Beecham Mastitis Series

Teat skin lesions and mastitis

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Healthy teat skin is highly desirable for the welfare, comfort and health of the cow, for aesthetic reasons (the production of wholesome milk) and for the health and comfort of those involved with milking.

Teat lesions are usually invaded by mastitis pathogens especially *Staphylococcus aureus* and *Streptococcus dysgalactiae*. Reservoirs of infection in the vicinity of the teat orifice will have a marked adverse effect on mastitis control measures. Some teat lesions originate from the action of a single bacterial, viral, chemical or physical agent but several of these may be involved by the time a veterinary surgeon is requested to examine a teat. Lesion healing may be assisted more by advice on husbandry changes than by therapy.

NON-INFECTIOUS TEAT CONDITIONS

Milking machine damage

Excessive or fluctuating vacuum levels, faulty teat cup liners, incorrect pulsation ratios and other faults attributed to insufficient maintenance and/or careless use of milking machines have been shown to cause teat damage. Damage is caused either directly by trauma or indirectly via degenerative changes resulting from circulatory disturbances in the teat tissue (Fig. 1)

A teat stretches by 30 to 50% on entering a teat cup liner and teat length does not increase thereafter. During the peak flow stage of milking, the teat is stabilized inside the liner by friction between the teat and the liner mouthpiece and its barrel, assisted by pressure inside the teat sinus. When peak milk flow is over, teat sinus pressure falls and the liner mouthpiece beomes the main site of friction between the teat and the liner. From this time some movement of the teat wall along the liner barrel occurs and an increase in liner mouthpiece vacuum is likely, especially in large bore liners. If garter marks at the teat/udder junction and teat tissue congestion are seen when clusters are removed (Fig. 2), the milking machine must be under suspicion. Each time the teat cup liner collapses, it applies a compressive force to the teat. A high milking vacuum combined with a relatively low pulsation chamber vacuum can result in bruising of the teat end by the slapping action of the liner.

The period of reduced milk flow occupies about 50% of the unit-on time, so teats can be abused by a faulty machine for three or more minutes at each milking time. Haemorrhages in the teat epithelium, and haemorrhage and necrosis of the dermal papilla of the streak canal with a loss of keratin have been seen *post mortem*, after teats have been abused by milking machines.

Teat orifice abnormalities

Lesions at the teat orifice have been described as teat canal eversion, teat canal prolapse, prolapse of the meatus, eversion of the meatus, blackspot and teat orifice erosion. The term 'erosion' should only be used when epithelium has been lost.

It is normal to see a 2 mm wide white ring around each teat orifice of machine milked cows (Fig. 3). The first stage of a teat orifice abnormality occurs when this ring undergoes hypertrophy, keratinization and radial cracking (Fig. 4). Progression leads to increased hypertrophy, secondary bacterial infection, scab formation (Fig. 5), eversion of the distal teat canal and eventually orifice erosion (Fig. 6).

Incorrect milking machine function can produce teat orifice abnormalities. A prospective study of teat orifice condition of 701 cows over an 18-month period in Somerset by P. G. Francis & J. Sumner (unpublished observations) showed that around 22% of teats had teat orifice abnormalities. Static and dynamic tests on the milking machines, carried out at ten-week intervals, revealed no serious mechanical faults and the operators on the survey farms were judged to be competent. Abnormalities were common in high-yielding cows and commoner during the first 120 days of lactation than later in lactation. A similar high prevalence in early lactation has been recorded by Sieber (1980) during a survey in

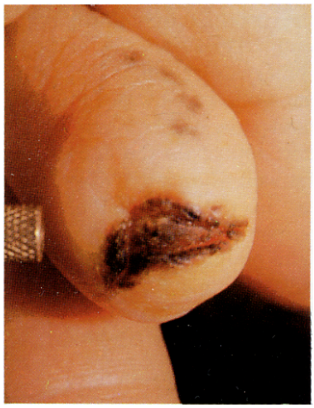

Fig. 1. Subcutaneous haemorrhages and teat orifice lesion associated with milking machine damage.

Fig. 2. Teat chaps and garter mark.

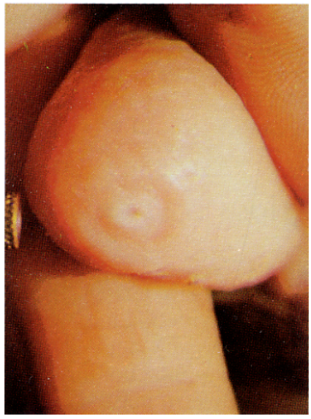

Fig. 3. Normal teat orifice with white ring.

