A considerable range of infectious actinomycetes and fungi are recognized as potential causes of disease of the skin and internal organs in many species and in all cases they offer significant diagnostic and therapeutic challenges to the clinician. The causative organisms involved and the treatment options in the two broad groups of fungal pathogens are also very different. Fungal skin infections in horses range from some very common superficial disorders such as dermatophytosis (ringworm) caused by almost ubiquitous *Trichophyton* and *Microsporum* fungi, to the exotic and difficult disorders related to secondary wound infections such as pythiosis (‘Florida leeches’) due to *Pythium* spp., and conidiobolomycosis (black-grained mycetoma) caused by cutaneous and subcutaneous infection with *Conidiobolus coronatus*. There is an increasing awareness, too, of the role of fungal skin infections on and within the skin. Some result in nodular fungal granuloma.

Classification of the fungal dermatological infections in other species is usually based on the location of the infection. This system is also applicable to equine mycotic disease (Table 7.1). Three broad groups of fungal skin disease can be identified:

1. **Superficial mycoses** are those in which the pathogen is confined to the stratum corneum (and does not affect hairs). As might be expected, there is little or no tissue reaction or inflammation and as result they tend to be rather benign. Some of the conditions are viewed as facultative pathogens, or even commensals, rather than primary pathogens and in either case there can be secondary seborrhoeic changes that result in excessive scale and flake on the skin surface. There is usually little immunological or cellular response associated with the organisms and no obvious seroconversion. A good example of this is *Malassezia* spp. infection, which has recently been recognized as a potentially significant skin infection in horses.

2. **Cutaneous mycoses** affect all keratinized tissue including hair, horn and skin, although most of the pathogens are confined to the non-living layers of the skin and the appendages. The organisms are capable of causing significant destruction of keratinized tissue and there are usually immunological responses in the host that result in obvious lesions and seroconversion with variable protective properties.

3. **Subcutaneous/deep mycoses** constitute a heterogeneous group of fungal diseases that are more deep-seated. They involve subcutaneous tissues, possibly in addition to the dermal/epidermal involvement. Whilst most of these remain localized (e.g. *Alternaria alternate*), some do spread insidiously to contiguous tissues, e.g. *Basidiobolus haptosporus* and *Conidiobolus coronatus* infections. Some spread via lymphatic vessels occurs, e.g. histoplasmosis (epizootic lymphangitis) due to *H. farcinosum* and sporotrichosis due to *Sporothrix schenckii*. There may be difficulties with diagnosis and treatment in this group of diseases. Seroconversion may take place but seldom seems to result in an effective protective process. Most of the conditions are chronic and progressive.

The deep mycoses are necessarily much more serious in most cases but are fortunately geographically restricted. Although some of the diseases are sporadic others occur in epidemics or are enzootic. For the most part, however, apart from the major disease epizootics, deep fungal infections are rare or very rare in horses.

Generally the clinical signs of cutaneous mycoses (both deep and superficial) are not pathognomonic in that they may closely resemble those elicited by other microorganisms and some other non-infectious pathological states. For example, some early dermatophytosis cases can closely resemble those elicited by other microorganisms and some other non-infectious pathological states. For example, early dermatophytosis cases can closely resemble those elicited by other microorganisms and some other non-infectious pathological states. For example, early dermatophytosis cases can closely resemble those elicited by other microorganisms and some other non-infectious pathological states. For example, early dermatophytosis cases can closely resemble those elicited by other microorganisms and some other non-infectious pathological states. For example, early dermatophytosis cases can closely resemble those elicited by other microorganisms and some other non-infectious pathological states. For example, early dermatophytosis cases can closely resemble those elicited by other microorganisms and some other non-infectious pathological states. For example, early dermatophytosis cases can closely resemble those elicited by other microorganisms and some other non-infectious pathological states.
### Table 7.1 The main fungal and actinomycete infections of the skin of the horse

