



Equine Pastern Dermatitis

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ABSTRACT: Equine pastern dermatitis is not a single disease but rather a cutaneous reaction pattern in horses. This dermatitis can affect any equine breed but is most common in draft horses. Identifying and treating the predisposing and perpetuating factors are just as important as addressing the primary underlying cause of equine pastern dermatitis. This dermatitis usually affects the caudal aspect of the pasterns but can spread dorsally, with the hindlimbs most commonly affected. The clinical presentation varies from a very mild dermatitis to a chronic, very proliferative form. This article reviews the clinical signs, diagnosis, and treatment of equine pastern dermatitis.

Equine pastern dermatitis is not a single disease but rather a cutaneous reaction pattern in horses. This dermatitis should be considered a syndrome rather than a diagnosis.^{1,2} Uncovering the underlying causes before treatment is key to minimizing treatment failure and frustration. To achieve a positive therapeutic outcome, treating the predisposing and perpetuating factors is just as important as addressing the primary cause of equine pastern dermatitis² (see box on page 216). This article reviews the clinical signs, diagnosis, and treatment of equine pastern dermatitis.

CLINICAL SIGNS AND PATHOGENESIS

Equine pastern dermatitis can affect any equine breed but is most common in draft horses.^{1,2} Feathering over the pasterns is a predisposing factor.^{1,3} Equine pastern dermatitis occurs without a sex predilection and mostly in adult horses.¹⁻⁴ The dermatitis typically involves the caudal aspect

of the pasterns, with the hindlimbs most commonly affected.^{2,5} If not addressed, the lesions can spread cranially to the front of the pastern and fetlock.^{1,2} The lesions are usually bilaterally symmetric; however, they can affect just one limb. The lesions are most often detected on, but are not limited to, the nonpigmented areas of the pasterns.⁵ The clinical signs vary, depending on the cause, duration, and previous therapy. Initially, there is edema, erythema, and scaling, which rapidly progress to exudation, matting of the hair, and crusting.^{1,2,5} If the underlying cause is vasculitis, ulcers may be noted.^{2,5} Secondary bacterial infection is a common complication and a perpetuating factor.^{1,2} With chronicity, the skin may become thickened and fissured due to constant movement and flexion.⁵ The lesions are often painful and can result in lameness.⁵ There are three different presentations of equine pastern dermatitis:

- The **mild form** (scratches, mud fever, mud rash; Figure 1) is the mildest and most prevalent form of equine pastern dermatitis. This form is characterized by alopecia, dry

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Pathogenesis of Equine Pastern Dermatitis²

<p>Predisposing Factors</p> <p>Genetic</p> <ul style="list-style-type: none"> • Nonpigmented skin and hair on lower limbs • Chronic progressive lymphedema in shires and Clydesdales • Feathers on pasterns • Keratinization disorder <p>Environmental</p> <ul style="list-style-type: none"> • Climate or moisture • Poor stable or pasture hygiene • Alkaline soil • Sand (especially Arabians) <p>Iatrogenic</p> <ul style="list-style-type: none"> • Irritating topical products • Training devices • Poor grooming habits 	<p>Primary Factors</p> <p>Physical or chemical irritants</p> <ul style="list-style-type: none"> • Blistering • Creosote or motor oil • Treated bedding/shavings <p>Immune mediated</p> <ul style="list-style-type: none"> • Allergy (insect, contact) • Photosensitization • Vasculitis • Pemphigus complex <p>Infectious</p> <ul style="list-style-type: none"> • Dermatophytes • Spirochetes <p>Parasitic</p> <ul style="list-style-type: none"> • <i>Chorioptes</i> spp • Trombiculosis (chiggers) • <i>Pelodera strongyloides</i> • <i>Strongyloides westeri</i> larvae <p>Neoplastic</p> <ul style="list-style-type: none"> • Sarcoids (fibroblastic, verrucous) 	<p>Perpetuating Factors</p> <p>Secondary infections</p> <ul style="list-style-type: none"> • <i>Staphylococcus</i> spp • Botryomycosis • <i>Dermatophilus congolensis</i> • Fusiform bacteria • <i>Malassezia</i> spp <p>Pathogenic skin changes</p> <ul style="list-style-type: none"> • Trauma • Insect bites • <i>Culicoides</i> spp, tick bites <p>Environmental</p> <ul style="list-style-type: none"> • Ultraviolet light exposure • Cold
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scales, and crusts with potential epidermal hyperplasia. Pruritus and pain are variable.

- The **exudative form** (grease heel, dew poisoning; Figure 2) is characterized by erythema, erosion,



Figure 1. Mild form of equine pastern dermatitis (scratches, mud fever, mud rash). (University of Florida case files)

alopecia, and serous to purulent crusting dermatitis. Epidermolysis and vasculitis are often involved.

