Viral Skin Diseases In Cattle

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Skin Conditions Caused by Viruses

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A. Skin Lesions Caused By Pox Viruses

There are seven pox viruses that can cause skin lesions in cows: (1) cowpox virus; (2) vaccinia virus, which is the smallpox virus vaccine; (3) variola virus, which is the smallpox virus; (4) pseudo-cowpox virus, which may be a separate virus or may be either: (5) bovine papular stomatitis virus or (6) orf virus; and (7) the "Neethling" agent which causes lumpy skin disease.

Cowpox - may be caused by the cowpox virus; the vaccinia virus, which is a strain of cowpox virus which has been serially transferred on the sides of calves for the production of the smallpox vaccine; and variola virus, which is smallpox.

The lesion is found on the udder and is the classic pock lesion. It begins as a papule that forms into a pustule. A red areola forms around the lesion. The center of the lesion sinks, leaving a doughnut-shaped ring of raised edematous tissue with a central pit, the so-called "Umbilicated pock". The udder lesions dry up and heal rapidly (two weeks), but because of the trauma caused by milking, the teat lesions may become raw, painful 2-3 cm erosions. These lesions are difficult to differentiate from other teat lesions.

True Cowpox - occurs rarely and may not be a primary disease anymore. Cows develop solid immunity to it once they have been exposed.

Pseudocowpox - may be caused by the pseudocowpox virus, bovine papular stomatitis virus, or contagious eczema virus. Actually, there may not be a true pseudocowpox virus, but pox viruses that cause disease elsewhere may cause the pseudocowpox lesion on the udder.

The lesion begins as a papule which forms into a very small vesicle which, as it breaks down, leaves a raw area. As this heals and forms a dry scab, the microvesicular reaction spreads forming a ring or U-shaped lesion which may involve a significant portion of the teat. Umbilicated pocks may or may not form. Usually the pseudocowpox lesion tends to be larger than the cowpox lesion and is not manifested by the umbilicated lesion. These are the viruses that cause milker's nodules.

Most cows in a herd will be affected. There is little immunity to reinfection and there is no cross immunity between it and cowpox.

Scrapings may be beneficial in diagnosing pox lesions. Intracytoplasmic eosinophilic inclusion can be seen in such scrapings. Differentiating one pox virus from another is exceedingly difficult and only a few research laboratories have the ability to do so.

B. Bovine Papular Stomatitis

Bovine papular stomatitis (BPS) is a pox virus in the paravaccinia subgroup. Lesions begin as a 2-15 mm erythematous macule. The central area becomes elevated as a papule is formed. The periphery remains hyperemic and the center becomes grey and may slough, leaving a crater. The course of the lesion is usually one week or less.

These lesions are usually found in one of several areas including the muzzle, nares, gums, buccal papillae, floor of the oral cavity, behind the incisor teeth, and the tongue. This virus seems to have a predilection for the oral cavity and is transmitted when exposed to a scarified surface. The teat lesion may be associated with calves nursing with oral lesions, but they are not confined to such cases. One hallmark of classical BPS is the lack of systemic signs, and lesions are often incidental findings during a physical examination for some other reason. It is more commonly seen in 2-6 month-old calves, but the immunity developed is of short duration and cattle of any age may have the disease. The lesion may be transient or there may be repeated crops of lesions over several months.

Bovine papular stomatitis virus has been recently implicated in "rat-tail" syndrome in Texas feedlot cattle. This syndrome is characterized by chronic weight loss and poor performance. These cattle do well for the initial feeding period but by 60 days they develop diarrhea, salivation,

suffer weight loss, and lose the hair from the end of their tail. The morbidity is usually 1% but may be as high as 10%. One interesting difference between the oral lesions found in this disease and those of classical BPS is that lesions are usually found on the dorsal tongue, whereas classical BPS very rarely presents with dorsal tongue lesions. Some of these "rat-tail" tongue lesions are chronic and may have a Dermatophilus (Streptothricosis) infection superimposed. The "rat-tail" syndrome does seem to be associated with a pox virus infection, but a causal relationship to BPS virus has not been proven.

C. Lumpy Skin Disease

Lumpy skin disease is caused by the "Neethling" agent which is in the pox virus group (Allerton in herpes). The disease is currently confined to South Africa. As the virus is transmitted by biting insects (mosquitoes), the disease is seen during warm weather.

The skin lesions usually develop with the second fever spike (seven to ten days after the first fever spike). The lesions are firm circumscribed ½-5 cm nodules which may coalesce. The nodules are usually intracutaneous, although they occasionally extend into the subcutis. The number of nodules seems to correlate with the severity of the disease. The disease may be mild with no fever and only a few nodules, or it may be severe with systemic manifestations such as inappetence, enteritis, respiratory disease, lymphangitis, lymphadenitis, and the cow may have hundreds of nodules.