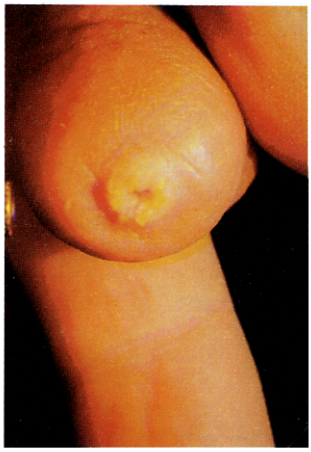

Fig. 4. Slight hypertrophy of tissue surrounding teat orifice.

Fig. 5. Scabbing and cracking of tissue surrounding teat orifice.

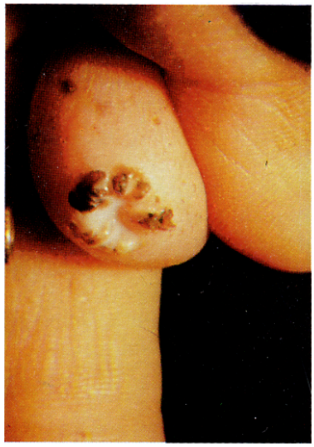

Fig. 6. Tissues separation and erosion at teat orifice.

the United States. An accurate inventory of abnormal teat orifices is therefore necessary before the prevalence of orifice lesions can be classed as abnormal.

Teat chaps

Skin cracks usually initiated by milking machine action can be aggravated by environmental factors to form chaps (see Fig. 2). The condition is common when adverse weather conditions follow turn-out in spring and also during grazing of autumn forages and kale. Linear lesions appear on the teat barrel near the teat/udder junction and extend transversely around the teat. They are often more severe on the anterior aspects of the fore teats and on the posterior aspects of the hind teats. Healing is often delayed by further adverse enviromental conditions and secondary bacterial invasion.

Blackspot

This term was used by Gold (1943) to define a teat orifice lesion infected with *Fusiformis necrophorus*. Blackpox appears to be an alternative description of the same lesion but associated with *Staphylococcus aureus* infection. These conditions are in the author's view, synonymous with the well developed orifice abnormality described above.

Chemically-induced teat lesions

These are caused by the application of faulty or unsuitable products—mainly teat dips. The time interval between the use of the product and the appearance of lesions will vary according to the degree of abnormality or faulty mixing of the product. Many animals show lesions, all of which appear at more or less the same time.

Interactions between chemical products and the environment can cause teat skin lesions:

1) When incompletely slaked lime has been added as a drying agent to cubicle bedding.
2) When chemicals have been used too frequently or too generously to disinfect cubicle litter.
3) When the sand used as cubicle litter contains large amounts of lime material which reacts with residual hypochlorite teat disinfectant on teats.

Photosensitization

This condition may arise when photodynamic agents are eaten in their preformed state during grazing or when phylloerythrin (a normal end product of chlorophyll metabolism) accumulates in the body due to impaired biliary excretion.

Erythema followed by oedema occurs in unpigmented skin on exposure to sunlight. Lesions thus appear on the lateral aspects of the teats as well as on other sites. Exudation may occur in the oedematous areas and secondary bacterial infection is common. Irritation can be intense and cows traumatize the lesions by licking. Parasites of the genus *Stephanofilaria* have been associated with summer sores in Norway (Bakken, 1980).

Udder impetigo

This condition is presented as small pustules, 2 to 4 mm in diameter, which usually appear on the skin at the base of the teats, but the lesions may spread to other parts of the teats and udder. *Staph. aureus* can be recovered from the lesions, and spread from cow to cow takes place during milking times. A large proportion of the herd can become infected and the infection can spread to the hands of the machine operators.

INFECTIOUS TEAT CONDITIONS

Papillomatosis

Warts are aesthetically unattractive and are likely to interfere with milking, when trauma may disturb the cow and cause blood to enter the milk. Secondary infection of traumatized warts will predispose to mastitis.

A papovavirus virus is the cause of warts on cows' teats. The warts may be sessile or pedunculated, the former may be round and flat or resemble a rice grain, the latter may be simple or branched. Self cure is common and immunity following an attack persists for at least two years. Several distinct strains of virus have been identified so that immunity to one strain (or wart shape) may not confer immunity to other types.

