

Case Report – Herd Dermatophilosis Outbreak in a Desert Climate

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Abstract

An acute, rapidly spreading, mid-summer outbreak of a dermatologic condition in 60 cows on a 3000 cow dairy was accompanied by anorexia and agalactia. Lesions consisted of multiple, 2-5 mm, thickened and crusted scabs which when removed revealed inflamed and ulcerated dermis. The herd was located in a desert environment where daytime ambient temperatures often exceeded 100°F. Management practices to cool the cattle included continual application of water misters. Initial on-farm diagnostic attempts were inconclusive so a representative animal was sent to the Washington State University Veterinary Teaching Hospital for evaluation. The dermatologic condition clinically resembled dermatophilosis, and diagnostic testing supported this diagnosis. It was concluded that despite environmental conditions that were inconsistent with dermatophilosis, continual misting of cattle resulted in skin maceration facilitating development of the condition. Treatment consisted of daily application of a topical disinfectant and alteration of misting times so cattle would dry between sessions. More severely affected cattle received a one-time parenteral injection of a broad-spectrum antimicrobial. Complete recovery was observed within 14 days of the initiation of treatment and management changes.

Résumé

Une flambée aiguë et très rapide, en plein été, d'un problème dermatologique chez 60 vaches d'une ferme laitière de 3000 têtes était accompagnée d'anorexie et d'agalactie. Les lésions comprenaient de multiples croûtes épaissies de 2-5mm qui lorsque enlevées révélèrent un derme enflammé et ulcéré. Le troupeau était gardé en milieu désertique où la température ambiante durant le jour dépassait souvent 100°F. L'utilisation de nébulisateurs d'eau était l'un des moyens

employés pour rafraîchir les animaux. Le diagnostic initial à la ferme n'étant pas conclusif, un animal représentatif a été envoyé au centre hospitalier universitaire vétérinaire de l'université d'état de Washington pour un examen plus approfondi. Le problème dermatologique ressemblait cliniquement à la dermatophilose et les tests diagnostiques ont confirmé ce diagnostic. Bien que les conditions environnementales n'étaient pas conciliables avec la dermatophilose, l'utilisation continue de nébulisateurs d'eau pour les bovins a pour sa part favorisé la macération de la peau et le développement de la condition. Le traitement comprenait une application topique d'un désinfectant et incluait un changement dans l'utilisation des nébulisateurs d'eau afin de permettre aux bovins de sécher entre les périodes d'aspersion. Les bovins les plus affectés ont reçu une injection parentérale simple d'un agent anti-microbien à large spectre. La récupération complète pris place dans les 14 jours suivant le début du traitement et le changement dans la pratique d'aspersion des animaux.

Introduction

Dermatophilosis is an exudative, suppurative, superficial bacterial dermatitis that primarily affects ruminants as well as other species, including humans. The causative agent, *Dermatophilus congolensis*, is a branching filamentous actinomycete bacterium. Among mammalian species, the different forms of the disease are distinguished both by the clinical appearance and the anatomic areas affected. Various colloquial terms for dermatophilosis include, "rain scald" (horses), "cutaneous streptothricosis" (cattle and goats), "lumpy wool" or "mycotic dermatitis" (wooled areas of sheep), and "strawberry foot-rot" (extremities of sheep).

Dermatophilosis occurs worldwide and there is no sex or age predilection. The exact origin of the organism is unknown, however, it is suspected to have a

saprophytic existence in soils. Nonetheless, chronically infected animals are implicated to be the source of infections where the organism is transmitted via contact, contaminated fomites, or via various species of flies and ticks.

History

A 5-year-old, 1485 lb (675 kg) Holstein cow was referred to the Washington State University Veterinary Teaching Hospital for evaluation, diagnosis and treatment of a dermatological condition. The cow was one of approximately 60 cows similarly affected within a 10-day period. The herd consisted of approximately 3000 lactating cows and heifers located in central Washington, in a desert region known as the Columbia Basin.

All 60 affected cows were housed in a dry lot fresh-cow pen, and were less than 20 days into lactation. Skin lesions on all cows appeared abruptly during a period of hot, dry weather where daytime ambient temperatures often exceeded 100°F. All affected animals had varying degrees of raised crusts, from small localized areas of a few crusts, to lesions covering the majority of the body surfaces, including head and face, dorsal regions of the thorax, abdomen, and limbs, and the udder. The owners were particularly concerned because of rapid spread of the condition, as well as a concurrent and significant decline or cessation of milk production by affected animals.

