Infectious Folliculitis and Dermatophytosis

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KEYWORDS

• *Staphylococcus* • Methicillin-resistant *Staphylococcus aureus* • *Dermatophilus* • Rainrot • Dermatophytosis • Ringworm

KEY POINTS

- Bacterial, dermatophilosis, and superficial ringworm infections are common skin diseases noted in equine dermatology.
- The ability to recognize and accurately diagnose the skin condition is key to selecting an appropriate and successful treatment regimen.
- Addressing underlying etiology, environmental management, and infection control play a crucial role in preventing relapse of clinical signs.

BACTERIAL FOLLICULITIS

Staphylococcal Folliculitis

Staphylococci are common components of the commensal microbiota of the skin and mucous membranes, but are also important opportunistic pathogens. A wide variety of staphylococcal species can be found in or on healthy horses, and these differ greatly in their clinical relevance. It is reasonable to assume that 1 or more staphylococci can be found in or on virtually every healthy horse, typically with no clinical impact. However, staphylococci are leading causes of opportunistic infections that arise secondary to breaches in the normal physical and immunologic protective mechanisms.

An area of particular concern with staphylococci is their tendency to become resistant to antimicrobials. In particular, the emergence of methicillin-resistant staphylococci has caused much concern for both animal health and zoonotic infection. Although methicillin-resistant staphylococci are not inherently more virulent than their susceptible counterparts, they may be difficult to treat, outbreaks may occur, and zoonotic infections are of concern.

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Etiology

The *Staphylococcus* genus consists of a large number of different species, including minimally virulent commensals and important opportunistic pathogens. Staphylococci can be differentiated into coagulase-positive and coagulase-negative species (**Table 1**). Coagulase-positive species are the most important clinically, with *Staphylococcus aureus* being the most common cause of staphylococcal disease in horses.[1] However, although it is the most important staphylococcal pathogen it can be found on the skin, mucous membranes, or gastrointestinal tract of a small percentage of healthy horses. The canine-adapted *Staphylococcus pseudintermedius* is the leading cause of bacterial folliculitis in dogs and can also cause disease in horses,[2–4] although this appears to be rare. There is some concern that *S pseudintermedius* may be an emerging pathogen in horses or that it might be misidentified as *S aureus* by diagnostic laboratories, but it is probably an uncommon cause of infection.

Staphylococcus hyicus, a coagulase-variable species, is most often associated with exudative dermatitis in pigs (greasy pig disease), but has been implicated in pastern dermatitis in horses.[5,6] Experimental infection of horses can produce exudative skin lesions,[6] so this species should be considered potentially pathogenic. However, it is an uncommon cause of disease in horses.

Numerous different coagulase-negative staphylococci (CoNS) can be found in or on horses (see **Table 1**), and most healthy horses harbor multiple different CoNS species. Typically, CoNS are of limited virulence and predominantly cause infections in compromised hosts, but infections can occur in immunocompetent animals. Because of the commonness of CoNS in healthy horses and their limited virulence, there may be difficulty differentiating infection from contamination or colonization.

In the past 10 to 15 years, emergence of methicillin-resistant staphylococci has been identified in horses, a factor that has been accompanied by both animal and human health concerns. Methicillin-resistant staphylococci are resistant to virtually all β-lactam antimicrobials (penicillins, cephalosporins, carbapenems) by virtue of the *mecA* gene, and they often have acquired resistance to various other antimicrobial classes. These pathogens can therefore be difficult to treat and are refractory to most drugs used for empiric therapy in horses. Methicillin-resistant *S aureus* (MRSA) has received the most attention in horses because of its ability to cause infections,[7–12]

Table 1
Examples of coagulase-positive and coagulase-negative staphylococci that can be found in horses

Coagulase Positive	Coagulase Negative
S aureus	*S epidermidis*
S pseudintermedius	*S haemolyticus*
S delphini	*S warneri*
S hyicus[a]	*S xylosus*
	S equorum
	S sciuri
	S felis
	S simulans
	S chromogenes
	S cohnii
	S capitis

[a] Can be coagulase positive or negative.

Data from Refs.[2,6,49–52]

along with the potential transmission of MRSA between horses and humans,[12–16] and because it appears that MRSA is endemic in the horse population in many regions.[13,16–19] As with methicillin-susceptible *S aureus*, MRSA can be carried by healthy horses,[16,17,20–22] with colonization being much more common than clinical infection. It is apparent that a few major MRSA clones are established in the horse population internationally. One is sequence type 8 (ST8), particularly a strain called USA500 and Canadian epidemic MRSA-5 (CMRSA-5). This human epidemic clone has been found in horses in North America and Europe, and appears to have established itself as an endemic strain in some regions.[12,13,16,19] Recently, the livestock-associated ST398 MRSA has been identified in healthy and diseased horses predominantly in Europe.[13,17,22,23] Other strains, particularly human epidemic clones, have also been found less commonly.