Autogenous vaccines prepared from wart tissues have been used to speed the regression of lesions. Older warts appear to contain more virus than younger ones and intradermal injection is reputed to confer a stronger immunity than subcutaneous administration. Warts on teats respond less well to vaccine therapy than warts on other areas of the body.

Cowpox

A clinical syndrone resembling cowpox is common but the cause is usually parapox virus

or bovine herpes virus 2. Outbreaks of true cowpox are rare.

It is generally assumed that the virus enters through teat skin injuries and several stages of lesion development can be observed. After an incubation period of about five days an erythematous area appears on the teat and this develops into a firm, raised papule. Vesicles form and change into pustules with pitted centres (Fig. 7). Rupture is followed by the development of a thick red tenacious scab 1 to 2 cm in diameter. Scabs are frequently removed during the milking process so red ulcerated areas are the commonest presenting signs. The lesions spread rapidly throughout the herd. Healing occurs in two to three weeks although secondary bacterial infection may delay resolution. Immunity in recovered animals is solid, conferring protection for several years.

Farm staff who have recently received smallpox vaccination have been responsible for spreading a cowpox-like condition among cows.

Pseudocowpox

This condition is common in dairy herds and is caused by a parapox virus. The morbidity rate is usually 100%, but at any given time only 5 to 10% of animals may show lesions. The disease is a zoonosis, and may present as nodules on the hands of milkers.

The incubation period is about six days after which localized erythematous and oedematous areas appear on the teats (Fig. 8). The lesions at this stage are painful. Vesiculation rapidly occurs and progresses to the formation of a pustule which easily ruptures. A thick scab soon forms over the lesion. Granulation begins from the centre of the lesion and the primary scab is often shed after about 10 to 12 days, leaving the classic raised 'horseshoe' or 'signet ring' lesion (Fig. 9). Lesions may coalesce to form an area which resembles a ringworm lesion (Fig. 10). Healing usually takes four to five weeks and typically leaves no scars. A typical, incomplete resolution may result in multiple fissuring of teat skin (Fig. 11).

Increases in morbidity are often associated with inclement weather in spring and autumn. In addition, flare-ups may occur when newly-calved home-bred heifers, or purchased animals are introduced into the herd. Recovered animals have little immunity and this is of relatively short duration. The condition therefore usually persists in a small percentage of cows with periodic flare-ups occurring when non-immune animals become infected and act as virus multipliers.

Some outbreaks of pseudocowpox have been associated with coexistent orf in sheep on the same farm. Pseudocowpox occurs in cows having no contact with sheep, and sheep are refractory to cowpox virus. However, mouth lesions have been produced experimentally in lambs by inoculating fluid from milkers' nodules, and lesions on calves' muzzles have been produced experimentally by injection of orf virus. Cross-infection may therefore be possible.

Bovine herpes mamillitis (BHM)

Bovine herpes virus 2 (BHV2) causes painful oedematous teat swellings about seven days after teat skin infection. Vesicles, 1 to 3 cm in diameter develop and are accompanied by pyrexia (Fig. 12). After about 24 h the vesicles rupture exposing the congested dermis from which much serous fluid exudes (Fig. 13). Lesions may coalesce to leave the teat largely denuded of epidermis (Fig. 14). The exudate dries and a flat smooth scab is formed (Figs 15 and 16). The scabs are shed after about three weeks but in milking cows resolution is rarely uncomplicated. The action of milking removes the scabs, secondary bacterial infection is common and many cases of mastitis are the customary accompaniment.

Outbreaks generally occur between August and November, and the appearance of lesions on the teats of most, or only a few, cows in a herd depends on the residual immunity from previous encounters with the virus. Virus can survive within the host for long periods. Lesions have re-appeared when corticosteriods were administered six to 17 months after experimentally-produced BHM lesions had healed. Virus could be recovered from nasal swabs at this stage. This may be a mechanism whereby BHM lesions can occur from time to time in a small number of cows in a herd. An examination of epidemiological data in the United Kingdom suggests that insect transmission may be the means of spread between herds. This is supported by a recent report (Scott & Holliman, 1984) of the detection of serum neutralizing antibodies in pregnant heifers which had no direct contact with cows known to have been previously infected.

DIFFERENTIAL DIAGNOSIS

The clinical signs of the diseases mentioned above are usually sufficiently characteristic to

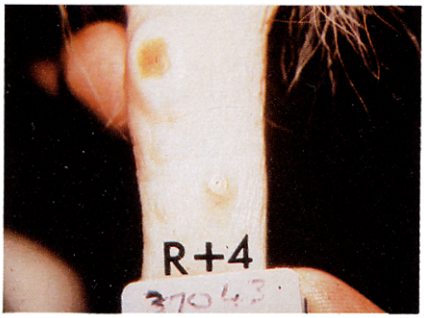

Fig. 7. Early lesion of cowpox.