- The **chronic proliferative form**^{2,6} (grapes, verrucous pododermatitis; Figure 3) is characterized by excessive granulation tissue (fibroblastic proliferation) that becomes cornified. Nodular proliferations of hyperkeratosis and lichenification occur. Fissures and papillomatous areas may develop and are common sequelae in draft breeds.^{2,6,7}

Photosensitization, both systemic and contact forms, may affect the pasterns of horses with white extremities.⁸ The most common cause of equine contact photosensitization is exposure to clover pastures; this form primarily involves the muzzle and distal extremities.⁵ Systemic photosensitization is subdivided into primary and hepatogenous causes. Primary systemic photosensitization occurs when a preformed or metabolically derived photosensitizing agent reaches the skin by ingestion, contact, or injection. Causes of primary photosensitization are St. John's wort (*Hypericum perforatum*), buckwheat (*Polygonum fagopyrum*), and perennial rye grass (*Lolium perenne*).^{8,9} Hepatogenous systemic photosensitization occurs when an increased concentration of phyloerythrin is in the blood as a result of liver disease. This



Figure 2. Exudative form of equine pastern dermatitis (grease heel, dew poisoning). (University of Florida case files)



Figure 3. Chronic proliferative form of equine pastern dermatitis. (Courtesy of Dr. Luc Beco, DECVD, Cabinet Vétérinaire, Belgium)

type of photosensitization is most commonly due to pyrrolizidine alkaloid toxicosis.^{5,8,9} All types cause dermatitis in the presence of ultraviolet light.

Chronic progressive lymphedema of draft horses, which is characterized by progressive swelling, hyperkeratosis, and fibrosis of the distal limbs in shires, Clydesdales, and Belgian draft horses, has been under investigation at the University of California, Davis¹⁰ (Figure 4). The clinical signs and pathologic changes are simi-



Figure 4. Primary lymphedema. (Courtesy of Drs. Hilde De Cock and Verena Affolter, University of California, Davis)

lar to those of chronic lymphedema (elephantiasis nostras verrucosa) in humans. Factors that have been proposed to contribute to this disease are abnormal functioning of the dermal lymphatic system, resulting in severe swelling and fibrosis; a compromised immune system; and secondary skin infections.^{10,11} Researchers suspect that a deficiency or abnormality in elastin, a connective tissue component, is the underlying element and may cause lymphatic degeneration in these horses. The lesions do not respond well to therapy. As the disease progresses and becomes more chronic, enlargement of the lower extremity becomes permanent, and the swelling is firm during palpation.¹¹ Progressive skinfolds and nodule formation are first noted on the caudal aspect of the pastern, affecting both pigmented and nonpigmented skin; with chronicity, skinfolds and nodules encompass the entire lower extremity. Over time, the lesions cause a lack of mobility and often become traumatized during normal exercise. The prognosis is poor because of the lack of a specific treatment for the structural defect; development of secondary infections, which in turn cause further degrada-



Figure 5. Pastern leukocytoclastic vasculitis. (Courtesy of Dr. Anthony Yu, Ontario Veterinary College, University of Guelph)

tion of elastin; poor delivery and response to supportive symptomatic therapy; and development of systemic illness and debilitation as the disease progresses.^{10,11}

Pastern leukocytoclastic vasculitis (photoaggravated vasculitis) is an additional clinical cause of equine pastern dermatitis (Figure 5). This disease is poorly understood and affects mature horses. It is unique to horses and often, but not exclusively, targets unpigmented distal extremities.^{5,12} Pastern leukocytoclastic vasculitis is believed to be due to immunocomplex deposition on the blood vessel walls of the distal limbs, triggering vasculopathy and resulting in clinically well-demarcated circular, erythematous, exudative lesions with tightly adherent crusts.⁵ The prevalence of clinical signs in the summer suggests that it is photoaggravated. The medial and lateral aspects of the pasterns are most commonly affected. The lesions appear painful rather than pruritic. Edema of the affected limb(s) and lameness are common sequelae. Chronic cases may develop a rough or warty surface.⁵

DIAGNOSIS

A detailed history is very important in the dermatologic workup of equine pastern dermatitis. Important information includes the age of the patient at onset, the month the problem was first noted, and whether the condition has been seasonal and/or pruritic. Before the examination, additional questioning should investigate the use of topical medications or home remedies and whether the lesions improved or worsened with each treatment. Environmental conditions can be predisposing or primary factors in equine pastern dermatitis; therefore, when possible, a detailed description of the environment (bedding, pasture, sand, insect burden, moisture) should be provided or a personal inspection conducted. Long-term exposure to moisture, such as wet bedding or muddy pastures, is retained by the long hairs of the fetlock and pastern in draft horses and contributes to skin maceration.^{1,5-7} In cases of contact irritant or allergic dermatitis, all four pasterns are usually affected. To rule out potential infectious or zoonotic conditions, such as dermatophytosis (*Trichophyton equinum*, *Microsporum canis*, or *Microsporum gypseum* infection) and *Cheyletiella* infection, the clinician should consider whether other in-contact animals or humans are affected.