Pathophysiology

Staphylococci are opportunistic pathogens, and primary staphylococcal skin disease occurs rarely (if ever). Factors that affect the body's normal immunologic or physical defenses are critical predisposing factors. Accordingly, wounds, surgical incisions, inflammatory skin conditions, immunosuppressive disorders (eg, Cushing's disease), and skin damage from tack, trauma, or excessive moisture are presumed to be important risk factors for staphylococcal infections. Risk factors for staphylococcal folliculitis, however, have been poorly explored in horses.

Clinical Signs

Staphylococcal folliculitis can present in a variety of ways, ranging from very mild focal and relatively innocuous lesions to rapidly progressive, extensive, and painful disease. Initially, small (1–2 mm) lesions are present, with progression from papules to pustules. These lesions may not be noted initially but can rapidly enlarge and coalesce. Crusts may be present, often in a circular pattern that may lead to empiric (and unsuccessful) ringworm treatment. Epidermal collarettes or encrusted pustules may also be noted.

Pruritus is variable but is usually present, and may be severe. Self-trauma from pruritus may be present and can potentially obscure the underlying staphylococcal lesions. In some cases the affected areas are very painful and edematous, something that is strongly suggestive of staphylococcal dermatitis rather than dermatophilosis or dermatophytosis.

Methicillin resistance does not alter the clinical presentation or progression of disease. These strains are no more inherently virulent than their susceptible counterparts, but more severe disease can occur as a result of failure of empiric treatment.

Diagnosis

Achieving a definitive diagnosis is important because of differences in treatment and overall management of staphylococcal dermatitis in comparison with skin diseases that may have a similar appearance (eg, dermatophilosis, dermatophytosis, *Corynebacterium pseudotuberculosis*). Increasing resistance to commonly used antimicrobials also highlights the need for prompt culture and susceptibility testing. Further, the emergence of MRSA emphasizes the need for additional public health and infection control considerations that are best addressed as early as possible.

Careful clinical examination is required to characterize the disease and to identify optimal areas for sample collection. Small lesions may be overlooked if careful examination is not performed, especially is self-trauma has occurred. Collection of an appropriate diagnostic specimen is critical. Such collection is not always simple,

depending on the type and chronicity of lesions, and it is important to maximize the likelihood of recovering the offending organism rather than other bacterial commensals or contaminants. Ideally, samples are collected from intact pustules by opening the pustule with a sterile needle and depositing the material onto a culture swab. Swabs of the undersides of crusts can also be collected but are less preferable. Swabs of the outer surface of older lesions are of little diagnostic utility.

Cytologic examination of the specimen is useful, and often overlooked. Abundant neutrophils with intracellular and extracellular cocci should be present (**Fig. 1**). Bacterial culture and susceptibility testing is a key component of diagnosis and is crucial in guiding therapy. Culture and susceptibility testing should be performed if a good specimen can be obtained. These tests are particularly important when there is severe or rapidly progressive disease, when disease is recurrent, and if initial treatment has failed. Culture results should always be scrutinized carefully. Isolation of coagulase-negative staphylococci should be interpreted with care. Though potentially pathogenic, coagulase-negative staphylococci, including methicillin-resistant strains, are commonly found on healthy skin[24] and are common contaminants. Isolation of a CoNS along with another more convincing pathogen (eg, coagulase-positive *Staphylococcus, Dermatophilus*) should typically be interpreted as contamination. If a CoNS is the only bacterium identified, the diagnosis should be reconsidered, with treatment of the CoNS most justified when there is cytologic evidence of staphylococcal disease and there is no indication that other causes are involved.

Skin biopsies can provide useful information and are particularly important in severe, atypical, nonresponsive, or refractory cases. Multiple biopsies should be collected from areas with active (and ideally new) lesions. Biopsy samples should be submitted for histologic examination and culture.

One aspect of diagnosis that is often overlooked is determination of the underlying cause. Staphylococcal folliculitis is almost always a secondary problem, and efforts to identify health or management factors that predispose to infection are critical. This approach may involve combinations of diagnostic testing (eg, for Cushing's disease, allergies), environmental assessment (eg, stall, turnout areas), and evaluation of management (eg, blanketing, tack, uses that might result in skin trauma, bathing practices).

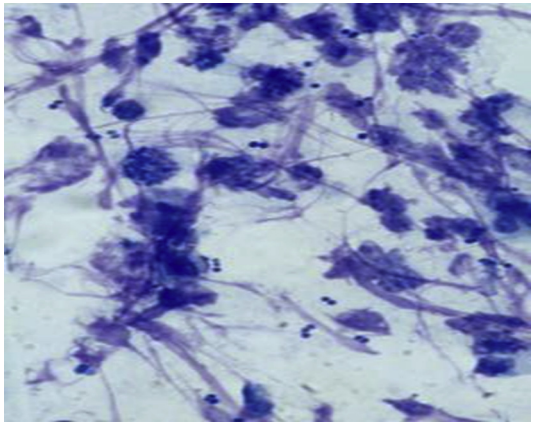

Fig. 1. Cytologic examination of the specimen is useful, and often overlooked. Abundant neutrophils with intracellular and extracellular cocci should be present.

