Review
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Review of Pododermatitis Circumscripta (Ulceration of the Sole) in Dairy Cows

Sarel R. van Amstel and Jan K. Shearer

Sole ulcers are among the most frequent causes of lameness in dairy cattle. They are found most commonly in the hind lateral claw, are frequently bilateral, and have a high rate of reoccurrence. The pathogenesis of sole ulceration is primarily based on mechanical injury by the 3rd phalanx to the corium, basement membrane, and basal layers of the sole epidermis as a result of failure of the suspensory apparatus in the claw. The main pathways in the failure of the suspensory system include inflammatory (dermal vascular changes followed by disruption of keratinocyte proliferation and differentiation caused by local and systemic mediators) and noninflammatory (hormonal and biochemical changes in the peripartum period resulting in alterations of connective tissue in the suspensory system) pathways. Sole ulcers tend to occur in specific locations; the most reported site is the junction of the axial heel and sole. Other locations include the apex of the toe and the heel. Varying degrees of lameness may result, and the most severe are seen with complicated cases in which ascending infection affects the deeper structures of the claw. Pathologic changes at the ulcer site include dyskeratosis and dilated horn tubules with microcracks. Vascular changes include dilatation and thrombosis of capillaries with ''neocapillary formation.'' Areas of dyskeratosis may remain for as long as 50 days at the ulcer site. Treatment includes corrective trimming and relief of weight bearing. Complicated cases may require surgical intervention.

Key words: Corrective trimming; Dairy cows; Etiopathogenesis; Presentation; Sole ulcer.

The 1st accurate description of sole ulcer was given by Rusterholtz in 1920.[1] The condition was and still is commonly referred to as Rusterholz ulcer. Later, the condition was given the scientific designation *Pododermatitis circumscripta*.[1] Different terms have been used to describe sole ulcer depending on the specific location of the ulcer within the area of the sole (Fig 1), such as toe ulcer (Zone 5), sole ulcer at the typical place (Zone 4), and heel ulcer (previously called necrotic heel tract and under run heel; Zone 6).[2] Sole ulcer is defined as a full-thickness break in the epidermis, which may present clinically in different stages. The 1st stage is hemorrhage of the corium without a break in the horn (*Pododermatitis circumscripta hemorrhagica*). The 2nd stage is perforation of the corium or dermis through the full-thickness break in the epidermis (*Pododermatitis circumscripta perforata*). Trauma, granulation tissue formation, and ascending infection may follow, resulting in the term *P. circumscripta complicata or purulenta profunda*.[3]

Sole ulcer is the most important cause of lameness in dairy cattle and occurs worldwide where dairy cows are kept on hard or concrete flooring in tie stalls or free stalls.[4] Hemorrhage of the sole is regarded as an early sign. In one study, 94% of primiparous and 66% of multiparous cows had sole hemorrhages at trimming 2–4 months after calving.[5] Friesian dairy cows seem to be most susceptible to sole ulcers.[6] In a study in the United Kingdom, 13.9% of 927 lame cows surveyed were affected by sole ulcers.[1] In a recent survey in North America extending over a 1-year period in a large dairy herd with 3,221 lactating cows, the incidence of sole ulcers was 21.5%. The highest incidences were recorded during the summer and early fall (September, 4%.; October, 3.4%) and occurred in cows with 3 or more lactations (van Amstel and Shearer, unpublished data). Another study found that in lactation 1, 20% of cows had sole ulcer occur in 1 foot and 29.7% had them occur in more than 1 foot, whereas in lactations 2 to 9, 23.5% of cows had sole ulcer in 1 foot and 24.7% had ulcers occur in more than 1 foot.[6] The presence of sole ulcer in any given lactation increased the risk of sole ulcers in the subsequent lactation.[6]

Numerous factors have been implicated in the development of sole ulcers, mostly those factors associated with laminitis, including systemic disease, high carbohydrate or protein rations, roughage quantity or quality or both and ratio to concentrates, time of year, cow comfort, familial and genetic predisposition, and calving. Other risk factors include age, claw overgrowth, sole thickness, interdigital dermatitis, claw size, lack of exercise, milk yield, body weight, and structure of the legs and bones.[6,7]