Pastern folliculitis or pyoderma is mainly caused by infection with *Staphylococcus aureus* or *Dermatophilus congolensis*.^{1,6,7,13} Initially, papules and/or pustules (rare) are noted with a *Staphylococcus* infection; however, with chronicity, they may not be seen. The lesions of *D. congolensis* infection typically include crusting and exudation; when the crusts are removed, they have a “paintbrush” appearance and the underlying skin surface is ulcerative.^{14,15} Chronic moisture and trauma to the epidermal barrier are required for this organism to cause infection.^{14,15} Because culture samples obtained by swabbing can produce misleading results due to surface contamination, biopsy for culture is needed for a definitive diagnosis. Ideally, an area of intact, nonulcerated skin (plaque or nodules) of the affected pastern is surgically scrubbed and deep biopsies are performed with sterile precautions. The specimens are then placed into culture medium and submitted to a microbiology laboratory for tissue maceration and culture and sensitivity testing.

Dermatophytosis rarely causes pastern folliculitis; however, it is important to rule out.^{1,6,7,13} A definitive diagnosis requires positive dermatophyte test medium (DTM) culture results in conjunction with positive microscopic identification of macroconidia. It is essential to add vitamin B complex to DTM in the office or at a commercial laboratory because *T. equinum* requires

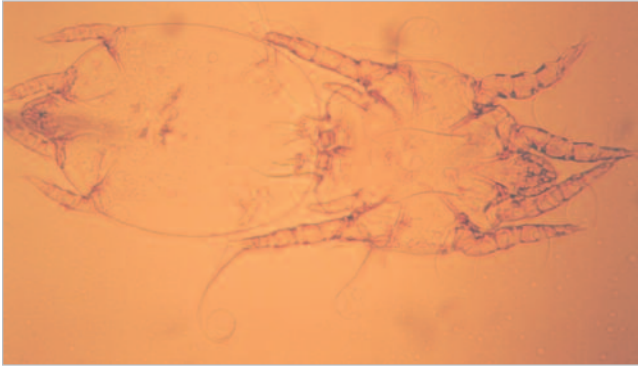


Figure 6. Male *Chorioptes* mite with nymph. (Courtesy of Dr. Ellis Greiner; University of Florida case files)

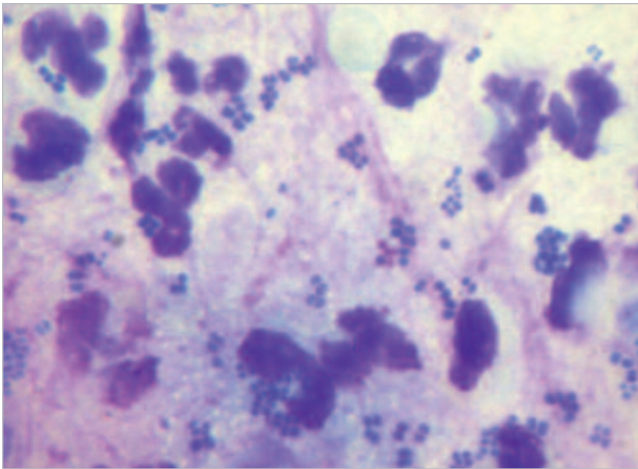


Figure 7. Degenerative neutrophils and intracellular and extracellular cocci. (Courtesy of Dr. Randall C. Thomas, Mount Pleasant, South Carolina)

niacin to grow. *M. canis* and *M. gypseum* grow readily on a standard fungal culture medium and DTM.

Chorioptic mange is a significant cause of pastern dermatitis in draft horses because of their long pastern feathers, which provide an ideal microenvironment for the mites.⁵ This condition is intensely pruritic. Affected horses may constantly rub the area and are often observed stamping their feet.⁵ *Chorioptes* infection should be highly suspected if other in-contact horses are affected and have clinical signs of pruritus. The mites typically are easily harvested from the skin.

