

Non-Infectious Lesions of the Bovine Teat ¹

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In addition to infectious lesions of the bovine teat are those caused by traumatic events, chemical injury, environmental conditions, insects and the milking machine. Teat lesions, regardless of cause, are frequently colonized by staphylococci and *Streptococcus dysgalactia*. Consequently, high new infection rates and increased numbers of clinical cases of mastitis are common sequelae in herds where teat lesions are prevalent.

TEAT TRAUMA

Traumatic lesions of the teat are most commonly the result of the cow stepping on her teats or wire cuts. They are a troublesome problem for the veterinarian as well as the dairymen. Histologically, the teat wall contains an abundance of elastic connective tissue which provides for expansion and contraction of the teat as it fills and evacuates milk in the lactating cow. The near constant movement associated with these physical dynamics of the teat combined with milking preparation procedures, and milk collection complicate the normal process of healing.

The dairyman's challenge is in getting cows with teat lesions milked. Because these lesions are generally painful and cows resist preparation and milking procedures they are difficult if not hazardous to milk.

A further complication is mastitis. Teat lesions are readily colonized by bacteria and thus serve as an important reservoir of infection. Udder preparation cloths, hands of the milker and milking machine components facilitate the transfer of infectious

organisms between quarters of the same cow and can be responsible for cow to cow transmission as well. Emphasis on milking hygiene procedures becomes crucial in control of new infections whenever teat lesions are present.

Depending on severity and the period of time prior to discovery, teat lacerations may be repaired surgically. Fresh superficial lacerations of the teat skin (within 12 hours of occurrence) in which the vascular supply has not been significantly damaged have the best prognosis. These are generally amenable to surgical closure. If, on the other hand, such lesions go unnoticed for a couple of days and become heavily contaminated cleansing in mild disinfectant solution and removal of the skin flap tissue are likely the best therapeutic approach.

Teat lacerations which extend into the teat cistern are of greater concern and generally carry a more guarded prognosis. The exposed edges of the cistern lining must be sutured using a suture pattern that will turn the edges inward creating an impervious seal. If this is not achieved healing cannot occur and draining fistulas develop. The teat wall muscle layers and the skin may be closed separately or individually. Most advise intramammary and/or systemic therapy for 4 to 5 days as a precaution against the development of mastitis. A protective bandage allowing access to the teat end for milking is recommended. Milk should be retrieved from the gland through the use of teat cannulas.

Pastured cattle have a lower incidence of teat trauma than confined cattle. Housing factors of primary importance are associated with the amount of

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space available to the cow for resting and rising. Further, individual cow characteristics and conformation increase the potential for teat trauma in some cows.

CHEMICAL INJURY

Teat lesions resulting from chemical injury most often occur as a result of the application of a defective teat-dip product. Iodophor based teat dips, possibly because of their widespread use, are frequently the offenders. However, the problem has been observed with quaternary ammonium dips, chlorhexidine-based dips, dodecyl benzene sulfonic acid and hypochlorite teat dips. Lesions appear as dry, roughened, proliferative regions around the teat end which are usually discolored by the teat dip. This discoloration may be present on 40 to 50% of the teats in the affected herd. Changing to a dip with a better conditioning properties may result in the rapid improvement of teat skin health.

In the case of iodophor dips, problems have arisen secondary to inadvertent freezing of the dip on-farm or in transit. When these solutions freeze they separate sending emollients to the bottom of the container and leaving excessive amounts of the active ingredients in a layer suspended above. The subsequent application of this concentrated iodine causes teat irritation and lesions. Depending on the degree of insult these lesions can be quite severe.

The mistaken use of a concentrated udder wash solution in place of a teat dip causes a more severe type of lesion and often affects up to 80 to 90% of the herd. The lesion is characterized by scab formation over the distal portion of the teat exposed to the dip solution.

On occasion, the addition of lime or other chemicals to the bedding material will result in teat skin and udder irritation. Lesions tend to be worse on the lateral sides of the teats and may extend onto the sides of the udder.

TEAT CHAPS

Teat chapping is a common environmental injury problem in the more temperate regions of the world where climatic conditions favoring dampness and cooler temperatures prevail. Activities associated with milking such as udder preparation, the milking

process and post-milking teat dipping all exacerbate chapping problems. Chaps usually occur as horizontal cracks in the teat skin. Serum exudates from these cracks result in the formation of linear scabs. The surrounding teat skin may appear dry or leather-like and flake.

The primary significance of chaps is that they are readily colonized by staphylococci and *Streptococcus dysgalactia* and thus constitute a threat to individual cows affected as well as the herd. Drying of the teats and udders before cows exit the milking parlor, particularly during inclement weather conditions, is an important preventive measure. Further, the use of teat dip products containing hygroscopic skin softening agents such as glycerin or lanolin are helpful in controlling chapping problems.