Pathogenesis

The 3rd phalanx is suspended within the claw horn capsule by the laminar corium and a series of collagen fiber bundles that stretch from the insertion zone on the surface of P_3 to the basal layer of the epidermis via the basement membrane.[8] The interface between dermal and epidermal components is the interdigitating dermal (sensitive laminae) and epidermal laminae (horn leaflets). The result is that P_3 hangs within the claw capsule and weight is transferred as tension onto the wall of the claw capsule.[8,9]

From the Department of Large Animal Clinical Sciences, College of Veterinary Medicine, University of Tennessee, Knoxville, TN (van Amstel); and the Department of Large Animal Clinical Sciences, College of Veterinary Medicine, University of Florida, Gainesville, FL (Shearer).

Reprint requests: Sarel R. van Amstel, BVSc, MMedVet(Med), DABVP, DACVIM, Department of Large Animal Clinical Sciences, College of Veterinary Medicine, University of Tennessee, Knoxville, TN 37996-0001.

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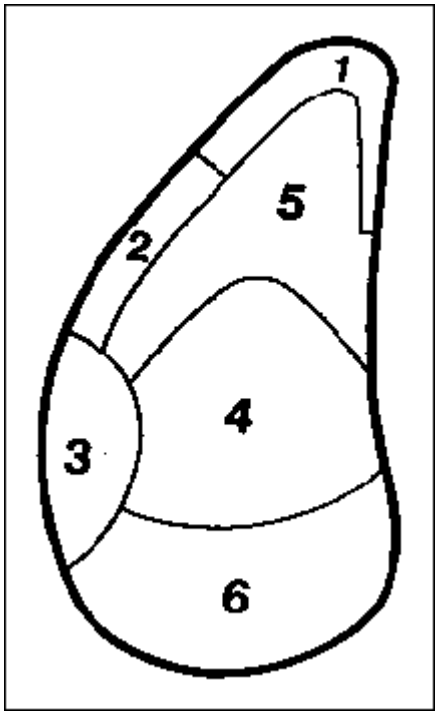

Fig 1. Zones of the claw.

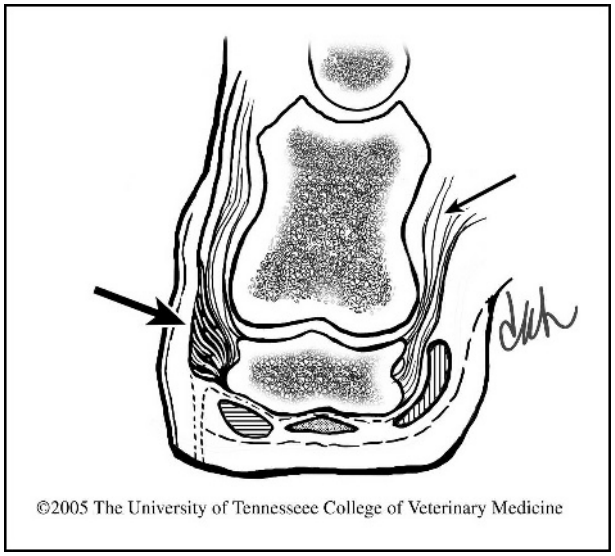

Fig 2. Small arrow: In the wall segments (axial and abaxial walls), the connective tissue of the suspensory system inserts at the distal phalanx in a cartilaginous insertion zone. Large arrow: Axially fiber bundles from the distal interdigital cruciate ligament form part of the suspensory system.

The suspensory system in cattle differs considerably from that of horses. The laminar corium is much less extensive in cattle than in horses, and there are no secondary laminae in the laminar corium (sensitive laminae) of cattle.[9] In the horse, weight bearing is primarily on the wall, whereas cattle cannot carry the same amount of mechanical load on the walls of their claws. Instead, weight bearing in cattle requires displacement of weight to other support structures within the sole and heel.[9] The primary structures within the supportive apparatus of the bovine claw are divided into the corium and digital cushion.