Treatment

Scabs should be removed and the affected area scrubbed with a biocidal soap (eg, 2%–4% chlorhexidine). A contact time of 15 to 30 minutes should be provided before rinsing. Topical therapy is ideally performed daily for the first few days, with less frequent treatment as lesions start to resolve. Sedation may be required if lesions are painful. Analgesic therapy, typically with nonsteroidal anti-inflammatories, may also be required for patient comfort and to reduce self-trauma. Affected areas should be clipped to facilitate cleaning and topical therapy, and to help keep the area dry. Cold hydrotherapy may be beneficial in removing debris, reducing inflammation, and reducing edema. Topical application of antimicrobials such as mupirocin, fusidic acid, or silver sulfadiazine may also be useful with focal superficial infections. Care should be taken to ensure that the horse cannot ingest any topical antimicrobials because of the potential for antimicrobial-associated colitis. Local therapy may be adequate for superficial lesions that are not extensive. Systemic therapy is often required, and treatment should be based on susceptibility-testing results. Penicillin (20,000 IU/kg every 12 hours intramuscularly) may be effective, but the prevalence of β-lactamase production by staphylococci is high. Ceftiofur (ceftiofur sodium 2.2 mg/kg intravenously or intramuscularly every 12 hours, or ceftiofur crystalline free acid 6.6 mg/kg intramuscularly every 4 days) should be effective in the absence of methicillin resistance. Trimethoprim-sulfonamide (24–30 mg/kg by mouth every 12 hours) is often effective, but resistance may be present. There are typically a few viable options for MRSA and other methicillin-resistant staphylococci, but treatment must be guided by susceptibility results because of the potential for resistance to virtually any commonly used drug class.

Treatment should continue until 7 days beyond full clinical resolution. The frequency of adjunctive measures such as hydrotherapy can decrease over time as lesions and associated inflammation improve.

An additional aspect of treatment that cannot be overemphasized is attempting to address any identified or suspected underlying problems through medical, environmental, or management measures. Failure to address underlying risk factors may decrease the likelihood of successful treatment and increase the chance of recurrence.

Prevention

A critical aspect of prevention is reducing the incidence and severity of predisposing factors. In some cases, this may be simple while in others, impossible. Elimination of staphylococci from skin or mucosal reservoirs is not a viable (or desirable) approach. Staphylococci are important components of the commensal skin microbiota, and there is no indication that elimination of staphylococci from skin or mucosal surfaces is either possible or useful. However, there may be instances whereby periodic bathing in biocidal shampoo (eg, 2%–4% chlorhexidine) may be useful to reduce the staphylococcal skin burden on an animal with a flare-up of an underlying condition that might predispose to staphylococcal folliculitis. Prophylactic administration of antimicrobials is not recommended because there is no evidence of efficacy, and it might possibly be associated with increased likelihood of subsequent antimicrobial-resistant infection.

Public Health

Little information is available about risks posed by horses with folliculitis. It is reasonable to assume that there is some risk of zoonotic transmission from contact with

infected skin sites, as well as clinically normal skin sites and mucosal surfaces such as nasal passages where staphylococci may reside. *S aureus* is the main public health concern, mainly directed at MRSA. However, if MRSA can be transmitted between horses and humans, it is reasonable to assume that methicillin-susceptible *S aureus* can do the same. Yet the focus of attention is on MRSA because of the significant concerns about this pathogen in human medicine and the clear evidence of transmission of MRSA between horses and humans (in both directions). Zoonotic MRSA infections associated with horses have been reported,[12,14,15] and high rates of MRSA carriage have been identified in people who have contact with horses.[25,26]

Zoonotic risks of other staphylococci, including methicillin-resistant species, are limited. A very small number of methicillin-resistant *S pseudintermedius* (MRSP) infections have been reported in humans[27] and none from horses, but considering the high prevalence of this bacterium in dogs and the low apparent incidence of disease in humans, the risk posed by infected horses is probably limited. Nevertheless, the highly drug-resistant nature of most MRSP strains and the few reported human cases indicate that some degree of prudence is warranted. Close attention to basic hygiene practices is the most important measure.

Infection Control

Because staphylococci are common commensals, isolation of most horses with staphylococcal infections is unnecessary. Some basic infection control and management practices are indicated in routine cases, particularly good hygiene practices (especially hand hygiene), avoidance of contaminating common-use items, preventing the sharing of high-risk items such as blankets, wraps, and brushes, and other measures that would help reduce the risk of direct and indirect transmission of staphylococci. Staphylococci can survive in some environments for weeks, given the appropriate conditions, but they are killed by routine disinfection if done properly. Staphylococci are susceptible to most disinfectants, but the efficacy of disinfectants is often hampered by the surface material (eg, porous surfaces) and organic debris (eg, dirt, pus). Some disinfectants (eg, accelerated hydrogen peroxide) are more effective in contaminated environments, have shorter required contact times, and are compatible with most surfaces. Tack and other items that have been in contact with an infected horse should be laundered and hot-air dried, cleaned and disinfected, or discarded, depending on the surface type and value.