DIAGNOSTIC TESTING

Superficial Skin Scrape

Multiple skin scrapings are key to ruling out superficial mites, especially *Chorioptes* spp. Superficial scrapings of

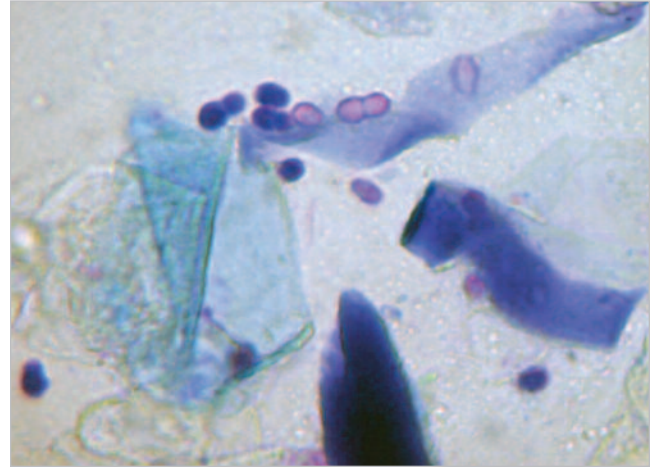


Figure 8. *Malassezia pachydermatis*. (Courtesy of Dr. Randall C. Thomas, Mount Pleasant, South Carolina)

crusts and debris can be collected using a dulled #10 blade and put into mineral oil on several glass slides. Other collection methods include the use of a stiff scrubbing brush or denture-type toothbrush to sweep the dander, crusts, and debris into a petri dish and the use of acetate tape preparations.^{4,6,7,16} The mites are often easier to detect during cool weather because of favorable environmental factors, and they tend to localize at the coronet during the summer (Figure 6). Because the mites are quite mobile, the slides should immediately be examined with a microscope using a 10× objective lens. Some authors¹⁷ suggest applying a small amount of insecticide to the slide to immobilize the mites.

Cytology and Diff-Quik Stain

Cytology is for immediate evaluation of secondary bacterial and *Malassezia* infections, which are considered perpetuating factors of equine pastern dermatitis. Scotch 3M Gloss Finish Multitask Tape stained directly with methylene blue or direct impressions using a glass slide, heat fixation, and Diff-Quik stain are simple, economical methods of collecting and processing diagnostic samples. Observation with a 100× microscope objective lens for bacteria engulfed within degenerative neutrophils (Figure 7) and variably stained budding yeast organisms (Figure 8) is often rewarding.^{4,5,7} Cytology may also provide insight into autoimmune, nodular, and neoplastic conditions. Acantholytic cells within a group of nonvacuolated neutrophils, in the presence of minimal to no bacterial infection, support further dermatohistopathologic investigation to obtain a definitive diagnosis of a pemphigus

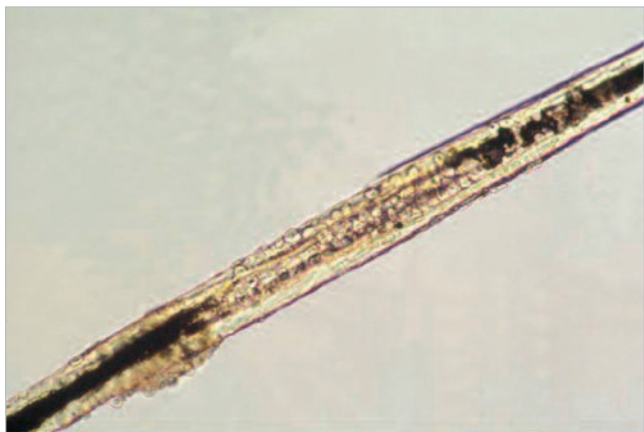


Figure 9. Arthrospores within a hair shaft. (Courtesy of Dr. Diane Lewis; University of Florida case files)

complex. Eosinophils should indicate a differential list composed of allergic, ectoparasitic, intermediate fungal, or foreign body causes. A homogenous population of cells with an increased mitotic index and atypical nuclear:cytoplasmic ratio predicates the need for a biopsy to rule out neoplasia.

Direct Examination of Hairs

A trichogram provides information on the stage of hair growth (anagen versus telogen) and can identify dermatophytic spores (Figure 9). These spores can be evaluated by adding one or two drops of a clearing agent (e.g., 10% potassium hydroxide solution) to affected plucked hairs, applying a coverslip, and then gently heating the slide for 15 to 20 minutes using the microscope lamp. Under a 4× objective lens, infected hairs appear pale and swollen and have identifiable small circular arthrospores within or on the surface of affected hair shafts.^{4,18} The potassium hydroxide preparation is time-consuming and can take several attempts to master; thus it is not often used in general practice.