Prior to admission, the referring veterinarian had performed physical examinations and skin scrapings on several animals. Cytologic examination of scraping samples was inconclusive. Skin biopsies were also obtained and were *en route* to the diagnostic laboratory at the time of the cow's admission to the teaching hospital.

Clinical and laboratory findings

On physical examination the cow was lethargic and anorectic. The cow was tachycardic (96 bpm, normal 40-60 bpm), and the rectal temperature and respiratory rate were within normal limits. Rumen contractions were absent. Skin lesions were most prominent along the back, neck and lateral thorax and consisted of numerous 2-5 mm diameter, severely thickened and crusted areas of skin. Thick crusts containing hair could be removed from the affected regions, exposing an inflamed, eroded or ulcerated dermis. Manipulation and removal of the crusts seemed to be painful.

Blood was obtained for a complete blood count (CBC) and a serum biochemical profile. The CBC revealed a left shift (1720 band cells/ μ L; reference range <100 band cells/ μ L) and hyperfibrinogenemia (1000 mg/dl; reference range, 300-700 mg/dl). Serum biochemical analysis revealed slightly elevated gamma-glutamyltransferase (35 IU/l; reference range, 16-30 IU/l), aspartate aminotransferase (470 IU/l; reference

range, 50-83 IU/l), and creatine phosphokinase (618 IU/l; reference range, 60-305 IU/l), as well as hypoalbuminemia (2.5 g/dl; reference range, 3.2-4.2 g/dl). Dipstick urinalysis was normal.

Based on history, clinical signs and blood work, preliminary rule-outs included dermatophilosis, mange, staphylococcal folliculitis and furunculosis, pemphigus foliaceus, dermatophytosis, viral infections and zinc responsive dermatosis.¹² The left shift and hyperfibrinogenemia were attributed to inflammatory processes associated with the skin lesions. Elevated gamma-glutamyltransferase and aspartate aminotransferase were attributed to anorexia and resultant hepatic lipid infiltration, though primary hepatic disease was not ruled out. The slight increase in creatine phosphokinase was not deemed relevant and was likely associated with transportation. Hypoalbuminemia was not thought to be associated with gastrointestinal or urinary losses, and primary liver disease was thought to be unlikely. Loss of albumin was most likely from the extensive skin lesions.

Skin scrapings were stained with Wrights Giemsa^a stain. While few bacteria were seen, the parallel rows typical of *Dermatophilus congolensis* were not observed and the slide was determined to be non-diagnostic. Because dermatophilosis was suspected and the skin scrapings were inconclusive, impression smears were obtained. Impression smears of both the crusts and the underlying inflamed ulcers were stained with Wrights Giemsa^a stain. The impression smears had typical Gram-positive, branching, filamentous, parallel rows of cocci, also known as the "railroad track" configuration.

From the impression smears a presumptive diagnosis of dermatophilosis caused by *Dermatophilus congolensis* was established. Histopathology and bacterial culture were performed to further confirm the diagnosis of dermatophilosis, and to rule out other secondary infections.

Skin biopsies (5 μ m) were fixed in 10 % buffered formalin. The tissue was embedded in paraffin and 5 μ m sections stained with hematoxylin and eosin, a tissue Gram stain (Brown-Hopp's), and Grocott's Ammoniacal Silver (GMS). Microscopic lesions consisted of severe, multifocal to coalescing suppuration with large numbers of intracorneal bacterial filaments and moderate superficial, suppurative, perivascular dermatitis. Multifocal to coalescing regions of marked epidermal hyperplasia and hyperkeratosis were accompanied by the formation of multiple layers of intracorneal pustules which contained aggregates of degenerate neutrophils, cellular debris and finely granular eosinophilic fluid. Large numbers of bacterial filaments composed of parallel rows of Gram-positive coccoid cells were within the intracorneal pustules and in adjacent layers of keratin.

Multifocal areas of epidermal necrosis resulted in ulceration of the epidermis, which was replaced by a thick mat of serocellular debris. Moderate infiltrates of neutrophils, lymphocytes and macrophages were present at the dermal-epidermal junction and surrounded vessels in the superficial dermis. No fungal elements were seen in GMS stained sections.

Skin samples were plated onto blood agar^b and incubated in 5% CO₂ at 98.6°F (37°C). Multiple small, dry, grey-yellow colonies with complete hemolysis were visible after 24-48 hours. Colonies were firmly adherent in the agar. Older colonies developed a wrinkled appearance. Microscopic examination of Gram-stained specimens revealed predominantly Gram-positive cocci, with occasional filamentous forms. Characteristic septa were observed forming parallel rows of coccoid cells on horizontal and vertical planes. Morphologic characteristics combined with the clinical signs, history and previous test results confirmed the diagnosis of dermatophilosis.