The nodules may appear anywhere on the body. Some lesions persist as hard lumps for months, while others resolve rapidly and completely. However, most undergo necrosis and a process called separation. Separation occurs when the central necrotic core drops out, leaving a raised but deep ulcer. The open sore leads to secondary bacterial infection and lymphangitis and lymphodenitis.

Most of the animals recover but the immunity is short-lived. Inapparent infections are probably common.

D. Bovine Herpes Mammillitis

Bovine herpes mammillitis (BHM) is a herpes virus infection usually confined to the udder area. As with other herpes infections, animals may become carriers and shed the virus when stressed. Mammillitis seems to be most severe in younger animals, especially recently calved heifers. This disease is always seen between July and December in the Northern Hemisphere (very interesting fact).

Several different types of lesions can be seen with BHM. First, vesicles may form. They may be quite large, up to 1.5 cm, and may coalesce. Once the vesicle ruptures, the erosion forms a scab quickly---except those on the teat where trauma caused by milking keeps the lesions from healing. The second type of lesion usually begins as a circumscribed 1-2 cm area which, within 24 hours, progresses quickly from normal skin to an edematous plaque to a dull red plaque which becomes a dry, dark-red scab. This scab can result in a deformed teat after it heals. "Thimbling" may also occur to a teat. These teats lose the skin in one piece, and the sloughed piece of skin is thimble-shaped. The lesions heal slowly if the cow is milked and the teat may be raw for two months or more. A fourth change that is associated with BHM is swelling of the entire teat. The teat may become acutely swollen and resemble a traumatic lesion. The necrotic skin dries and deforms the teat. Again, this may take months to heal. Lastly, the necrotic plaques of skin associated with BHM may not be restricted to the udder, but may extend up the legs and into the perineum area. Calves may also have lesions on their mouth and head, especially if they nurse from an affected cow.

Bovine herpes mammillitis seems to be frequently subclinical in the United States. Dr. Robert Dellers (personal communication) found 20% of the dairy cattle in New York to have a titer. One breeding stud had an incidence of 11% positive bulls. An occasional diary herd may have up to 40% positive animals. One study in England reported a 19½% incidence of positive titers. The titers are often low (1:4 to less than 1:100).

The morbidity is variable. Lesions may be found on only a few cows in the herd or may involve up to 100% of the cows. Herd outbreaks usually last two to seven weeks, however an individual cow may recover in two weeks if the lesions aren't traumatized by milking or secondary bacterial infection does not develop.

The mode of transmission is not definitely known, but flies are suspected. The virus will not penetrate intact skin. Milking machines will not transmit the virus if the teat skin is intact. The virus is killed easily by a disinfectant-type teat dip. Herds with BHM often also have pseudocowpox, but the relationship is not well understood.

Diagnosis of BHM can be made from a scraping showing multinucleated syncytia and intranuclear eosinophilic inclusions, as opposed to the intracytoplasmic inclusions of the pox virus group. Virus isolation may be easily made by inoculating the fluid aspirated from a vesicle or edematous swelling into a bovine cell culture (aspirate fluid, cap needle with rubber stopper, and ship at 4° C). Electron microscopic examination of the fluid can also be useful in differentiating BHM from foot-and-mouth disease, vesicular stomatitis, and pox viruses.

Experimentally, one intramuscular injection of live, unattenuated virus will protect against teat and udder lesions. This type of planned infection may be of value in an outbreak. The virus can be obtained directly from a lesion or from the viral isolation culture.

E. Bovine Papilloma Virus - Warts

Typical warts caused by bovine papilloma virus are fibropapillomas. The virus initially induces a fibroblastic reaction, followed by epithelial hyperplasia. Typical warts usually occur in animals less than 2 years old.

The typical lesions are hairless, pedunculated, protruding growths with thick, firm fronds. All of the skin adnexa are replaced by the fibropapilloma. Typical lesions regress spontaneously in one month to one year and rarely recur after regression. It is usually edemic in an area, but how the

virus is introduced to new herds is not well understood. Herd outbreaks have been reported following the use of a dehorner and following herd vaccination.

The major differential diagnosis for typical warts are atypical warts. Atypical warts are papillomas without the dermal fibroplasia. The virus found in atypical warts is indistinguisable from bovine papilloma virus, but it differs in its ability to induce tumors in experimental animals, in the antigenic makeup of the tumor tissue, and in the host antibody response to the virus. Cattle with atypical lesions are usually older cattle (3-16 years) but occasionally young animals have both types of warts.

Atypical warts are low, flat, circular lesions with a broad base and with delicate papular fronds. Often there is hair growing between the fronds since the skin adnexa are not replaced. They do not form stalks and often coalesce, covering a large area. This is frequently the type of wart on teats.

In contrast to typical warts, atypical warts usually persist for at least two or three years. They may regress but will often recur either in the same place or in a different place. New cases may occur in any age animal. Morbidity can be 17-37% of the herd in any one year. These warts will not respond to a wart vaccine. It is important to distinguish between the two types of warts, since the therapeutic approach to treatment and prognosis differs.