Measures to control MRSA on farms and in equine hospitals are poorly described, but general recommendations can be made based on information from human medicine and basic concepts of infection control and staphylococcal biology (**Box 1**).

DERMATOPHILOSIS

Introduction

Dermatophilosis, also referred to as rain scald or rain rot, is a relatively common exudative and crusting dermatitis in horses. Disease is usually sporadic, although multiple cases can occur on a farm, most likely because of common risk factors (eg, poor stable and turnout management).

Etiology

Dermatophilus congolensis is the etiologic agent. This facultatively anaerobic non–acid-fast gram-positive actinomycete can be found worldwide, particularly in tropical regions, and can infect a wide range of animal species. The bacterium has an unusual life cycle involving 2 forms, hyphae and zoospores. Zoospores are created from

Box 1
Measures for the control of methicillin-resistant *Staphylococcus aureus* on farms

Individual Horse Measures

- Isolate infected horses if possible
- Use dedicated water buckets, feed bowls, brushes, and other items for affected horses. Decontaminate before using on other horses or discard after use
- Use personal protective equipment (dedicated outerwear such as coveralls and gloves) when handling infected horses or entering their stall
- Wash hands or use an alcohol-based hand sanitizer after contact with the horse or its environment, including after glove removal
- Clean and disinfect the stall after resolution of disease
- Turnout only in a dedicated pasture or paddock

Group Measures

- Limit antimicrobial use
- Cohort horses into different risk groups and limit cross-contact (direct and indirect) between groups
- Have a good facility infection control program and preventive medicine program to reduce the risk of opportunistic infections
- Quarantine new arrivals
- Ensure that culture and susceptibility testing is performed on horses that develop opportunistic infections
- Ensure good general hygiene practices by farm personnel and other individuals that have contact with horses (eg, farriers, veterinarians)

coccoid cells that break off the filamentous hyphae, and these represent the infectious stage. Among domestic animals, infections are most often identified in horses, sheep, goats, and cattle, but this bacterium can cause disease in various wild and domestic species.

Epidemiology and Pathophysiology

Dermatophilus congolensis is an opportunist that causes disease secondary to factors that affect the skin integrity and/or immune response (eg, allergies, Cushing's disease, malnutrition). Skin damage (eg, a portal of infection) and/or alteration of the normal skin environment and/or immune system are required for disease to develop following exposure. Despite the commonness of this disease in horses, little research has been published regarding risk factors. However, it is apparent that skin damage from excessive moisture or insect bites and conditions that affect the immune system are the main predisposing causes. Cushing's disease is a particular concern because it can combine an excessive hair coat, increased sweating and moisture trapping, and decreased immune response.

There is limited information about the epidemiology of *D congolensis*, including the prevalence on healthy horses and main routes of transmission. The natural reservoir of this bacterium is unknown, but there may be a wide range of animal hosts. The bacterium can be found on the skin of healthy horses in the absence of disease, and carriers might be an important reservoir. However, the relative role of carriers and diseased horses in transmission is unclear. The bacterium can be transmitted by direct

and indirect (eg, fomites) contact. Crusts from infected horses pose the highest risk because of the large bacterial burden. Though perhaps relevant for short-term transmission on farms, the environment probably plays a minimal role in the broader epidemiology.

Clinical Signs

As with most exudative skin diseases, dermatophilosis starts with a papular stage and progresses to pustules. These early abnormalities are often not detected, and disease is not noted until the classic presentation with the development of epidermal collarettes, focal alopecia, coalescing of exudative lesions with matted hair ("paint-brush lesions") and thick crusts. Crusts are easily removable, with abundant pus underneath and potential bleeding caused by skin erosion (**Fig. 2**A).[28] As disease progresses, there is typically less purulent discharge and dry crusts may predominate. Pruritus is variable. Lesions may be regional or generalized, with lesion distribution being representative of the underlying cause, such as areas where excessive moisture is present (ie, dorsum, race, rump), where biting insects prefer, or where skin trauma is common (ie, girth and saddle area, lower limbs). Crust and scab formation is most common with longer hair coats, so in temperate climates the appearance of lesions in summer differs from that in winter.

Diagnosis

Clinical appearance is strongly suggestive, particularly in horses that are kept in wet environments or with other recognized risk factors. Impression smears of the undersides of crusts are very useful because of the characteristic "railroad-track" cytologic appearance of the bacterium (see **Fig. 2**B). Early lesions with ample exudate are optimal. Crusts can be plucked and pressed onto a glass slide. Alternatively, crusts can be mixed with saline and macerated before placing on a slide, a technique that is most effective for older or dry lesions.