FREEZING OR FROST-BITE

Initially frozen teats will appear reddened or pale. If severe the lesion progresses to the state where a scab forms over the distal half of the teat. In time, usually several days, this scab will loosen and fall off exposing a raw denuded teat end. As a second scab begins to develop the duct becomes occluded. Milking becomes difficult and may require opening of the streak canal surgically.

In less severe cases, scab formation does not occur and cows become receptive to milking after only a few days. Cows immediately exiting the milking parlor with wet teats (from dip or milk) to areas with inadequate protection during cold weather may develop frost-bite on the teat ends. Freezing of the droplet on the teat end confines the lesion to the teat end orifice area. The result is as described above for frozen teats.

Treatment consists of attempts at keeping the teat duct patent and preventing the development of mastitis. Severely affected cows may need to be culled. Drying of the teats and udder prior to exit from the milking parlor and providing adequate wind breaks and shelter for milking cows is essential. The suspension of teat dipping procedures should be considered during extremely cold weather.

SUNBURN

Severe reddening and drying of the teats and udder are observed in sunburn conditions. When severe blisters may form. The application of moisturizers, ointments, and salves to the affected areas are advised for treatment.

PHOTOSENSITIZATION

This condition occurs when photodynamic agents are eaten in their preformed state. Upon exposure to sunlight, the unpigmented skin areas develop an erythema and edema which results in severe lesions that most commonly appear on the lateral aspects of the teats and udder. These lesions are highly susceptible to secondary bacterial infection.

MILKING MACHINE INDUCED TEAT LESIONS

Malfunctioning milking equipment can result in damage to the teat. This damage may be by direct trauma to the teat or indirect, occurring over an extended period of time through the induction of degenerative changes in the teat. These changes in teat tissue health are primarily associated with circulatory disturbances. Proper pulsation is essential for the circulation of blood and lymph in the teat. When normal circulation is disrupted teat end health diminishes.

Direct damage to the teat may be caused by excessive milking vacuum, inadequate pulsation, and careless use by the operator. Subcutaneous hemorrhages in the teat epithelium and prolapses of teat duct tissue are possible consequences with severe malfunctions of milking equipment or its use.

INSECT INDUCED TEAT LESIONS

Summer mastitis is an acute suppurative disease of the non-lactating mammary gland. First described in Europe, in recent years it has been reported in the USA and other countries as well. It occurs sporadically throughout the year in Europe with annual incidences in England and Wales estimated to be around 2% of heifers and dry cows. In the United States some estimate incidence of 5-6% on certain farms during the summer months.

While some questions remain, in both the USA and Europe epidemics of summer mastitis have been coincident with periods of greatest fly challenge.

Further, data indicate that effective fly control reduces disease incidence. These findings support the possibility of insect involvement. European data suggest that biting flies are responsible for the initial damage to the teat end and implicate the cattle fly, *Hydrotaea irritans* as the infection vector in summer mastitis.

TEAT ORIFICE ABNORMALITIES

Producers are often concerned about everted teat-ends or teat-end erosions. In many cases, these abnormalities are neither eversions or erosions but rather excessive pigmentation of the teat skin. British investigators studying the same cows at 10-week intervals over an 18 month period found that 22 percent of teats had orifice abnormalities. They reported that abnormalities began as white ring extending about 2mm from the orifice. In this study, as ring formation progressed, radial cracking occurred, and in some severe cases, hypertrophy progress to scab formation, actual erosion, and secondary infection. The excessive pigmentation was more prevalent during the first 120 days of lactation, after which there was a tendency for regression. These workers concluded that some increase in skin pigmentation should be expected in high-producing machine-milked cows. Bacteriologic data was not reported.

Minnesota researchers reported a 22-herd, cross-sectional study in which they investigated the relationship of teat-end lesions and intramammary infection (a cross-sectional study, as opposed to the British longitudinal study, may be thought of as a "snapshot" or one-look in time). They classified teat ends as:

1. normal
2. smooth chronic rings
3. rough chronic rings and
4. acute teat orifice lesions

Only those teats in the acute category (orifice with ulcerative or hemorrhagic appearance with or without scab formation) had a concurrent increased incidence of intramammary infection.

Few producers or veterinarians would disagree with the desirability of maintaining as healthy a teat-end and teat orifice as possible. Of particular interest in the Minnesota study was the finding of markedly

different rates of teat end abnormalities among herds. For example, in one herd less than 5 percent of teats had chronic rough lesions, whereas almost 50 percent of teats in another herd had similar lesions. Questions remain regarding both the cause and importance of teat-end pigmentation.