Dermatophyte Test Medium Culture

When hair and crust samples are embedded in an in-house dermatophyte culture, a few drops of niacin (vitamin B complex) must be added to the DTM to satisfy the growth requirements of *T. equinum*. If samples are submitted to a commercial laboratory, contact the microbiologist to ascertain whether niacin is contained within or added to the culture medium. When preparing the site to take samples, use isopropyl alcohol to cleanse the hairs of saprophytic (clinically irrelevant) fungi. The

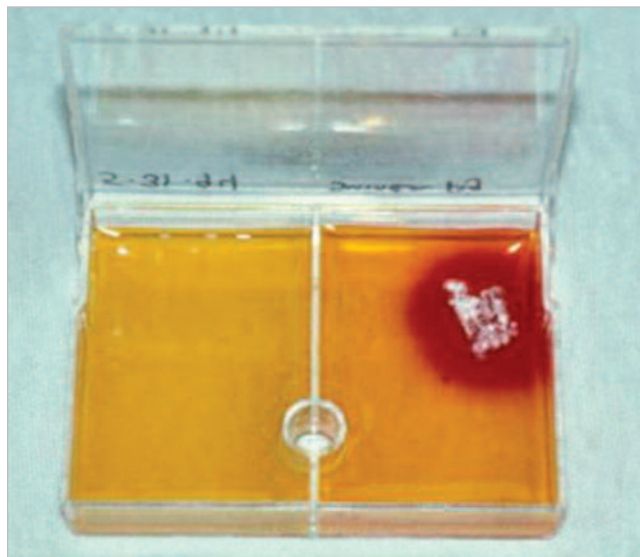


Figure 10. DTM culture color change to red concurrent with colony growth. (University of Florida case files)

alcohol must be allowed to dry before collection to avoid a false-negative result. DTM helps suppress growth of saprophytes and contaminant bacteria because it contains chlortetracycline, gentamicin, and cycloheximide.^{4,18,19} Dermatophytes metabolize the protein first, creating alkaline metabolites that interact with the phenol red pH indicator, resulting in a color change to red, along with colony growth. False-positive color changes occur when saprophytes have exhausted the carbohydrate source on the plate and then use protein, causing a late color change to red.¹⁸ Seven to 14 days of growth on the medium are typically required to visualize macroconidia. Once the color change to red has been identified with concurrent colony growth (Figure 10), microscopic examination is important to confirm the diagnosis. Use clear cellophane tape and press lightly onto the colony within the culture medium to collect the macroconidia. Then apply one or two drops of lactophenol cotton blue, or methylene blue (last dip in Diff-Quik stain) if the former is not readily available, and examine the tape with a microscope under a 40× objective lens.¹⁹ If no macroconidia are visible, wait a few days for the colony to mature; then repeat the procedure.

Dermatophilus congolensis Preparation

To identify *Dermatophilus* spp, peel off a crust, clip the excess hair from the sample, and place it into one or two drops of saline on a clean slide. Allow the sample to macerate (soften) for 15 minutes, and remove the larger

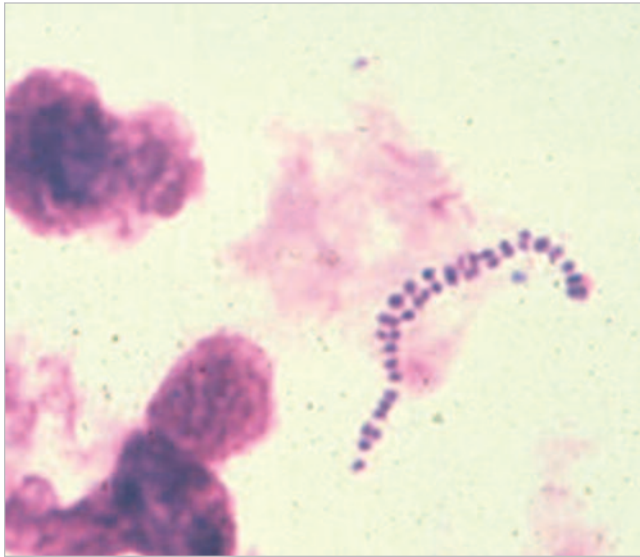


Figure 11. *Dermatophilus congolensis*. Note the “railroad track” orientation. (University of Florida case files)

pieces. Crush the remaining material on the slide and allow it to air-dry; then heat-fix the slide, stain it with Diff-Quik or methylene blue, allow it to dry, and examine it under a microscope with a 100× objective lens. Oil immersion should allow visualization of filamentous, branching, parallel rows of cocci-shaped bacteria in a “railroad track” orientation^{4,19} (Figure 11).

Biopsy

Biopsy should be considered if immune-mediated disorders, nodular or draining tract lesions, or neoplastic conditions are suspected. Biopsies are also recommended when treatment has been pursued and failures or relapses have occurred.¹⁹ In most cases, especially when pastern leukocytoclastic vasculitis is suspected, skin biopsies should be interpreted by a dermatohistopathologist with an interest in equine skin diseases.⁷ Acute changes, including leukocytoclastic vasculitis, thrombosis, and vessel wall necrosis, are often scarce and can easily be overlooked.^{5,7} If severe secondary bacterial dermatitis is present (Figure 12), administration of antibiotics for a minimum of 21 days is recommended before performing a biopsy to provide a clearer picture of the underlying cause.¹⁹

Biopsy for Culture

If a bacterial or fungal infection is suspected or is not responding to appropriate therapy, a biopsy is warranted.¹⁶ When a biopsy specimen is obtained for



Figure 12. Feathering and coronary band dermatitis. (University of Florida case files)

culture, surface contaminants need to be eliminated by clipping the hair and performing a surgical scrub. The biopsy specimen should be obtained in as sterile a manner as possible and submitted in a sterile container or medium. The specimen should be sent to the laboratory as soon as possible to minimize the growth of superficial contaminants.