In the wall segments (axial and abaxial walls), the connective tissue of the suspensory system inserts at the distal phalanx in a cartilaginous insertion zone. This insertion zone is particularly well developed in the caudal wall and around the apex of the distal phalanx (Fig 2).[8] Collagen fiber bundles of the dermis in this area are oriented to align with tensile forces. Axially, fiber bundles from the distal interdigital cruciate ligament form part of the suspensory system (Fig 2).[8,10]

Ventral to the distal phalanx, bundles of collagen fibers of the dermis enclose cylindrical transverse-orientated compartments filled with fatty tissue (digital cushion) arranged in a series of 3 parallel cylinders (Fig 3).[8,9] The volume of all 3 pads in a single claw adds up to approximately 5.7 mL.[11] All 3 cushions extend from the junction of the skin and horn at the heel toward the tip of the 3rd phalanx. The abaxial and middle pads are shorter than the axial pad. The shorter pads lie superficial to the deep digital flexor tendon and do not reach farther distal than the insertion of the tendon. The axial fat pad courses from the axial border of the heel bulb toward the middle of the sole surface where it ends in the middle 3rd.[11]

The pathogenesis of sole ulceration is primarily based on mechanical injury to the solar corium, particularly the basement membrane and basal layers of the sole

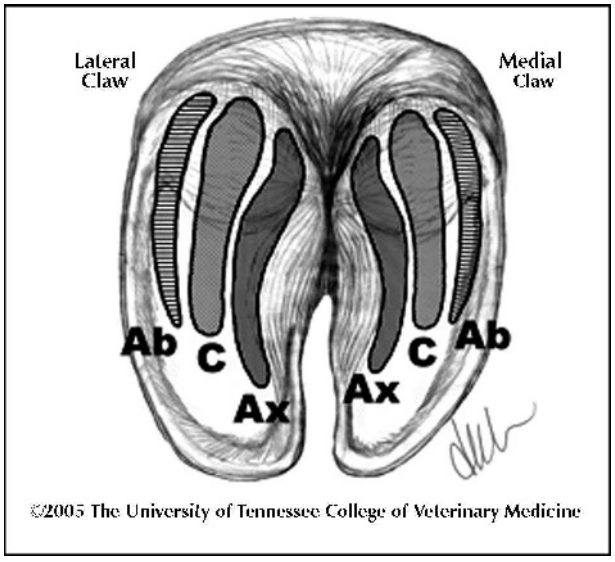

Fig 3. Digital cushion. Ventral to the distal phalanx, bundles of collagen fibers of the dermis enclose cylindrical transverse-orientated compartments filled with fatty tissue (digital cushion) arranged in a series of 3 parallel cylinders All three cushions extend from the skin-horn junction at the heel toward the tip of the 3rd phalanx. The abaxial (AB) and middle pads (C) are shorter than the axial (AX) pad. The shorter pads lie superficial to the deep digital flexor tendon and do not reach further distal than the insertion of the tendon. The axial fat pad courses from the axial border of the heel bulb toward the middle of the sole surface where it ends in the middle 3rd.

epidermis (sole horn). The lesion originates from inside the claw capsule and may not necessarily be associated with primary inflammatory or vascular changes, such as those associated with laminitis.[12] Such mechanical injury has been associated with laxity of the collagen fiber attachment (suspensory apparatus) between the caudal part of the 3rd phalanx and the basal layers of the wall epidermis, resulting in sinking of the bone.[9,12] Collagen is a structural protein and the main component of the connective tissue attachment (suspensory apparatus) that keeps the caudal aspect of the 3rd phalanx suspended above the digital cushion and solar corium. Weakening of the suspensory apparatus may have a biochemical origin. Hydrocortisone and prolactin, a lactogenic hormone, were observed to decrease protein synthesis in bovine claw explants.[13] High concentrations of a 52-kd protease (''hoofase'') were found in heifers after calving but not in maiden heifers.[14] These findings may explain the high incidence of sole hemorrhages seen in heifers 2–4 months after calving.[5]