Definitive diagnosis is based on isolation of the bacterium from crusts or biopsies, although this is uncommonly performed as an initial diagnostic test. Alternatively, histopathology may be used for diagnosis. A thick crust composed of palisading layers of parakeratotic stratum corneum, dried serum, and degenerating neutrophils is the most characteristic change (see **Fig. 2**C). A superficial folliculitis may be a prominent feature

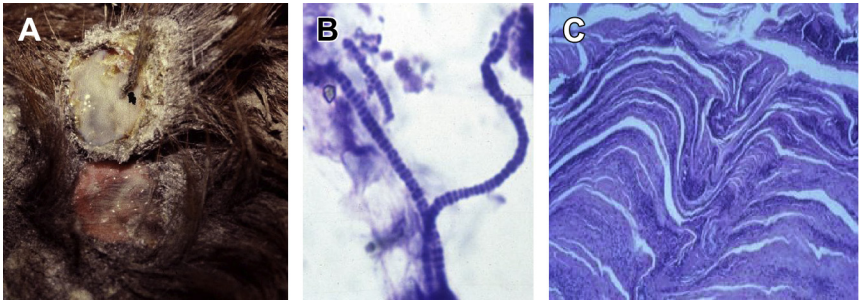

Fig. 2. (*A*) Thick crust with purulent exudate peeled off the skin from a patient with dermatophilosis. (*B*) Cytologic appearance of *Dermatophilus congolensis*. Note the filamentous appearance of chains of parallel "railroad-track" gram-positive cocci. (*C*) Thick crust from a patient with dermatophilosis, composed of palisading layers of parakeratotic stratum corneum, dried serum, and degenerating neutrophils, which constitute the most characteristic change. Organisms are typically sandwiched between the layers.

of the disease. In sections stained with Gram stain, the branching, filamentous organisms can be observed in the crusts and in the follicles. More extensive diagnostic testing should be considered to rule out other conditions or concurrent infections, particularly in atypical cases or those that fail to respond as expected.

Consideration of the underlying problem should also be part of the diagnostic process. This approach can involve investigation of environmental factors and management, or diagnostic testing (eg, diagnosis of Cushing's disease, intradermal allergy testing).

Treatment

Infection can be self-limiting if underlying problems are corrected, but specific therapy is usually indicated. Affected horses should be kept in a dry environment to facilitate skin healing. Crusts should be gently removed after soaking with biocidal shampoo or rinses (eg, 2%–4% chlorhexidine, benzoyl peroxide). Vigorous scrubbing should be avoided. Clipping affected areas and adjacent areas can facilitate topical therapy and maintain a dry skin environment. Antibacterial shampoos or rinses containing 2% to 4% chlorhexidine, benzoyl peroxide, accelerated hydrogen peroxide, or other nonirritating biocides should be used. A 10- to 15-minute contact time should be provided before rinsing.[28] Bathing should occur every 1 to 2 days initially, and at least once weekly until 1 week after clinical resolution. Focal lesions can be treated more often with biocidal sprays or ointments (eg, 4% chlorhexidine spray). Topical antimicrobials such as mupirocin or fusidic acid should also be effective, but it is unclear whether they confer much more benefit than topical biocides.

Systemic antimicrobials can be considered, but are rarely needed. It is ideal to reserve systemic antimicrobials for severe or refractory cases or for cases whereby topical therapy is not possible. Systemic therapy can be effective, but is accompanied by a risk of antimicrobial-associated complications (eg, diarrhea) and emergence of antimicrobial resistant pathogens. If systemic therapy is elected, bathing and removal of crusts should be performed if possible. *D congolensis* is almost always susceptible to a wide range of antimicrobials, with penicillin (20,000 IU/kg intramuscularly every 12 hours until 1 week after clinical resolution of lesion) being the drug of choice.

Concurrent measures to address any identified underlying problems are critical. This approach may involve changes in housing (eg, providing better cover outside, improving ventilation), management (eg, blanketing practices, improved nutrition), ectoparasite control, or treatment of underlying diseases (eg, allergies, Cushing's disease).

Prevention

Prevention involves good management and animal care practices that reduce predisposing causes, particularly ensuring that horses have access to proper shelter, and control of biting flies.

Public Health

Human infections have been reported, but are rare and mainly occur in tropical regions.[29] Although the potential for zoonotic transmission should not be dismissed, considering the incidence of this disease in horses, the limited use of strict infection control and hygiene measures around infected horses, and the paucity of reports of human infection (especially infections linked to horses), the risk is exceedingly low. Basic hygiene practices, particularly good attention to hand hygiene after handling infected horses, wearing some form of protective outerwear (eg, coveralls) when

bathing infected horses, and other basic barrier protection approaches, should be adequate. People with skin lesions or underlying skin disease should be particularly careful and use rigorous hand-hygiene practices. Alcohol-based hand sanitizers should be highly effective against this bacterium, and equally as effective as hand washing.