Biochemical tests were not performed since diagnosis is readily made on microscopic appearance and colony characteristics.⁷ However, biochemical testing characteristics of *D. congolensis* include catalase positive, urease positive and gelatin positive, and production of acid from glucose, fructose and maltose. Furthermore, *D. congolensis* is indole negative, does not reduce nitrate, and does not attack sucrose, salicin, xylose, lactose, sorbitol, mannitol or dulcitol.⁷

Therapeutic management

Existing recommendations for treating animals affected with dermatophilosis include thoroughly brushing to remove crusted lesions followed by thorough cleansing with either a povidone iodine shampoo or chlorhexidine solution daily for seven days, then weekly until the crusts disappear.⁵ Because of the large number of cows affected, such intensive treatment was deemed impractical. Instead, it was recommended that the affected cows be sprayed daily with a 1:10 chlorhexidine^c solution for seven days, and then once weekly until the crusts disappeared. More severely affected animals with generalized dermatophilosis were given one treatment of long-acting oxytetracycline^d (9 mg/lb; 20 mg/kg, IM) in addition to the topical treatment.⁵ During hot weather misting times were altered to allow the cows sufficient time to dry. Once ambient temperatures cooled, use of the misting system was discontinued. Fly control measures were reviewed and deemed to be appropriate. Fourteen days after instituting management changes and treatments, all affected cows were recovered. No new cases of dermatophilosis were reported and milk production increased to pre-outbreak levels.

Discussion

Development of clinical dermatophilosis involves the classical interaction of host (cattle), agent (bacteria) and environmental factors. The disease is most commonly associated with seasonal environmental conditions that involve high humidity or rainfall, in combination with events resulting in mechanical trauma to the skin (insect bites, thorns, maceration, etc.). In the Pacific Northwest region of the United States, dermatophilosis typically occurs during the spring and winter months. The presentation of this case was unusual since it occurred during the dry summer months in a desert climate. While the natural environmental conditions were unfavorable for the development of disease, it was apparent that the implementation of management practices had a direct influence. Specifically, a misting system was in operation 24 hours a day because of the high environmental temperatures, and cows had the opportunity to be under the misters for prolonged periods while waiting to be milked. Farm operators noted that during days of extreme high temperatures, the cows would stay under the misters as long as they were allowed. Furthermore, the most severely affected cows were those noted to be under the misters the longest amounts of time.

To establish infection, the protective barriers of the skin (sebum, surface antibodies, stratum corneum) must first be penetrated.^{6,11} Disruption can result from maceration, mechanical trauma or via hematophagous fly bites.² Importantly, even skin damaged by ectoparasites or mechanical trauma still requires moisture for clinical disease to occur. In the case described here, the opportunity for mechanical trauma was deemed unlikely, however, the opportunity for hematophagous flies or simple maceration via prolonged misting was present. Furthermore, the seasonal presence of large numbers of non-hematophagous flies were believed to be responsible for spreading the disease between cows. While not applicable to this particular disease outbreak, infestation of cattle with the ixodid tick *Amblyomma variegatum* has been shown to be a risk factor for dermatophilosis in other regions of the world. Evidence suggests that infestation by *A. variegatum* results in systemic immunosuppression of cattle, which in turn can lead to a severe, progressive and chronic form of dermatophilosis.²

Results from field observations have suggested that different breeds of cattle may vary in their susceptibility to dermatophilosis.⁸ While most studies have examined breed susceptibility to dermatophilosis in relation to susceptibility to *A. variegatum* infestation, several BoLA (bovine major histocompatibility complex, MHC) polymorphisms have been correlated to groups of resistant and susceptible cattle. Examination of MHC

haplotypes in the infected cattle in this report was not pursued. Other host factors such as malnutrition have also been shown to affect the progression of dermatophilosis.¹⁰ In many regions of the Pacific Northwest there are soils which are both copper and selenium deficient. However, the effects of these trace minerals on herd health and immunity were not considered likely in this case since supplementation was provided.

D. congolensis is not considered to be a highly pathogenic organism, and infection is most significant in hosts immunocompromised by skin damage.² However, variation between isolates with regard to putative virulence factors have been described. In *in vitro* studies, variation in hemolytic and proteolytic activity, as well as expression of phospholipase and sphingomyelinase activity, has been demonstrated.^{3,4,9} Nonetheless, production of *in vivo* virulence factors and their effects on the host's immune system have yet to be investigated.