<table>
<thead>
<tr>
<th>Group disease</th>
<th>Disease classification</th>
<th>Organisms (examples)</th>
<th>Pathogenicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial mycosis</td>
<td></td>
<td>Malassezia sp.</td>
<td>Secondary Facultative</td>
</tr>
<tr>
<td>Cutaneous mycosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dermatomycosis</td>
<td></td>
<td>T. equinum</td>
<td>Secondary (trauma)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T. verrucosum</td>
<td>Pathogenic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T. canis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>T. mentagrophytes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Microsporosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>M. equi</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>M. canis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>M. gypseum</td>
<td></td>
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<tr>
<td>Subcutaneous mycosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mycetoma (eumycotic)</td>
<td>Curvularia geniculata</td>
<td>Secondary (subcutaneous introduction/trauma) Pathogenic</td>
<td></td>
</tr>
<tr>
<td>Subcutaneous phaeohyphomycosis</td>
<td>Alternaria alternate</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Drechslera spicifera</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Pythium sp.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sporotrichosis</td>
<td>Sporothrix schenckii</td>
<td>Pathogenic</td>
<td></td>
</tr>
<tr>
<td>Subcutaneous zygomycosis</td>
<td>Basidiobolus haptosporus</td>
<td>Secondary (skin trauma)</td>
<td></td>
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<tr>
<td></td>
<td>Conidiobolus coronatus</td>
<td>Pathogenic</td>
<td></td>
</tr>
<tr>
<td>Systemic mycosis</td>
<td>Aspergillosis</td>
<td>A. fumigatus</td>
<td>Mucosal secondary pathogen</td>
</tr>
<tr>
<td></td>
<td>A. nidulans</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blastomycosis</td>
<td>B. dermatitidis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cryptococcosis</td>
<td>C. neoformans</td>
<td>Secondary pathogen</td>
<td></td>
</tr>
</tbody>
</table>
| Histoplasmosis               | H. farcinomus         | Pathogen requires subcutaneous implantation |}

Significant dermatological disease states are highlighted in bold.


*Examples are shown of the commoner reported organisms only. Rarely, others may be encountered.

Apart from the superficial dermatophytosis (ringworm) disorders, in which culture and direct microscopic examination of hair shafts and skin scrapings are diagnostic in most cases, the best definitive and practical procedure is biopsy and in some cases histological demonstration of the definitive organism. This also has some difficulty because the juvenile or early forms of some of the deeper mycoses are not ‘typical’. New methods of immunohistochemistry involving immunofluorescence and PCR can be used in some cases.

**Trichophytosis**

*Profile*

Cutaneous mycosis (ringworm) due to *Trichophyton* spp. is a very common, highly contagious disease which affects horses of all ages. Younger horses are naturally less resistant and take longer to recover than older ones. Transmission is by direct or indirect contact with a source of infection.

The most common species are *Trichophyton equinum* var. *equinum* (TEvE), *T. equinum* var. *autotrophicum* (TEvA), and less commonly *T. verrucosum* (most often from direct or indirect contact with infected cattle) and *T. mentagrophytes* (most often derived from contact with infected rodents and cats) (Pascoe 1979, 1984). *T. equinum* var. *equinum* tends to be the predominant species in the northern hemisphere while *T. equinum* var. *autotrophicum* tends to dominate in the southern hemisphere and the Antipodes.

The spores are highly resistant to environmental destruction and may persist in stables and on tack, etc. for many years. Most cases occur in winter months when horses are closely grouped and groomed heavily using shared or unhygienic tack, harness, clothing and equipment. Wet, warm weather has also been associated with outbreaks but sunshine is a significant inhibitor of the fungi in general. The distribution of lesions in some cases suggests that biting flies may be a significant vector.

Infection relies upon the presence of active (live) spores and mechanical skin abrasion (even if very mild) and this is the reason for most lesions developing on girths and saddle and jockey boot friction areas. The spores become vegetative in the damaged stratum corneum and the fungal hyphae penetrate the anagen hair follicles. In *Trichophyton*
species infection, the hyphae invade the hair shafts and relatively few spores are produced within the hair shafts (endothrix spores). As the fungal hyphae penetrate downwards towards the hair bulb, keratolytic enzymes are produced. These enhance and facilitate further penetration. Damage to the anagen hair shaft occurs so that the outer portion is shed; the fungus may be expelled in it. In order for the fungus to thrive the hair has to be actively growing and so as soon as the hairs enter the telogen stage the fungus cannot easily survive. At this stage the highly resistant spores are produced and may be shed into the environment with the hair shaft. Most cases resolve spontaneously as a result of hair shedding in telogen phase or as a result of hair breakage or following an immuno-excitatory inflammatory folliculitis; this can be a result of local hypersensitivity responses to the secretions of the fungus. In some cases the complex of secretory and induced inflammatory products results in a more florid inflammation — often with some mild irritation to the horse. Following infection, lesions are visible at around 7–21 days depending largely on the immune status of the horse. Reinfection of a single hair follicle and its hair shaft does not occur until the hair re-enters its anagen phase. If immunity is strong at this stage, the infection is unlikely to re-establish. Clinically the lesions may expand and continue to spread across the horse for some 2–4 months depending on the stage of hair growth and the extent of immunity. Immunity to *Trichophyton* spp. fungi is short-lived; there are some common antigens and so reinfection with another *Trichophyton* species is less likely. However, there does not appear to be any significant correlation between circulating antibody concentrations and the extent of resistance to reinfection. The natural antifungal effects of healthy untraumatized skin seem to be at least as important in the overall resistance to the disease. Repeated degreasing shampoos are probably not helpful because sebum has a significant protective property.