Composition of the digital cushion also may play a role in the pathogenesis of sole and heel ulcers. In heifers, the digital cushion consists primarily of loose connective tissue and a small amount of fat primarily in the form of saturated fatty acids, whereas in older cows, the digital cushion has more fat consisting mainly of monounsaturated fatty acids that make the cushion softer, thus providing more shock absorption.[15] This difference may be one of the reasons for the greater incidence of sole ulcers in heifers housed on concrete.[6] Upon sinking, which occurs most commonly at the heel, the flexor tubercle (tuberculum flexorium) of the 3rd phalanx compresses the digital cushion and solar corium leading to vascular compromise, including ischemia due to congestion, edema and thrombosis, and interrupted or abnormal keratogenesis.[3,15–19] Both the corium and digital cushions of cows with sole ulcers are thinner and contain less fat than cows with normal claws.[9,10,15] The fat in the digital cushion is displaced by collagenous connective tissue.[10] As a consequence, the claws of cows with sole ulcers had significant ventral displacement of the 3rd phalanx, and the abaxial and palmar surfaces of the distal phalanx had more exostosis.[10,15] In cattle with chronic sole ulcers, extensive remodeling occurs in the bottom of the 3rd phalanx with the cortex consisting of osteoporotic bone along with exostosis, indicating poor distribution of compressive forces (Department of Pathology, College of Veterinary Medicine, University of Tennessee, unpublished data). Sole ulcers have a high rate of reoccurrence, and the caudal aspect of the suspensory system appears to have limited ability to regenerate.[3,6]

Mechanical injury to the epidermis with resultant abnormal keratogenesis caused by displacement (''sinking'') of the 3rd phalanx may occur without primary inflammatory, hormonal, or biochemical changes. This mechanical injury originates from outside the claw capsule and is primarily caused by overgrowth of the outer claw of the rear leg.[20] Normal weight distribution in the rear legs results in more weight being borne by the outer claw, resulting in accelerated horn growth particularly at the heel of the outer claw.[20] The cow tries to compensate for increased weight bearing on the outer claw by transferring more weight to the medial claw. To do this, she rotates her feet outward and her hocks inward, thus becoming progressively more ''cow-hocked.'' This change in posture, however, transfers more weight onto the inner portion of the heel and sole area of her lateral claw, resulting in a thick ledge of horn overgrowth at the interdigital space. This overgrowth of the hind lateral claw leads to concussion of the solar corium and contributes to sole ulcer formation.[20,21]

Mechanical injury without primary inflammatory changes is also seen in thin-soled dairy cattle where the protective function of sole horn becomes inadequate, particularly on hard walking surfaces.[22] Erosion of the heel, which may be a complication of interdigital dermatitis, also leads to a reduction in adequate horn thickness in the heel, causing increased pressure on the heel-sole junction and a predisposition for sole ulceration.[6,23]

Sinking of the flexor tubercle with mechanical injury to the solar corium may, on the other hand, follow production of inflammatory mediators associated with subclinical laminitis, which induce vascular changes and activate local matrix metalloproteinases.[13,15,24] An angiographic study of claws with sole ulcers revealed constriction of the lumen of the terminal part of the proper digital artery with an avascular area at the ulcer site.[18] Vascular corrosion casting studies have revealed that the arterial supply to several areas of the claw, including the typical sole ulcer area, are prone to circulatory disturbances because of inadequate inter-arterial anastomoses.[25] The same study revealed that pretreatment with lactate and histamine resulted in shunting of blood away from the dermal papillae, including those of the sole and heel. Other vascular changes at the ulcer site include congestion, edema and thrombosis of the microcirculation, proliferation of the tunica media in arterioles, and an increase in the number of arterio-venous anastomoses that occur as an adaptation to vascular changes, such as vasoconstriction and arteriosclerosis of the diseased claw. These changes are particularly prominent in chronic cases of sole ulceration.[16–19,25,26]

Separation of the laminar corium (sensitive laminae) and the claw has never been demonstrated in cattle to the same degree as it has in horses, and most of the pathology associated with laminitis appears to be confined to the heel area.[9] The laminar corium in the cow is smaller than that of the horse and lacks secondary folds. Therefore, the bovine heel provides additional support to counteract the tensile forces associated with suspension of the 3rd phalanx within the claw. The pathogenesis of inflammatory-based mechanical injury to the dermal-epidermal interface (suspensory apparatus) in the heel area is reported to occur in 3 phases.[3,9,27] Phase 1 consists of circulatory changes in the dermis as described above. This change leads to tissue hypoxia, edema formation, and activation of matrix-metalloproteinases, resulting in degradation of collagen with variable degrees of failure of the suspensory appara-

tus.[3,9,15,27] Phase 2 is associated with sinking of the 3rd phalanx and compression of the dermis (digital cushion and solar corium). Epidermal changes are secondary to the compression and are caused by interference with nutrient supply leading to abnormal cell proliferation and differentiation.[9] Phase 3 is associated with the appearance of sole horn hemorrhages and development of full-thickness horn defects.[15] Thus sole horn hemorrhages commonly seen in Zone 4 and the transition of Zones 4 and 6 are due to breakdown of the suspensory system in the caudal part of P3.[3,27,28] Thus this form of "laminitis" in cattle is different from classical equine laminitis in which the primary lesion is present in the area of the sensitive laminae.[9] Because the inflammatory and vascular changes primarily affect a different part of the corium, a more appropriate term in cattle could be coriosis.

