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Pathology in Practice

Figure 1—Photographs of the right (A) and left (B) hind limbs of a 13.5-year-old Belgian gelding referred for evaluation of ulceration and crusting of the coronary bands with progressive sloughing of all frogs (cuneus ungulae) and chestnuts (tori carpeus and tori tarseus) over a period of 12 weeks. In panel A, notice that the coronary band (arrow) is thickened and covered by hyperkeratotic scale. Hoof wall (star) adjacent to the coronary band is eroded and rough. In panel B, the ergot (thin arrow) is thickened by keratin fronds. Proliferative tissue covers the frog (thick arrow).

History

A 13.5-year-old 984-kg (2,164.8-lb) Belgian gelding was referred for evaluation of ulceration and crusting of the coronary bands with progressive sloughing of all frogs (cuneus ungulae) and chestnuts (tori carpeus and tori tarseus) during the preceding 12 weeks. All ergots (tori metacarpus and tori metatarseus) were thickened by tan keratinaceous fronds. The frogs were thickened by papillary fronds of friable brown-tan tissue. All 4 hooves had horizontal ridges. Treatment of the horse, which had been instituted within 1 week after the owners noticed the initial seeping cracks at the coronary bands, involved topical administration of nitrofurazone and metronidazole paste, dilute iodine footbaths, and debridement of the frogs. Management recommendations included promoting a hygienic environment for the feet with bandaging and avoidance of wet or muddy conditions. Phenylbutazone was administered orally initially, supplemented with repeated epidural administration of morphine in the caudal portion of the vertebral column as signs of foot pain increased in severity. The disease progressed during the following 11 weeks despite treatment. The horse became recumbent and was euthanized by IV administration of an overdose of pentobarbital-phenytoin solution.

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Gross Findings

On postmortem examination, lesions of the coronary bands, hoof walls, and frogs of all 4 feet were similar. Coronary bands were circumferentially thickened and covered in dry to moist, yellow-white hyperkeratotic scale (**Figure 1**). The hoof adjacent to the coronary band had a 2-cm-wide circumferential band of dark brown, eroded, and rough surfaced to granular dry wall. More distal portions of the hoof wall had horizontal ridges, vertical cracks, and splayed lateral and medial quarters. Frogs were thickened by proliferative fronds of friable brown-tan tissue with a strong musty to fetid odor. All ergots were thickened by wet, tan fronds of keratin with reddening of the underlying dermis. All chestnuts were eroded nearly to the level of adjacent haired skin and covered by a layer of moist yellow-white scale.

Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page →

Histopathologic Findings

In sections through the coronary bands, the epidermis was hyperplastic with marked acanthosis and formation of long papillary fronds (**Figure 2**). A thick layer of orthokeratotic hyperkeratosis with patchy parakeratotic hyperkeratosis affected the epithelium over the coronary band dermis. Neutrophils were observed focally and rarely in the stratum corneum. Horn that had a normal appearance was not observed. Midlevel to superficial stratum spinosum cells had ballooning degeneration with coalescence into vesicles. Cells of the more superficial stratum spinosum were necrotic with pyknosis and karyorrhexis. Few lymphocytes and plasma cells were in the superficial dermis. Dermal blood vessels had high numbers of neutrophils.

The ergots, frogs, heel bulbs, and soles had histopathologic changes similar to those detected in the coronary bands. However, in these tissues, there was accumulation of proteinaceous exudate mixed with numerous neutrophils between layers of the stratum corneum. The lymphoplasmacytic perivascular dermatitis was more severe. Bacteria colonized the stratum corneum in most sections. No spirochetes or fungi were observed in sections of frog or sole following staining with Warthin-Starry silver or Grocott methenamine silver stain. Sections of the ergots and heel bulbs were not evaluated with these stains.

In the chestnuts, superficial stratum corneum (horn) was eroded to the stratum spinosum. Superficial stratum spinosum cells had ballooning degeneration and lytic necrosis. Few lymphocytes and plasma cells were in the superficial dermis. Dermal venules had a large number of luminal neutrophils.

Biopsy specimens of all 4 coronary bands had been obtained 12 weeks prior to necropsy, and biopsy specimens of the left hind limb frog and white line (zona alba) were obtained 4 weeks prior to necropsy. Histologic changes in these biopsy specimens were similar to those identified in those tissues at necropsy. In a section of coronary band skin, there were scattered, round, periodic acid-Schiff-positive, 80- to 100-µm-diameter structures, consistent with arthropod eggs in the stratum corneum and overlying crust. No mites were detected in these biopsy samples or skin scrapings performed 5 days after the first biopsy. No spirochetes were identified with silver histochemical staining in these biopsy specimens.

Morphologic Diagnosis and Case Summary

Morphologic diagnosis: epidermal proliferation and hyperkeratosis with degeneration and necrosis of stratum spinosum keratinocytes in the coronary bands, ergots, and chestnuts and proliferative pododermatitis with degeneration and necrosis of stratum spinosum keratinocytes in the frogs, soles, and heel bulbs.

Case summary: coronary band dystrophy with proliferative pododermatitis in a horse.