Infection Control

Multiple cases of dermatophilosis can occur in the same group of horses, although this relates largely to commonness of the underlying problem. Presumably this bacterium can be exchanged readily through direct or indirect (eg, fomites) means, and basic practices can reduce any risks. Crusts may pose the greatest risk, so care must be taken not to contaminate the environment or items during crust removal and patient treatment. Tack, blankets, and other items that have contact with the skin should not be shared between infected and uninfected horses. Items used on infected horses should be cleaned and disinfected after resolution of infection and/or before use on another horse. Many items are difficult to thoroughly disinfect, but the limited risk of transmission decreases concern about low-level residual bacterial contamination. Blankets, wraps, and similar items should be washed and hot-air dried. Items that cannot be laundered should be cleaned and ideally be sprayed with a routine disinfectant; however, surface compatibility must be considered. Routine cleaning and disinfection of the stall should be adequate. Infected horses do not require strict isolation; rather, keeping them away from other horses during the early treatment period, when bacterial burdens are highest, is a prudent measure. Particular care should be taken to avoid direct or indirect contact with horses that have underlying skin disease or immunosuppressive disorders (eg, Cushing's disease). A single case report of *D congolensis* placentitis and abortion in a mare exists,[30] so it may be prudent to adopt a stricter approach around pregnant mares, although the risk is presumably low.

Personnel working with an infected horse should use contact precautions to reduce the risk of transmission to other horses, low as it may be.

DERMATOPHYTOSIS

Dermatophytosis, commonly referred to as ringworm, is an important and highly contagious fungal infection caused by dermatophytes of the *Microsporum* and *Trichophyton* genera. Though of limited morbidity, ringworm can be problematic because of the potential for outbreaks, prolonged disease (at the individual or herd levels), cost and bother of treatment, and the potential for human infection.

Etiology

Dermatophytes are keratinophilic fungi. A wide range of dermatophyte species exist, which can be divided into zoophilic and geophilic groups. Most animal infections are caused by zoophilic species, with *Trichophyton equinum* and *Microsporum canis* (also referred to as *Microsporum equinum*) most common in horses,[31–33] and *T equinum* predominating in older horses.[32] Other species such as *Trichophyton mentagrophytes, Trichophyton verrucosum*, and *Trichophyton bullosum*, and the geophilic *Microsporum gypseum,* may be found occasionally in some regions.[32,34]

Epidemiology and Pathophysiology

The incidence of disease and prevalence of dermatophyte shedding are poorly characterized in horses. Dermatophytes can be found on a small percentage of healthy horses,[31] but it is unclear whether equine infections originate mainly from clinically

infected horses or clinically normal equine carriers. Horses can also be infected by other animal species, such as dogs, cats, and cattle, although the importance of these sources is unknown.

Zoophilic dermatophytes are transmitted by direct contact with infected animals or contact with arthroconidia (spores) in the environment or on fomites (eg, tack, blankets). Specific risk factors for horses have not been adequately investigated, although one study reported a higher incidence of disease on training farms than on breeding farms.[35] A study of Australian thoroughbreds identified a predominance of lesions in the girth area, supporting tack as an important route of transmission.[36] Young age (<3 years) and high humidity were also risk factors. Geophilic species are found in the environment, and contact with contaminated environments, particularly soil, is the main source of exposure.

Dermatophytes have keratinophilic and keratinolytic properties,[32] and clinical infection involves the superficial keratinized layers of the hair coat and skin.[37] After exposure, dermatophyte arthroconidia attach and germinate, then invade the stratum corneum, a process that takes approximately 3 days in human explant models[38]; and a similar time frame is likely present in horses. Clinical disease is usually apparent within 9 to 15 days of exposure.[39] Protease production digests keratin, providing a nutritional source and initiating skin and hair damage.[37] Differences in keratinase production can be present between strains, which may account for apparent differences in clinical virulence. Other factors also contribute to host damage, including the host immune response, host protease secretion, and various other likely pathogen and host factors.[37] An effective delayed-type hypersensitivity cell-mediated immune response is required for elimination of infection.[37] Animals with compromised immune systems may therefore develop more serious or more prolonged disease.

The reason why dermatophytes can be found on the skin and hair coat of animals without disease is unclear. It is possible that low-level exposure and an effective immune response result in temporary colonization with no disease, but this has not been adequately studied.

Clinical Signs

Classically, a circular area of alopecia and scaling with an erythematous margin is evident.[32] Lesions may grow centripetally, and with time there may be resolution of infection in central areas with new hair growth while active disease continues to extend outward (**Fig. 3**). However, this classic presentation is not always present, and affected animals can have variable distributions and shapes of affected areas. There may be single lesions or multiple lesions in a cluster or widely disseminated. The head, neck, and forelimbs are most commonly affected. Spread over the body may be rapid, particularly in young animals. Pruritus is uncommon but may be present in some animals. Close examination of affected areas may reveal papules and pustules,[28] depending on the age of the lesion. The classic appearance is not always present in horses, and in some cases the main signs are less pronounced, with broken hairs, small alopecic areas, and a hair coat that looks more “rough” than diseased.