Laminitis generally is accepted to be multifactorial in origin with rumen acidosis-induced endotoxemia viewed as the most important trigger in the pathogenesis of the condition.[29] Intravenous infusion of *Escherichia coli* endotoxin produced sole and wall hemorrhages and histopathologic changes in the laminar corium consistent with laminitis.[21] Other substances such as lactate and histamine also may play a role.[29]

Clinical Signs and Diagnosis

Sole hemorrhage is the early clinical sign of sole ulcer but only becomes visible several weeks or months after the initial injury.[5] Hemorrhages associated with sole ulcerations tend to occur in specific locations corresponding to common areas of traumatic damage to the solar corium. The most commonly reported ulcer site (ie, typical lesion) is the junction of the sole and heel (Fig 1, Zones 4,5).[1] Other locations include the apex of the toe (Fig 1, Zone 5) and the heel (Fig 1, junction Zones 4,6).[2,21,30] Affected animals may show different degrees of lameness, may stand with the hind feet placed well behind, or have an obvious cow-hocked stance in an effort to place more weight on the medial claws.[1] Early cases of sole ulcer are characterized by pain and hemorrhage at the sole ulcer site without an open horn defect. On 1st examination of mature lesions, the surface of the horn appears damaged and often is loose and undermined around the ulcer site and, when pared away, shows protrusion of the solar corium.[1,31] In early cases, the exposed corium shows little damage but becomes traumatized by the horn edges of the defect and the walking surface, resulting in the formation of granulation tissue.[31]

Less common sole ulcers, particularly those in Zones 4 and 6 (Rusterholtz and heel ulcers), can become infected and involve deeper structures.[1] Animals with complicated sole ulcer are severely lame, reluctant to move, lie down most of the time, have severe weight loss, and do not respond to conventional treatment.[1] Unilateral swelling of the affected digit occurs, particularly in the area of the heel, and may extend along the coronary band. The toe of the affected claw may become overextended because of avulsion of the of the deep flexor tendon at its insertion. Swelling above the fetlock in association with these signs may be indicative of tenosynovitis.[1,32,33] A draining tract extending from the ulcer site into the heel or from the skin of the dorsal coronary band to the distal interphalangeal joint may be present in chronic cases.[32]

In complicated sole ulcers, a necrotizing osteomyelitis involving the flexor tubercle of the 3rd phalanx usually results in a pathological fracture with avulsion of the deep flexor tendon.[28] The navicular bursa, retroarticular space (recess), and tendon sheath may be transformed into an abscess, which contains the navicular bone undergoing lytic changes and the avulsed necrotic deep flexor tendon with attached fragments of the flexor tubercle.[28] The distal interphalangeal joint also may become affected with destruction of the chondral and subchondral bone and thickening of the synovial membrane due to the formation of granulation tissue. Periosteal new bone formation occurs on the middle and distal phalanges at the insertion of the joint capsule.[28]

Pathology

Epidermal cells overlying the ulcer site become dyskeratotic, resulting in disturbed synthesis of keratin proteins.[16,34] Pathological changes of the cells in the lower epidermal layers include cloudy swelling. The synthesis and structure of the intercellular cementing substance between horn cells also are disturbed.[16,34] Increasing separation of epidermal cells caused by abnormal intercellular cementing substance and inflammatory exudates is present.[34] Horn surrounding the ulcer has dilated tubules and microcracks extending to the stratum spinosum, and necrotic areas are present in the epidermal layer.[4] Suprabasal epidermal mitosis and proliferation follow failure of normal proliferation and differentiation of the basal layer in an attempt to close the ulcer.[34] Vascular changes include dilatation and thrombosis of capillaries in the dermal papillae and proliferation of the tunica media in arterioles and vascularization (neocapillary formation).[7,26,34]