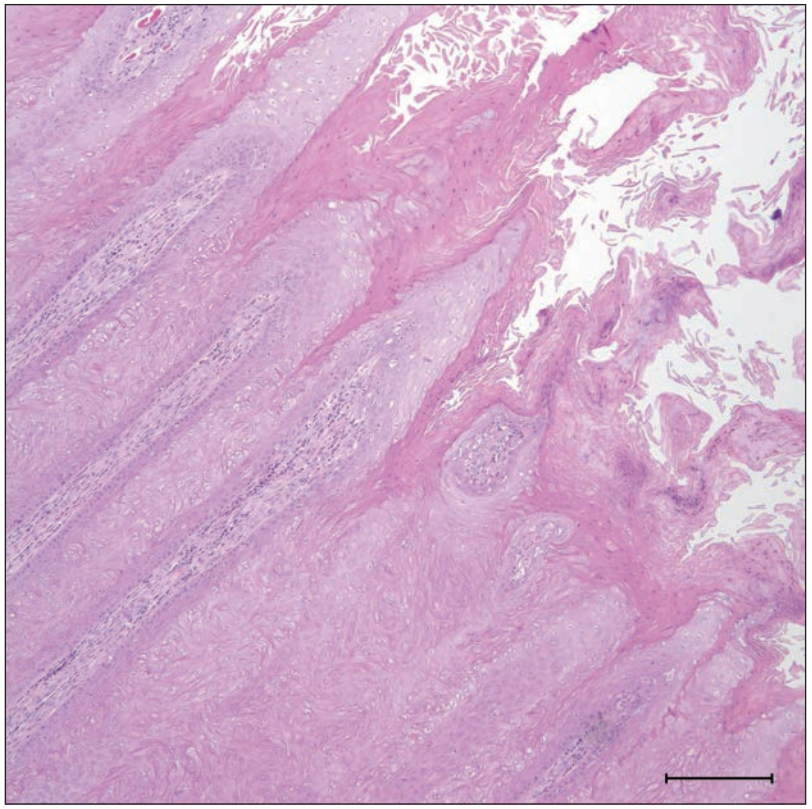

Figure 2—Photomicrograph of a section of a coronary band of the horse in Figure 1. The epidermis has orthokeratotic and parakeratotic hyperkeratosis with acanthosis and ballooning degeneration of keratinocytes in the stratum spinosum. Lymphocytes and plasma cells are in the dermis. H&E stain; bar = 200 µm.

Comments

For the case described in the present report, the symmetric distribution and histopathologic appearance of lesions and patient signalment supported a diagnosis of coronary band dystrophy. This idiopathic condition is anecdotally most common in mature draft horses, with no apparent sex predisposition.[1] The entire coronary band may not be affected, but all 4 hooves are usually affected to an equal extent.[1] Grossly, coronary bands are proliferative with crusting, scaling, and variable erythema.[1,2] Hooves may be thickened with irregular ridges and scaling, as was evident in the horse of the present report.[3] Chestnuts and ergots can be affected by this condition.[1,2]

The differential diagnosis for scaling and crusting of the coronary band in horses includes dermatophilosis, selenium toxicosis, dermatophytosis, pemphigus foliaceus, hepatocutaneous syndrome, eosinophilic exfoliative dermatitis, and infestation with *Chorioptes equi*.[1,3] Dermatophilosis, dermatophytosis, pemphigus foliaceus, and eosinophilic exfoliative dermatitis were ruled out on the basis of histopathologic findings. Hepatocutaneous syndrome was ruled out because the horse had no clin-

ical signs of liver disease, circulating hepatic enzyme activities were within reference limits 11 weeks before euthanasia, and the liver appeared grossly and histologically unremarkable at necropsy. Selenium concentration in serum or frozen liver tissue was not evaluated; however, chronic selenium toxicosis was considered unlikely because there was no change in texture of hair or loss of hair from the mane or tail.[3–5] Furthermore, this horse had no history of receiving supplemental selenium and came from a region of the country that does not have a high concentration of selenium in the soil.[4,5] Although histologic evaluation of a biopsy sample obtained from skin of an affected coronary band submitted 12 weeks prior to necropsy revealed scattered, arthropod eggs in the stratum corneum and overlying crust, no mites were detected histologically or in skin scrapings performed 5 days after the initial biopsy.

The proliferative pododermatitis affecting the frogs, soles, and heel bulbs could have been secondary to coronary band dystrophy, or the horse of the present report could have had concurrent canker. Equine canker is a proliferative pododermatitis that can affect frogs, bars, and soles, with extension into the adjacent hoof wall in severe cases.[6,7] Histologically, canker can resemble coronary band dystrophy with hyperplastic epithelium, intraepithelial neutrophilic inflammation, and superficial ballooning degeneration.[6] In a previous report[6] of 3 horses with canker, spirochetes were identified in proliferative epithelium of canker lesions following silver histochemical staining. By use of 16S rRNA gene sequencing, *Treponema* spp have also been identified in canker lesions of 2 horses.[7] For the horse of the present report, no spirochetes were identified with silver histochemical staining in the coronary band, frog and white line biopsy specimens obtained prior to necropsy or in postmortem samples of frog or sole. Involvement of the frog in coronary band dystrophy in horses has been reported,[3] but it is unknown how commonly frogs are affected or whether such involvement is related to coronary band dystrophy or a separate condition. Although we cannot definitively rule out the possibility of canker as a cause of the frog, sole, and heel bulb lesions in the case described in the present report, no spirochetes were observed and canker should not affect the coronary band.

Coronary band dystrophy should be considered in draft horses with scaling and crusting of the coronary bands. There is no known cure for this disease.[2]

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