Diagnosis

Consideration of dermatophytosis is important for any horse with dermatologic disease because of the variable clinical presentation and potential for rapid spread. Clinical appearance can provide a suspicion of ringworm, but confirmation should take place to rule out other similar-appearing conditions (eg, dermatophilosis, staphylococcal folliculitis) and atypical presentations of ringworm.

Fig. 3. Common dermatophytic lesions consist of a circular area of alopecia and scaling, with centripetal spreading and resolution of infection in central areas with new hair growth, while active disease continues to extend outward.

Wood's lamp can be used as a screening tool; however, false negatives are common because not all dermatophytes fluoresce. Negative results should never be used to rule out dermatophytosis. Wood's lamp is most effective as a monitoring tool when a fluorescing strain of *M canis (M equinum)* is known to be present in a group or on an individual. Fluorescence is best observed during early or active infection, when the entire hair shaft will fluoresce,[32] as later in disease only the most distal components will fluoresce. Wood's lamp can be useful for selection of optimal sites to collect samples for fungal culture.

Direct microscopic examination of infected hairs (trichoscopy) collected by plucking or skin scraping has provided variable success. After digestion with 10% to 20% potassium hydroxide to remove keratin and associated debris, dermatophytes can be noted in clusters or chains along the hair surface (**Fig. 4**).[32,40] Direct staining with Giemsa stain may also be used to visualize arthroconidia, although this is likely of lower sensitivity.

Adhesive tape impression (ATI) is used to collect and secure hair samples of affected and unaffected hairs, which are then placed onto a glass slide for microscopic evaluation. The sample is examined either directly using low-power (4×–10× objective) for evidence of ectothrix. Packing tape (such as 3M ScotchPad Packaging

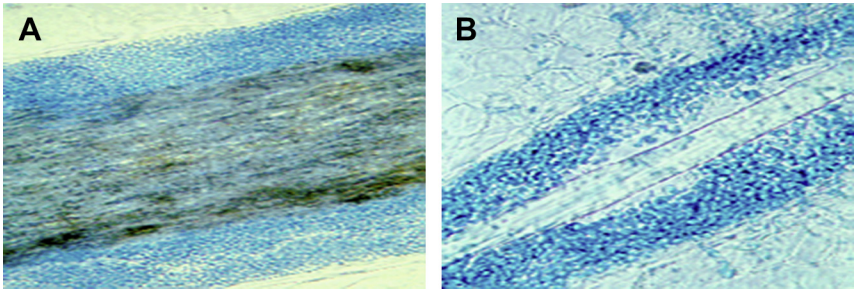

Fig. 4. Dermatophytes can be noted in clusters or chains along the hair surface with a lactophenol cotton blue stain used to enhance the spore along the hair shaft. (*A*) *Microsporum canis*. (*B*) *Microsporum gypseum*.

Tape Pad 3750PY) is used in preference to regular clear Scotch tape, as the former is stickier (especially useful for collecting affected hairs) and tends not to curl as readily when adhering to a slide. ATI is the sampling method of choice for dermatophyte evaluation, as it adheres fragile infected hairs rather than leaving the broken infected hairs behind when sampling with hemostats.

Fungal culture is the gold standard. Hair should be collected from clinically affected areas, with new lesions preferred. Hair is collected by plucking or toothbrush sampling. Biopsies can also be cultured, as can debris from skin scrapings. A diagnostic laboratory that has experience with dermatophyte isolation and identification is preferred. Selective media are required to prevent overgrowth of various environmental and commensal fungi. Commercial in-clinic assays are available. These assays can allow for a preliminary diagnosis based on colony morphology and color change of medium, but suspected positive cases should always be confirmed via microscopy or submission to a diagnostic laboratory to prevent false-positive diagnoses. An additional consideration is the biosafety requirements for in-clinic culture. Containment at biosafety level 2 should be used to reduce the risk of laboratory-associated infection. These practices are feasible in a veterinary clinic, but require consideration of physical layout, training, cleaning and disinfection, decontamination, and waste disposal.

Treatment

Ringworm is a self-limiting disease in immunocompetent animals,[32] although 1 to 4 months may be required.[41] Treatment typically is pursued to hasten recovery and reduce the risk of subsequent human or animal infections.

Topical and systemic options are available, and little objective information is available regarding optimal approaches in horses. Topical therapy can be effective alone or in conjunction with systemic treatment, and should be considered for all cases.[28] Affected areas can be clipped to facilitate treatment. Clippers and clipper blades should be disinfected after use. Scabs should be removed and disposed of in a manner that will not contaminate the environment. All lesions and the surrounding hair coat should be treated. Ideally the entire hair coat is treated because dermatophytes may be present widely over the body, not just at visibly affected sites. A wide range of topical options are available in rinse or shampoo formulations, including enilconazole 0.2% (Imaverol), natamycin, ketoconazole 1% to 2%, miconazole, chlorhexidine 2% to 4%, and combinations of ketoconazole and chlorhexidine.[28,42] A 2% lime sulfur solution is effective but is undesirable because of the odor and potential for staining of light hair coats.[32] Accelerated hydrogen peroxide may be another topical treatment option because of its efficacy against dermatophytes, but data are currently limited. Terbinafine 1% or miconazole 2% can be used as spot treatments but are not practical for treatment of the whole animal. Natamycin suspension is also useful for focal topical therapy.[41] Treatment intervals are not well described, and range from daily to weekly. At least 2 to 3 treatments per week should be administered initially.