Healing

Information is scant with regard to the healing process of sole ulcers.[35] Factors that are important in the healing of sole ulcers are those that may influence the structure of the newly formed epidermis and the basement membrane, which facilitates reattachment between the epidermis and dermis. The latter has a rich capillary network, which provides nutrients for successful keratogenesis.[4]

Healing of sole ulcers follows a course similar to that of healing of skin wounds.[9] Serial biopsies from cows with sole ulcers after treatment, which included elevation of the affected claw, indicated that regeneration and epithelialization with the formation of new dermal blood vessels started after 9 days and by day 14 progressive regeneration and proliferation of epidermal cells occurred. The ulcer is covered with a layer of soft cornified epithelium. Ultrastructural organization, however, is not complete by this time. By day 19, increased cornification has occurred, and maturation of the epidermis and

vascularization of the dermis are almost complete. Areas of incomplete differentiation and dyskeratosis are still present. By day 50, the ulcer is covered by a fully differentiated cornified epidermis, but small areas of epidermal dyskeratosis may still be observed.[34]

In cases in which severe damage to the corium has occurred, basement membrane integrity and attachment to basal epidermal layers are lost at sole ulcer sites. These changes lead to the loss of signaling cues between the basement membrane and basal epidermal layers.[13] Reattachment between the overgrowing epidermis and the basement membrane in the ulcer site is necessary for normal healing to take place (Mülling C, personal communication).

Elevation of the sound claw to relieve weight bearing on the affected claw and prevent further mechanical injury has a beneficial effect on the condition of the corium and the quality of the newly formed epidermis.[1,31] Supplemental dietary biotin may contribute substantially to improving histological horn quality in cattle with sole ulcer and may improve claw health in intensively managed primiparous dairy cows.[4,35] Cows with slower healing sole ulcers had significantly lower blood concentrations of vitamin A and iron compared to those whose sole ulcers healed more quickly.[36] When examined after 7 days, padded bandages consisting of cotton wool containing 20-g copper sulfate and covering the whole foot did not improve the healing rate of sole lesions, including ulcers, compared to applying a claw block and leaving the lesions open.[37] No difference in the healing rate of sole ulcers was found between cattle (typical sole and heel ulcers) that were bandaged weekly for 3 weeks and those whose lesions were left open. Wood blocks were used in all affected cattle to relieve weight bearing of the affected claws. Bandages consisted of nonirritating antiseptic ointment covered with gauze, expandable type material, and duct tape (van Amstel, unpublished data). Another study also found no differences in the healing rate of full-thickness claw horn lesions between bandaged and nonbandaged groups. In that study, no claw blocks were used to relieve weight bearing of the affected claws.[38]

Treatment

One of the most important treatment considerations includes elevation of the sound claw by application of a claw block to relieve all weight bearing from the affected claw.[1] This procedure provides pain relief and aids healing.[1] Proper application of blocks is important to provide a flat, stable, weight-bearing surface for the sound claw.[21,39] The claw should be pared flat to provide a weight-bearing surface that is perpendicular to the long axis of the metacarpus and tarsus. The block should provide proper heel support, but adhesive material should be cleared away from the area between the block and the heel because the heel horn is soft and can be damaged by the hard edges of the cured adhesive material.[21] Blocks that are left on too long (generally >2 months) or that have been applied incorrectly can cause further discomfort and lameness.[9,21] In early cases, lowering of the affected heel will transfer sufficient weight to the healthy claw for healing to take place.[40] Early lesions may have a pronounced area of subsolar hemorrhage. Such lesions can be soft on palpation but should not be opened or debrided, thus creating full-thickness horn defects and exposing the underlying corium. Relief of weight bearing on the affected claw by either lowering the affected heel or applying a claw block to the opposite healthy claw is very important. Corrective trimming procedures for ulcers with full-thickness horn defects include creating a steep slope of the horn around the ulcer, taking care not to damage the corium. This procedure is necessary because, in many cases, the horn covering and surrounding the ulcer is necrotic and undermined, resulting in the entrapment of dirt.[1] Creation of deep holes in the sole should be avoided because such defects will predispose the sole to trapping of manure and retarded healing.[21] Exuberant granulation tissue should be removed surgically to the level of the surrounding trimmed and sloped horn (Fig 4). Application of caustic agents to reduce granulation tissue is contraindicated because this approach will impede healing by interfering with cell growth from the edges of the ulcer.[1] Copper sulfate has been shown to penetrate the horn quite extensively and, as such, may make the horn more brittle.[41] Application of antibiotic dressings, such as oxytetracycline, may be necessary when the exposed corium has developed papillomatous digital dermatitis (Fig 5) (van Amstel, unpublished observation).