Complete Blood Count and Chemistry Panel

Evaluation of hematologic and biochemical parameters is useful to rule out hepatogenous photosensitization disorders and metabolic illnesses and to obtain a baseline before treatment of an immune-mediated disorder.⁷

CLINICAL MANAGEMENT

Choosing the appropriate therapy involves recognition and identification of predisposing, perpetuating, and primary factors.⁷

Environment

Environmental modifications to minimize exacerbation of a primary underlying problem include:

- Avoiding pastures and paddocks with mud, water, or sand (e.g., Arabian horses and sand)^{1,6,7}
- Keeping horses in clean, dry stalls during wet weather
- Avoiding turnout before the morning dew has dried^{6,7}
- Using an alternative source of bedding (treated or aromatic types of wood shavings contain chemicals that can cause contact hypersensitivity) if contact

allergy (affecting all pasterns) is suspected^{1,6,7,13}

- Clipping excessive hair over the pasterns, if present, to decrease moisture retention^{1,6,7}
- Avoiding ultraviolet light exposure by stabling or using leg wraps if pastern leukocytoclastic vasculitis is suspected⁶⁻⁹

Topical Therapy

Antibacterial

In equine pastern dermatitis, secondary bacterial infections with *Staphylococcus* spp are common sequelae that complicate the diagnosis.^{1,2,5,7} Antibacterial shampoos can help control overcolonization of bacteria when used daily for 7 to 10 days, then two to three times weekly (see box on this page). Available active ingredients include benzoyl peroxide (2%), ethyl lactate, and chlorhexidine (2%).^{2,4,5,7} A topical ointment containing 2% mupirocin has excellent penetration into the epidermis and can be used for both *Dermatophilus* or staphylococcal bacterial infections.² Close clipping and cleansing are paramount to the success of any ointment.

Providing a dry, stable environment is often the most effective treatment. Some dermatologists recommend using a padded, water-repellent bandage (changed every 1 to 2 days). Facilitator (Blue Ridge Pharmaceuticals) is a hydroxyethylated amylopectin liquid bandage applied every 1 to 3 days after cleansing.^{2,7} If lesions are exudative, astringent solutions, such as lime sulfur (4 to 6 oz/gal) or aluminum acetate solution, can be used.

Antifungal

Lime sulfur dips and sprays can be used for localized treatment of the pastern for dermatophytes and mites. Enilconazole is a topically labeled treatment for dermatophytosis in horses in many countries other than the United States and has shown good success.^{2,7} Shampoos containing a combination of miconazole or ketoconazole and chlorhexidine demonstrate synergistic activity and improved resolution of dermatophyte infections.

Steroids

Topical steroids can be used for immune-mediated conditions such as pastern leukocytoclastic vasculitis or pemphigus. Triamcinolone spray (0.015%; Genesis, Virbac) and/or hydrocortisone (1%) leave-on conditioner (Resicort, Virbac) can be used in conjunction with systemic immunomodulators to treat these conditions. In addition, good success has been noted with topical betamethasone (1%) or alclometasone (0.05%) applied to the lesions.²

Topical and Systemic Therapies for Equine Pastern Dermatitis

Antibacterial shampoos

- Benzoyl peroxide (2%; Micropearls, Vétquinol; SulfOxyDex, DVM-IVAX; Pyoben, Virbac)
- Ethyl lactate (Etiderm, Virbac)
- Chlorhexidine (2% and 4%; Chlorhexiderm, DVM-IVAX; Hexadene, Virbac; Equine Medicated Shampoo, Vet Solutions)

Topical antibacterial ointment

- 2% mupirocin ointment (Bactoderm, Pfizer)

Antifungal shampoo, sprays, and dips

- Miconazole shampoo (1%; Micropearls, Vétquinol)
- Miconazole (2%), chlorhexidine (2%; Malaseb, DVM)
- Ketoconazole (1%), chlorhexidine (2.3%; Ketochlor, Virbac)
- Enilconazole (Imaverol)
- Lime sulfur concentrate (Lym Dyp, DVM)
- Selenium sulfide (Selsun Blue, Abbott Laboratories)—effective against *Malassezia* spp only

Topical steroids

- Triamcinolone (0.015%; Genesis Spray, Virbac)
- Hydrocortisone (1%; Resicort, Virbac)
- Betamethasone valerate (1%)
- Alclometasone dipropionate (0.05%; Aclovate, GlaxoSmithKline)