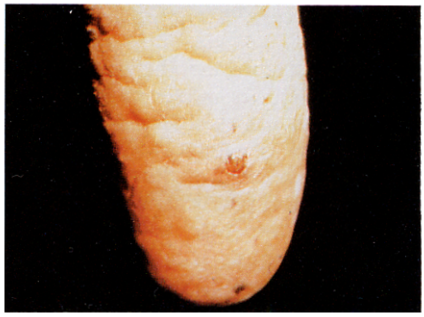

Fig. 8. Early lesion of pseudocowpox.

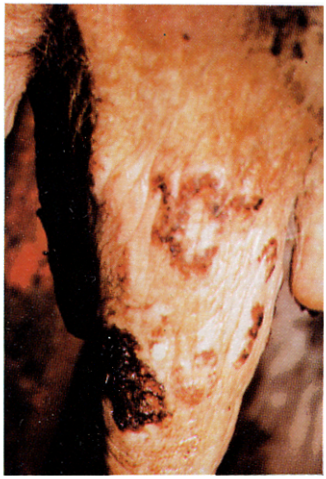

Fig. 9. Signet ring appearance of pseudocowpox lesion, with BHM lesion visible on the same teat.

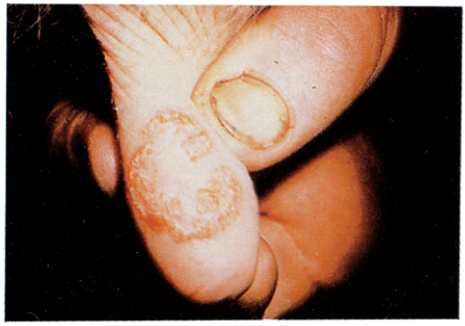

Fig. 10. Large pseudocowpox lesion showing central healing.

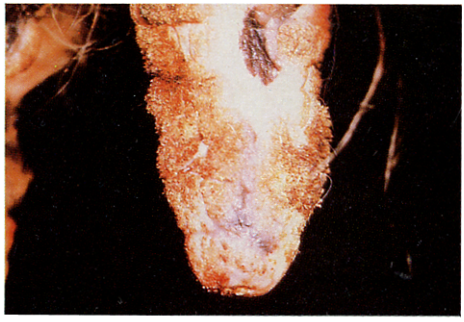

Fig. 11. Fissuring following pseudocowpox infection.

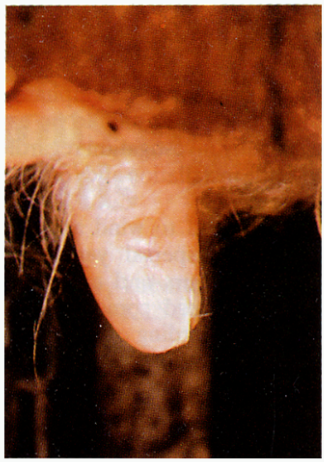

Fig. 12. Vesicle of early BHM.

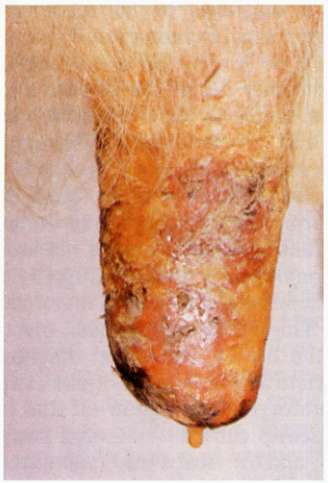

Fig. 13. Acute weeping BHM lesion.

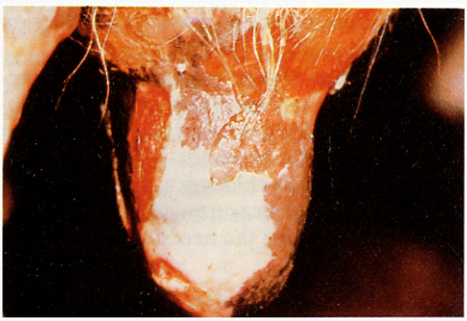

Fig. 14. Skin denudation in BHM.