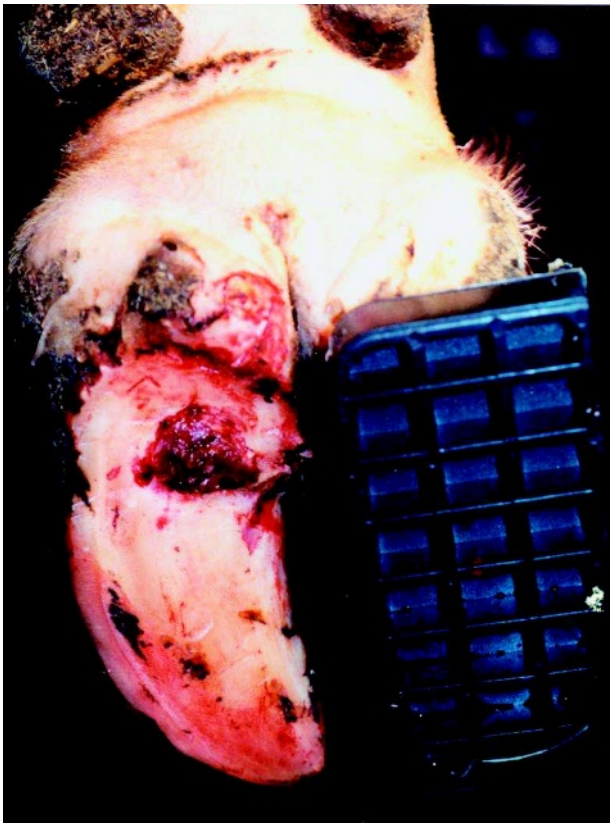

Fig 4. Granulating ulcer. Note the sloped sole surrounding the ulcer. The granulation tissue is cut at the level of the sole.

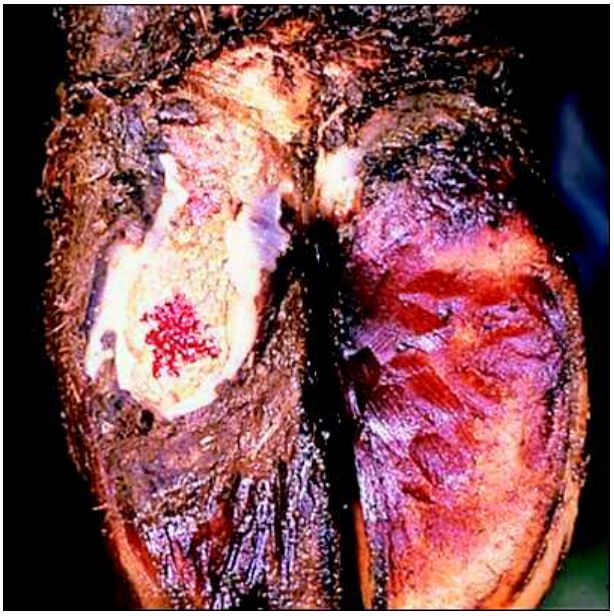

Fig 5. Digital dermatitis invading the corium of the heel and sole.

Prevention

The etiopathogenesis of sole ulceration is multifactorial, thus prevention is based on nutritional, management, and environmental factors, with the most important being those factors that predispose the animal to subclinical laminitis.[1,7] Normal hormonal and biochemical changes that occur during the time of calving are additional complicating factors.[12] The normal biomechanics of weight bearing lead to overgrowth of the outer claw of the hind leg.[20] This overgrowth puts additional strain on the suspensory system of the caudal aspect of the claw, leading to possible mechanical injury and abnormal keratogenesis.[20,23] Similarly, thin soles caused by excessive moisture, abrasive concrete surfaces, or over trimming also may predispose the animals to mechanical injury.[22]

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