Limited information is available about the safety and efficacy of systemic antifungals. Griseofulvin (5–10 mg/kg or 100 mg/kg by mouth every 24 hours) is occasionally used, although evidence of superiority to topical therapy is lacking. The variable reported dosing ranges question the efficacy of this product in equine dermatology. Griseofulvin is a teratogen, and should not be used in pregnant mares. Whereas medications such as itraconazole and fluconazole have been used to treat horses with systemic mycotic infections such as coccidioidomycosis and aspergillosis, there have not been any studies on their effectiveness in dermatophytosis. However, their safety record in horses in the face of the doses used (2–5 mg/kg every 12 hours) is

encouraging.[43–45] Terbinafine is widely used in dogs and cats, and although pharmacokinetics of this drug have been investigated in horses,[46] efficacy data are limited to a weak uncontrolled study of 2 horses that reported clinical cure (albeit for a typically self-limiting disease).[47] Alternatively, sodium iodide 20% may be given intravenously (250 mL/500 kg horse every 7 days, 1 to 2 times), although this also is contraindicated in pregnant mares as it may cause abortion.

Considering the cost and potential toxicity of oral antifungal medication, combined with the self-limiting nature of disease and relative ease of topical therapy, topical therapy alone is a reasonable approach, with oral treatment reserved for refractory or perhaps severe cases. Oral treatment is best reserved for situations whereby topical therapy cannot be performed adequately because of the horse's temperament or inability to bathe because of cold weather. Treatment typically is continued for 2 to 4 weeks after resolution of clinical signs, and after 2 negative cultures (collected 3–7 days apart) have been obtained.[41]

Prevention

There are no specific preventive measures. Vaccination has been shown to provide a reasonable degree of protection in horses,[48] but equine vaccines are not commercially available in most regions. General infection-control practices, including quarantine of new arrivals, preventing sharing of tack and other skin-contact items, and prompt diagnosis of suspected cases, are important, in addition to specific measures used in response to suspected or confirmed cases (see later discussion). Prophylactic topical treatment of exposed horses can be considered. If used tack is purchased, it should be cleaned and disinfected before use. Used blankets should be laundered and hot-air dried.

Public Health

Dermatophytosis is a common zoonotic disease, although horses are uncommonly implicated in human infections. The risk varies between different dermatophytes, with *M canis* posing the greatest risk.[32] Any infected horse poses a risk to humans through direct contact or through contact with contaminated fomites. The incidence of zoonotic ringworm in people in contact with infected horses is unknown, although all infected horses should be considered infectious.

Infection Control

Infected horses should be isolated to prevent direct or indirect contact with other horses and to facilitate the use of proper infection-control practices. Dermatophytes are spread through contact, not aerosol or airborne routes, so isolation within a main barn is acceptable as long as there is no potential for horse-horse contact around doors or over walls. If the potential for contact exists, temporary barriers should be erected or empty stalls should be left on either side. If infected horses are turned out, an individual pasture or paddock should be used, and there should be no potential for contact with other horses over fences. If affected horses are walked, personnel should be diligent to ensure there is no transient contact with other horses.

Dermatophytes can survive for months to years in the environment and on tack under the appropriate conditions.[32,36] Therefore, potentially contaminated areas and items should be cleaned and disinfected, and this should be done periodically during the treatment period. A thorough terminal cleaning and disinfection should be performed after resolution of infection. Disinfection of stalls may be difficult if unsealed wood, concrete, or dirt surfaces are present, but removal of as much organic debris as possible, thorough washing, and application of a disinfectant with

antidermatophyte activity (eg, accelerated hydrogen peroxide, 1:10 concentration of household bleach) should markedly reduce any dermatophyte burden and subsequent risk.

Items that have come into contact with infected horses should also be considered infectious. Items such as buckets and brushes should be soaked in disinfectant and thoroughly rinsed after 15 minutes' contact time. Blankets, wraps, and other items that can be laundered should be laundered and hot-air dried. Disinfection of tack can difficult because many items have porous surfaces. Tack should be thoroughly washed and sprayed with a disinfectant. Accelerated hydrogen peroxide is probably less damaging than bleach to surfaces, and is preferred. After 15 minutes' contact time, tack should be rinsed to remove residual disinfectant.

Contact with infected horses should be minimized. Personal protective equipment should be worn whenever horses are handled or when the stall is entered. The most important components are an item that covers the clothing (eg, coveralls) and is only used for the infected horse, and gloves. Protective outerwear should be stored so that it does not cross-contaminate other items, and should be donned and removed with care to avoid inadvertent contamination of the hands or other parts of the body. Hands must be washed after removal of gloves.

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