Systemic antibiotic medications

- Trimethoprim-sulfamethoxazole (Bactrim, Roche)
- Enrofloxacin (100 mg/ml; Baytril, Bayer Animal Health)

Systemic antifungal medications

- Ketoconazole (200 mg; Nizoral, Janssen Pharmaceuticals)
- Itraconazole (100-mg capsule; Sporanox, Ortho Biotech)
- Fluconazole (50, 100, 150, and 200 mg; Diflucan, Pfizer)
- Griseofulvin powder (Fulvicin-U/F Powder, Schering-Plough)

Antiparasitic therapies

- Ivermectin (1% solution; Eqvalan, Merial; DVMectin, DVM-IVAX)
- Fipronil spray (0.25%; Frontline, Merial)
- Selenium sulfide (Selsun Blue, Abbott Laboratories)
- Lime sulfur concentrate (Lym Dyp, DVM-IVAX)
- Malathion (0.5%)
- Coumaphos (0.06%)
- Permethrin (2%; Flypel, Virbac; Knockout LA, Virbac)

Immunomodulatory medications

- Dexamethasone (0.25-, 0.5-, 0.75-, 1.5-, and 4-mg tablets)
- Dexamethasone (10-mg/15-g packets; Azium, Schering-Plough)
- Prednisolone (Delta-Cortef, Upjohn)
- Pentoxifylline (400 mg; Trental, Hoechst-Roussel Pharmaceuticals)

Key Points

- Addressing the predisposing, primary, and perpetuating factors when working up a case of equine pastern dermatitis is important in obtaining a positive outcome.
- Clydesdales, shires, and Belgian draft horses with severe chronic progressive equine pastern dermatitis have recently been studied at the University of California. The clinical signs and pathologic changes in these horses are similar to those of chronic lymphedema or elephantiasis nostras verrucosa in humans.
- Pastern leukocytoclastic vasculitis (photoaggravated vasculitis) is a clinical cause of equine pastern dermatitis, is poorly understood, and affects mature horses. It is unique to horses and most often affects unpigmented distal extremities. Pastern leukocytoclastic vasculitis is an immune-mediated vasculitis, is diagnosed by dermatohistopathologic examination, and requires immunomodulatory or immunosuppressive therapy.

Systemic Therapy

Antibiotics

The antibiotics most commonly used in horses are trimethoprim-potentiated sulfonamides (15 to 35 mg/kg PO q12–24h for 2 to 3 weeks), often in conjunction with topical antibacterial shampoos^{2,4,7,16} (see box on page 225). Adverse reactions include drug-induced urticaria, pruritus, exfoliative dermatitis, erythema multiforme, anemia, and leukopenia. In horses that are intolerant to sulfonamide-based medications, enrofloxacin (the injectable form; 7.5 mg/kg PO q24h) has been used with success.^{2,7,20,21} This drug should be avoided in foals and growing horses because of its potential for chondrotoxicity and, therefore, reserved for severe staphylococcal infections.

Antifungals

Systemic antifungal therapy is often unnecessary in horses. Griseofulvin powder is available for horses; however, no pharmacokinetic data have been published for griseofulvin, and its efficacy is questionable.^{6,18} Ketoconazole, itraconazole, and fluconazole are effective for the systemic treatment of dermatophytosis in humans, cats, and dogs. These agents are not currently approved in the United States for use in horses.¹⁸ Ketoconazole (30 mg/kg/day) has very low absorption (23%) from the gastrointestinal tract in horses and can be very expensive.²² Itraconazole (5 to 10 mg/kg/day) has been used in horses

with variable success.^{2,7,18,23} One published pharmacokinetic report of fluconazole use in horses recommended a single loading dose of 14 mg/kg followed by 5 mg/kg/day.²⁴ The cost of these medications limits their use; however, recent patent expirations have allowed more affordable generic products.

Immunosuppressive or Immunomodulatory Therapy

Conditions such as pastern leukocytoclastic vasculitis and pemphigus foliaceus often require administration of immunosuppressive doses of steroids to control the immune-mediated reaction and decreased exposure to ultraviolet light to minimize photoaggravation of the disorder.^{2,5,7} Typically, dexamethasone (0.1 to 0.2 mg/kg) or prednisolone (2 mg/kg) is administered daily for 7 to 14 days, then gradually tapered to the lowest dose required to maintain remission of the lesions.^{2,7} Pentoxifylline (8 to 10 mg/kg PO q8–12h), a phosphodiesterase inhibitor, has immunomodulatory properties, including inhibition of tumor necrosis factor- α , interleukin-1, and interleukin-6; thus it is useful as a steroid-sparing agent.²⁵ In addition, pentoxifylline increases erythrocyte deformability and platelet aggregation, thereby inhibiting thrombosis and improving wound healing.^{2,25} Long-term control of immune-mediated equine pastern dermatitis can often be achieved using topical steroids and/or pentoxifylline once the lesions have been controlled.