F. Bluetongue

Bluetongue is caused by an arbovirus; thus, it is usually seen in the late summer and early fall. The principle vector is *Culicoides variipennis.* This species of Culicoides prefers cattle goats, and wild ruminants over sheep. Other possible vectors are the Aedes mosquitoe, sheep kids, and lice.

The disease in cattle begins first as a mild lameness, which may or may not be followed by fever. Lesions form on the nose and mouth. In mild cases these may be no more than a red superficial lesion which is ill-defined; in more severe cases the lesions may be extensive. As these lesions resolve, a severe coronitis may be seen with swelling and cracks along the coronary band and necrosis between the claws. The lesions may be so severe that the whole hoof sloughs. Cracks may develop on the skin of the withers or neck. The hair drops out and the skin may then slough. This can extend down the back and even to the switch of the tail, resembling ergotism. There also can be cracks and ulcers on the teats. Bluetongue lesions often begin with pityriasis, but go on to have secondary bacterial infection or are damaged further by solar radiation.

Frequently cattle have inapparent infections. Only 2-5% of infected cattle show signs, but 25% of cattle in so-called bluetongue areas have positive antibody titers. (It is extremely rare to find a ruminant with a positive bluetongue titer in the Northeastern United States.) Occasional serum samples will be anticomplementary, making the complement-fixation test (CFT) invalid. Bluetongue is also subclinical in goats and in many wild ruminants, being consistently severe only in sheep and deer. Immunity in cattle seems to be poor. The virus can be found in the blood of subclinical cases for at least four months and the clinical disease may be chronic and last up to six months. Except in severe cases, there is a spontaneous recovery, and the major losses are due to a decrease in weight gain and milk production.

G. Bovine Virus Diarrhea

Bovine virus diarrhea (BVD) is caused by an unclassified virus. It is usually found clinically in cattle 6 months to 2 years old. Most cases seem to occur between December and June (?).

Oral lesions are typically red erosions due to the loss of superficial layers of epithelium. They are found on the lips, gums, hard and soft palate, and buccal papillae which become red and blunt. The lesions may be large and they may coalesce, but usually are small and few. Foot lesions may consist of interdigital erosions and ulcers, which leads to a severe necrosis or coronitis with deep fissues in the skin. Founder is a common sequel. Hyperkeratotic skin lesions can occur in the inguinal area, perineal area, axilla, neck, shoulders, and inside the ears.

BVD is often subclinical. At least 50% of cows have antibody titers. The chronic cases will often have chronic skin changes as well as poor growth, weight loss, and diarrhea.

H. Malignant Catarrhal Fever

Malignant catarrhal fever (MCF) is caused by a herpes virus. It usually occurs in cattle less than 2 years old. Its natural hosts are sheep and wildebeest--transmission to cattle is associated with contact with these animals (sheep in the USA and wildebeest in Africa).

Skin lesions begin with areas of erythema which may develop into moist, red necrotic areas of skin. These areas become firm but remain covered with tufted hair. Lesions are often found in the perineum, axilla, groin, heals, and occasionally on the back, neck and shoulders. Teats may have a purple "blotched" pattern on them with occasional small encrustations. On palpation the skin often feels firmer than normal. Skin lesions never occur alone, however the extent of involvement of other systems varies.

The virus can be isolated from white blood cells, but is extremely difficult to do. A characteristic vasculitis will be seen on histological examination of any involved tissue (gut, brain, skin, lung, etc.). Most animals with MCF will die, but in recent Colorado outbreaks some of these animals survived. Similarly, an occasional animal will survive here in the Northeast (possibly those infected with the African strain of virus).

I. Vesicular Stomatitis

Vesicular stomatitis (VS) is caused by a rhabdovirus. It usually occurs in warm weather and may be insect-borne, but the method of transmission is not clear.

The lesion begins as a raised, flat, pale papule only a few millimeters in diameter. These papules become hyperemic and develop into 2-3 cm vesicles that coalesce. The vesicles may rupture and become superficial erosions. These erosions may still have an epithelial flap, which helps to differentiate the lesions from other erosions such as those that occur in bovine virus diarrhea. The lesions heal in one to two weeks, unless there is secondary bacterial infection or trauma caused by milking. Usually the viremia persists longer than the lesions, however there are no systemic signs. Lesions usually are confined to the oral cavity but can occur on the feet and teats.

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Malignant Catarrhal Fever—Case Histories

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Incidence

- Sporadic single cases in North America, Europe, Australia, New Zealand
- Enzootic form in Africa (African or wildebeest type?)

Clinical Picture

- 3—8 week incubation period
- nasal—ocular signs: mucopurulent nasal discharge; muzzle and buccal necrosis; dyspnea; panophthalmitis; blindness
- nervous signs, extreme depression, incoordination, weakness, tremors
- lymphadenopathy
- G—I, signs from constipation to profuse diarrhea
- persistent high fever is characteristic
- contact with sheep

Case Histories

Farm A: 135 head purebred dairy herd in Central Pennsylvania; show cow purchased January, 1978; she