The pathogenesis of dermatophilosis has been described.¹ Initially, minor trauma to the epidermis and prolonged wetting of skin allows for the establishment of small foci of infection. Repeated cycles of hyphae invasion into the epidermis, bacterial multiplication, infiltration by neutrophils, and regeneration of the epidermis results in crust formation. Rainfall or high moisture then aids in the spread of cocci and zoospores from crusts to healthy skin, forming new foci of infection. *D. congolensis* has been shown to remain viable in sloughed scabs for up to 42 months.^{5,12} Cow-to-cow transmission is then facilitated via contact with infected animals, exposure to sloughed scabs, or via flies acting as mechanical vectors.

Dermatophilosis should be strongly considered when a crusting dermatosis is observed, irrespective of the time of year.¹² A diagnosis can be typically be established after considering historical information of the animals' environment, observing clinical signs of exudation and crust formation, and examining stained impression smears or scrapings. In the case presented here, skin scrapings were less helpful as a diagnostic aid compared with impression smears because the mechanical action of scraping tended to disrupt the classical "railroad track" morphology of the organisms. Skin biopsies, histopathology and bacterial culture are also used to support a clinical diagnosis.

Conclusions

This report illustrates the need to consider dermatophilosis even when the presentation is not consistent

with seasonal conditions. Though dermatophilosis is primarily a disease that occurs in wet or humid climates and seasons, the disease can occur unexpectedly in the hot and dry climates if a particular environment, affected by management practices, is present.

Footnotes

- ^aDiff-Quick, Dade Behring Inc., Newark, DE.
^bColumbia Blood Agar, Remel, Lenexa, KS.
^cNolvasan Solution, Fort Dodge Animal Health, Fort Dodge, IA.
^dLiquamicin LA-200, Pfizer Animal Health, New York, NY.

References

1. Ambrose NC: The pathogenesis of dermatophilosis. *Trop Anim Hlth Prod* 28:29S-37S, 1996.
2. Ambrose N, Lloyd D, Maillard JC: Immune responses to *Dermatophilus congolensis* infections. *Parasitology Today* 15:295-300, 1999.
3. Ambrose NC, Mijinyawa MS, Hermoso de Mendoza J: Preliminary characterization of extracellular serine proteases of *Dermatophilus congolensis* isolates from cattle, sheep and horses. *Vet Micro* 62:321-335, 1998.
4. Ellis TM, Masters AM, Sutherland SS, Carson JM, Gregory AR: Variation in cultural, morphological, biochemical properties of Australian isolates of *Dermatophilus congolensis*. *Vet Micro* 38:81-102, 1993.
5. Evans AG: Diseases of the skin. in Smith BP (ed): *Large Animal Internal Medicine*, ed 2. St Louis, Mosby-Year Book, Inc, 1996, pp 1403-1443.
6. Evans AG: Dermatophilosis: Diagnostic approach to nonpruritic, crusting dermatitis in horses. *Compend Contin Educ Pract Vet* 14: 1618-1623, 1992.
7. Laboratory Diagnosis of *Dermatophilus congolensis*. in Quinn PJ, Carter ME, Markey BK, Carter GR (eds): *Clinical Veterinary Microbiology*, London, UK, Wolfe Publishing, an imprint of Mosby-Year Book Europe Limited, 1994, pp 153-155.
8. Leroy P, Marchot P: The resistance to dermatophilosis of Dinka cattle breed, Dinka crossbred and Boran, Friesian, Jersey, Sahiwal crossbreds. *Ann Rech Vet* 18: 107-109, 1987.
9. Masters AM, Ellis TM, Grien SB: *Dermatophilus congolensis*: strain differences in expression of phospholipase activity. *Vet Micro* 51:199-213, 1997.
10. Sanders AB, How SJ, Lloyd DH, Hill R: The effect of energy malnutrition on experimental infection with *Dermatophilus congolensis*. *J Comp Pathol* 103: 361-368, 1990.
11. Scanlan CM, Garrett PD, Geiger DB: *Dermatophilus congolensis* infections of cattle and sheep. *Compend Contin Educ Pract Vet* 6: S4-S8, 1984.
12. Scott DW: Bacterial diseases, in Pedersen D (ed): *Large Animal Dermatology*. Philadelphia, WB Saunders, 1988, pp136-146.