Antiparasitic Therapy

Topical organophosphates (malathion [0.5%], coumaphos [0.06%]) have been historically used as effective ectoparasiticides; however, because of concerns regarding toxicities and the environment, other treatment options should be considered. Permethrins at concentrations greater than 2% have both insecticidal and significant repellent activity. Therefore, these products are most useful in preventing *Culicoides*- and tick-related equine pastern dermatitis. Selenium sulfide shampoo followed by lime sulfur (6 oz/gal) sponged on every 5 days for 1 month has been successfully used to treat parasites embedded within equine pastern dermatitis lesions with significant amounts of crust or debris.^{4,7,17} Although a recent European study demonstrated that one treatment with fipronil spray (0.25%) was effective against *Chorioptes bovis* infestation, this use is considered to be off-label.¹⁶ Because these mites can live off the host for up to 70 days, environmental

decontamination of the barn, stalls, bedding, tack, and grooming equipment is essential. Ivermectin (1%) solution (300 µg/kg PO weekly for four doses, based on feeding habits) is most often the miticidal treatment of choice because topical treatments are labor intensive.^{2,7,16,17} To minimize treatment failures and relapses with contagious conditions, all in-contact animals must also be treated.

CONCLUSION

The prognosis of equine pastern dermatitis depends on identification of the underlying cause and the chronicity of the condition. The clinical presentation can vary from very mild to chronic and very proliferative. Encompassing predisposing, primary, and perpetuating factors in the diagnostic workup and treatment plan can enhance the likelihood of a positive outcome. A biopsy should be considered if immune-mediated disorders, nodular or draining tract lesions, or neoplastic conditions are suspected.

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ARTICLE #2 CE TEST

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1. The most common location for early equine pastern dermatitis is the

- forelimbs at the caudal aspect of the pasterns.
- forelimbs at the lateral aspect of the pasterns.
- hindlimbs at the caudal aspect of the pasterns.
- hindlimbs at the medial aspect of the cannon.

2. _____, which occurs in humans and can be confirmed through biopsy, has recently been identified in shires, Clydesdales, and Belgian draft horses with severe chronic proliferative equine pastern dermatitis.

- D. congolensis* infection
- Lymphedema or elephantiasis
- Dermatophytosis
- Pastern leukocytoclastic vasculitis

3. _____ is one of the most common perpetuating factors that have been identified in equine pastern dermatitis.

- Environmental moisture
- Malassezia* dermatitis
- Secondary bacterial infection
- C. bovis* infestation

4. Pastern leukocytoclastic vasculitis is unique to horses and

- most often involves the unpigmented distal extremities.
- involves all unpigmented areas, including the blaze and muzzle.
- does not involve worsening of the lesions or clinical signs.
- does not involve ultraviolet light.

5. The bacteria that most commonly contribute to equine pastern dermatitis are

- Pseudomonas* and *Staphylococcus* spp.
- D. congolensis* and *T. equinum*.
- Staphylococcus* and *Proteus* spp.
- Staphylococcus* spp and *D. congolensis*.

6. What growth requirement must be added to DTM to culture *T. equinum*?

- vitamin C
- vitamin A

- niacin (vitamin B complex)
- vitamin K

7. Which statement regarding dermatophyte growth on a DTM plate is correct?

- Macroconidia are produced by dermatophytes and grow in both infected tissue and DTM.
- Dermatophytes first use carbohydrates as nutrients in the medium, thereby creating alkaline metabolites and causing a color change to red; once the carbohydrates have been depleted, the dermatophytes use protein.
- Dermatophytes first use protein as a nutrient in the medium, thereby creating alkaline metabolites and causing a color change to red; once the protein has been depleted, the dermatophytes use carbohydrates.
- When dermatophytes are cultured, the colony growth is always white.

8. Which flea product has been proven to be successful in treating *C. bovis* equine pastern dermatitis?

- imidacloprid
- fipronil spray
- 2% permethrin spray
- lufenuron

9. Which drug is classified as a phosphodiesterase inhibitor and has immunomodulatory properties that have been successful in treating pastern leukocytoclastic vasculitis?

- trimethoprim-sulfamethoxazole
- ketoconazole
- pentoxifylline
- griseofulvin

10. Which is important when culturing a sample from an area affected by equine pastern dermatitis?

- Swab the surface and send it to a laboratory for bacterial culture.
- Clip the hair, scrub the superficial area, and use sterile procedures while performing a biopsy.
- Collect the sample from the edge of the lesion.
- Remove and submit crusts from the lesion.