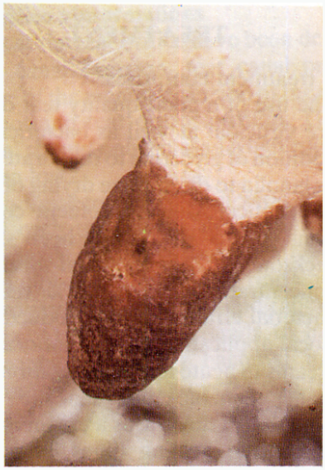

Fig. 15. Thick, extensive scab of BHM.

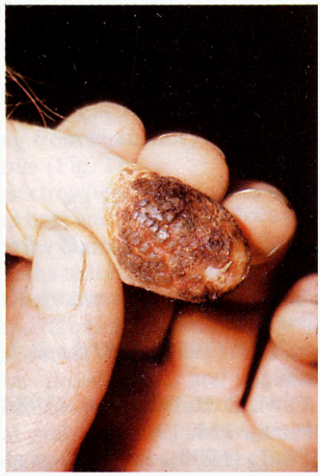

Fig. 16. Healing BHM lesion.

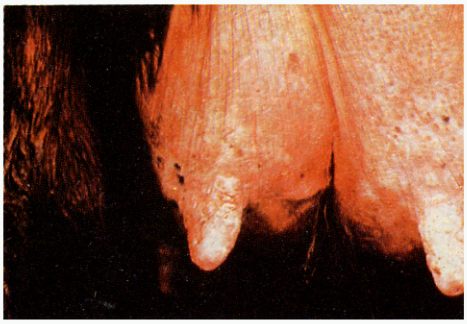

Fig. 17. Vesicles of foot-and-mouth disease.

enable a diagnosis to be made during a thorough clinical examination. Typical lesions may only be present on a few cows, mixed infections may occur, the disease may be at different stages in different cows, and husbandry and enviromental factors may modify the appearence of lesions. It is thus important to examine the teats of many rather than few cows during the investigation of outbreaks of teat lesions. Foot-and-mouth disease causes vesication of teat epithelium (Fig.17) together with systemic signs, as well as muzzle and foot lesions. This condition, however, must always be on the list of differential diagnoses and appropriate action taken if doubt exists.

Laboratory confirmation of the specific virus diseases can be obtained if swabs from vesicles and/or scabs are submitted to a laboratory for electron microscopy or tissue culture. Early, rather than mature lesions should be selected for this procedure.

TREATMENT

Local treatment comprises:

1. The use of a good quality teat dip. Most are virucidal as well as bactericidal. The addition of glycerine to rehydrate teat skin or lanolin to discourage drying of teat skin is beneficial. High emollient concentrations (up to 30%) have been shown by Jackson (1971) to speed healing, but concentrations above 15% reduce the bacterial efficacy of the product.
2. Local application of antibiotic/antiseptic creams to acute lesions. A combination of malic, benzoic and salicylic acids is available in the UK as a cream or a lotion to shorten healing times by gently removing scabs and encrustations.
3. Intramammary antibiotics should be considered as a mastitis prophylaxis. Large scale use in herd epidemics is costly because of the necessity of withholding from sale milk from treated cows.

Systemic treatment is of limited general value, but may be beneficial on specific occasions:

1. Antihistamines can be given to cases of photosensitization and to very early cases of BHM.
2. Autogenous vaccines are useful in udder impetigo, but the benefit is unlikely to persist for longer than six months.
3. Vaccines achieve a poor response when used against teat warts.

Management changes are usually as important as medication.

1. The potential damage of the milking machine to initiate and aggravate teat damage cannot be over emphasized. Static and dynamic tests should be carried out early in any investigation regardless of the age of the installation and/or the history of recent testing.
2. Cows exposed to wind and rain in collecting or dispersal yards around the milking parlours and at self-feed silage faces may need some protection with Yorkshire boarding or plastic netting.
3. Grazing plans may have to be changed if weather and ground conditions are unfavourable.
4. Materials used as litter may have to be changed because sawdust from some hard woods, short wheat straw and sharp sand, when used as cubicle litter can aggravate teat lesions. The treatment of cubicle litter with disinfectants, at best a substitute for good management, may make teat lesions worse.

Most treatments are palliative and symptomatic, with management changes used as supportive measures. The daily tasks of the person milking the cows are made infinitely harder during teat lesion epidemics. He or she deserves supportive measures, additional help at milking times being the necessary minimum.

ACKNOWLEDGEMENT

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