum obtained from animals that had shown lesions was at one time thought to be effective, however, experience over a period of time suggests that it is not particularly effective in prevention -- plus it is somewhat impractical because of the timing necessary. The use of various teat dips has often been suggested for prevention, however, observations suggest that there is no significant difference in the ability of products to prevent infections, since lesions occur in herds using most different types of teat dips.

Milking heifers first has also been suggested. This also may be a good practice for other reasons such as mastitis control, but again in general does not appear to prevent problems. If recrudescence is occurring in some herds, this would explain the lack of efficacy of either teat dips or milking order.

There is some evidence that reducing parturient edema in heifers may help reduce the severity of the problem in these animals. The use of diuretics to control edema has met with some success in reducing the skin sloughing and necrosis which occurs in some animals. A rather drastic prevention measure that appears to be meeting with some success is the use of seasonal calving. Since problems are normally

not seen from May to October, calving heifers during this time reduces the chances considerably. The poor economics of this approach, however, make it a rather drastic step since heifers have to be maintained longer and calved, in most instances, at an older age than desirable. However, if the alternative is losing the heifer to skin sloughing and mastitis, it may be economically feasible.

Treatment and healing of lesions is another area where many different approaches have been attempted. Again, it is difficult to evaluate the effectiveness of products. The use of products which result in drying has been successful in some instances. The methaline blue dye-type horse wound products have been helpful in some instances. Some producers prefer to bandage teat ends with protective ointments. The use of film forming-type teat dips has also been somewhat successful. The use of anti-viral drugs is often considered. This is usually not attempted because of expense and milk residue issues. There is also some suggestion that in humans these drugs need to be used before lesions appear. In humans with various herpes infections, the patient knows that the disease is coming on and can start the drugs ahead of time, a practice which appears to be more effective in prevention of lesions. There have been some instances of use that appear to be beneficial, however, there are no controlled data in this area.

The use of vaccines for prevention appear to be a reasonable possibility. There has been some limited experimental success but there has been a failure of some simple experimental products in this area. Currently there is no product available which has shown consistent success in cows. Development of a successful vaccine will require considerable research and development.

One of the biggest problems with teat end lesions is the incidence of secondary mastitis. The continuation of teat dipping can be of some help in preventing secondary infections as well as preventing exposure to environmental organisms. This is an area where bandaging or sealer-type teat dips may have some use.

The presence of herpes virus in a herd can be determined by observing classical lesions where they occur and by serology where lesion type is not definitive. Since there is presently no good control or treatment system, the value of a definitive diagnosis may be questioned. However, if a producer knows the problem is of viral origin, he is more likely to accept that he has to "live with it" and not continue to search futilely for solutions such as milking equipment changes that result in unnecessary expense.

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