Necrotic Vulvitis in Feedlot Heifers

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Necrotic vulvitis may be defined as a morbid inflammation of the external genitalia, characterized by hyperemic swelling, necrotic sloughing and large eschar formation which varies from focal lesions to complete obliteration of the area.

Necrotic vulvitis of feedlot heifers is not a new condition. The necrotic inflamed vulvas of heifers were first recognized in four different herds in Marshall County, Kansas in 1897. Contagious vulvitis was the name given to the condition by Dr. Sesco Stewart of Kansas City, Missouri, in 1898. In 1900, Dr. C. Miller, Ottumwa, Iowa, presented a paper on "Infectious Ulcers of the Vulva of Cattle."

Although the condition is found primarily affecting the vulvas of feedlot heifers, the anus of bulls and steers and on rare occasion the prepuce of the latter. However, the posthitis is not considered to be of an infective nature. This condition has also been called "anovulvitis" and posthitis" or simply "anovulvitis." Dr. R. S. Heer chose to call it "external anovulvitis" in his article because he did not believe that the condition was infective.

Cattle of all ages are susceptible, but it is found primarily in feedlot heifers and consequently is confined to the midwest and other areas where cattle are finished for market.

This condition must not be confused with granular venereal disease of cattle which is of a more chronic nature and involves primarily the vagina. The latter condition is characterized by hyperplastic lymph nodule formation in the mucosa of the vaginal wall.

PATHOGENESIS

The initial cause of the condition is not completely understood, but it is believed that it is caused primarily by trauma. Especially pig bites, frostbite, and manure matted tail switching. Spherophorus necrophorus invades the devitalized area from the feces during defecation or the muddy, filthy soil of the lots contaminate the devitalized area. This contamination is especially prevalent due to the flaccidity of the vulva when the heifers are in a reclining position. Trauma as the primary cause of necrotic vulvitis is exemplified by the absence of such a condition in Europe where cattle and swine are always separately housed and raised. Also, no mention of such a condition has been observed in European text books and literature.

It is also possible that the teeth of swine may introduce Spherophorus necrophor-
us when they bite the heifers, because this organism has been isolated from the intestinal tract of animals and appears to be a normal inhabitant of animal tissue. The coprophagous habits of swine in cattle feed lots would enhance the presence of this gram-negative, nonmotile, anaerobic organism in the oral cavity of swine. This correlates with mammary gland abscesses of lactating sows in which Sphero phorus necrophorus has been isolated. These abscesses are believed to have been caused by “needle” teeth punctures of the mammary gland when the pigs are nursing.

The infective nature of the disease is believed to be due to the increased virulence which is acquired by organisms in the initially infected heifers and consequently the normal role of an opportunist played by Spherophorus necrophorus changes to a more invasive and virulent organism. This theory and the failure to reproduce the disease by animal inoculation eliminates the viral or hidden primary causative agent of this affliction. Sphero phorus necrophorus is usually not the primary etiological agent in any condition or in any animal. It is usually thought of as being an opportunist. Its growth and multiplication occurs only where the oxygen is reduced as it is an anaerobe. This can occur in any area of the animal's body if the tissue is devitalized and can invade other areas by direct extension. This extension of the condition is probably due to the edematogenic exotoxin and the necrotogenic endotoxin produced by Sphero phorus necrophorus. It is a self-perpetuating organism, yet it extends only as long as the animal's defenses can be repelled, thus the localized nature of the condition.

The parallel or subsequent invasion of the affected area with aerobic bacteria such as the staphylococci and streptococci would enhance spherophorus infection symbiotically.

PREDISPOSITION

Necrotic vulvitis shows no respect for the quality of its host. It occurs in both top quality and poor grades of heifers. However, despite its lack of respect for the quality of cattle the predisposition of this condition is always the same. It occurs only in cattle that are confined to a restricted area — the feed lot. It occurs especially between October and the middle of March. Cold, damp, unsanitary conditions are present when the heifers are afflicted. The cattle are crowded, in direct contact with each other and are unable to exercise freely. It has been shown experimentally that guinea pigs which are scorbutic and suffering from necrobacillosis have promptly recovered from both conditions following vitamin C therapy. Therefore, the amount and type of green feed included in the rations of the heifers may be an important factor in necrotic vulvitis. To further strengthen this theory is the disappearance of the condition when the cattle are placed on pasture.

GROSS LESIONS

The lesions vary from small areas of necrosis of the ventral commissure to necrosis of the labia, clitoris and extending to the urethral orifice. Some lesions will show laceration caused by swine bites. The lesions are generally from 1-5 cm. in diameter, but may completely cover the external genitalia and extend dorsally to the ventral portion of the anus.

The ulcerous lesions are raw; they show jagged edges, and active hyperemia is present at the borders. The eroded surfaces are moist, dirty brown, often show hemorrhages, and the older lesions usually exhibit heavy eschars. There is prominent superficial sloughing of the necrotic tissue. The odor is characteristic of Spherophorus necrophorus.

In the acute cases, which are not common, a partial to complete stenosis at the vulvo-vaginal junction has been observed. The vaginal mucosa has not been reported as being affected in any of the cases.

MICROSCOPIC LESIONS

The ulcers at first are structureless, then the sub-epithelial tissue shows evidence of progressive necrosis. The base and rim show hyperemia and hemorrhage. In acute cases the lesion would show

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lamellar tissue reaction which can be attributed to the action of the toxins of spherophorus. The gram-negative, pleomorphic, organisms can usually be seen. Sections taken early in the condition show numerous leucocytes and cellular debris throughout the lesion. As the lesion progresses there is slow healing by the formation of granulation tissue which becomes scar tissue and there is also a marked increase in the number of lymphocytes and giant cells around the base and periphery of the ulcer. The scar becomes covered by regenerated epithelium.

SYMPTOMS AND HISTORY

Necrotic vulvitis is usually first noticed in three or four heifers within a relatively large group of heifers in the feed lot. There is a reddened edematous swelling of the lower portion of the vulva which progressively extends dorsally to the lower margin of the anus and laterally to the skin surrounding the vulva. The color is red at the onset, but becomes yellowish to a dirty brown as the condition progresses. The skin of the area sloughs leaving a denuded, ulcerous area. The surrounding tissue becomes cheese-like in consistency and presents a disagreeable odor, characteristic of Spherophorus necrophorus. In severe cases all portions of the vulva and anus are sloughed. Two to three days after the first few heifers are affected, a high percentage of the remaining heifers in the herd may show a swelling and reddening of the external genitalia as described above.

In severe conditions, which are rare, an elevation of temperature, anorexia, marked depression, rapid shrinkage and difficult urination may be seen.

TERMINATION AND SIGNIFICANCE

In mild cases there is a spontaneous recovery, after about 10-12 days following the first symptoms, when the cattle are removed from the feed lot to the pasture or if the swine and cattle are segregated. (The theory of a Cavitaminosis as a predisposing factor may be of significance in the movement of cattle to pasture). The more severely afflicted individuals may take several weeks or longer to completely heal.

Necrotic vulvitis rarely kills. Death could occur, however, if a complete stenosis of the external genital orifice took place. Uremia or peritonitis or both would be the probable cause of death.

Mortality, however, is not the concern of the farmer and consequently the veterinarian in cases of necrotic vulvitis. It is the loss of production, the shrinkage and decrease in quality of the farmer’s product that is of significance.

REFERENCES