Immunocompromised horses (such as those on steroid treatment or clinical cases of pituitary pars intermedia dysfunction [PPID/Cushing’s disease]) are liable to recurrent, severe and often overwhelming infections.

The positive identification of the species involved (in all dermatophytosis cases) provides useful information on the likely source of infection. The treatment is unlikely to vary but as immunity is generally poor, avoidance of reinfection may depend on a combination of avoiding the source of the infection (where this can be achieved) and sterilization of the environmental challenges arising as a result of spore contamination of buildings, tack harness, rugs, etc.

**Key points: Trichophytosis**

1. Very common cutaneous mycosis due to *Trichophyton* spp. dermatophytes. Worldwide distribution. Spores are highly resistant so repeated infections occur in stables/yards. The fungus requires epidermal damage to gain entry and remains inside the hair follicle and on the hair shafts. The sites of infection reflect areas of superficial skin trauma such as tack and harness contact points. Generalized infection can follow simply from grooming with infected brushes or transferring the fungus to fresh sites on the same horse. The spores can survive for many years.

2. Early clinical signs are erect hairs, some local swelling/ oedema with mild exudate in a few cases. As all hairs are involved within a local area, complete shedding of hair occurs and lesions are easily and completely epilated leaving a silvery exposed epidermis. Abrasions from jockeys' boots and girths are common sites for infection.

3. Diagnosis is relatively simple clinically but differential culture is important for control measures. Biopsy can be helpful.

4. Treatment involves isolation of affected horses and careful hygiene to prevent spread between horses and to humans. Topical antifungal washes are effective and oral griseofulvin can be given; it should not be given to pregnant mares. Environmental control is also important to limit the risks of spread so fungicidal disinfectants are used as sprays and waxes for tack and harness, etc.

5. Control by vaccination is possible in some countries but early recognition and isolation of cases, and stable and personal hygiene are by far the best ways to prevent its spread. Individualized tack, harness and rugs, etc. are essential, especially in stables with a history of dermatophyte infection. When handling any case of dermatophytosis, gloves should always be worn.

**Clinical signs**

The earliest lesions appear as erect hairs in circular areas of 5–20 mm diameter (Fig. 7.1). There is often a degree of localized inflammation resulting in a thickening of the skin within the infected area; this can be urticarial in nature and there may be some exudate, which dampens the site. By day 7–10 post-infection hair can easily be plucked from the site (Fig. 7.2), leaving a silvery, slightly reddened circular

**Note**

Although most species of dermatophyte are in theory at least transmissible to humans, this appears to be much less common with the specific equine species. The bovine and pet species of dermatophyte can infect horses and then the infectivity to humans may be greater than with the equine species. Human disease does nevertheless occur and it is always useful to enquire about any human skin disease and if anything is recognized, the person must be referred to a medical practitioner (carrying a note of the suspicion for the animal). Therefore it is important to establish the species involved in an outbreak so that proper measures can be taken to control it amongst the horses and limit the associated human risks.

**Figure 7.1** Ringworm. Early lesions of *Trichophyton equinum* var. *equinum* infection in the girth area (attributed to a contaminated girth some 5–7 days previously).
area of exposed epidermis (Fig. 7.3). Hair loss also occurs naturally but this is less abrupt and so the lesions may not be obvious until 14–21 days. One of the cardinal signs is the ease with which the hair is removed – this is because most (probably all) hairs are affected within the lesion (Fig. 7.4).

The infected areas expand centrifugally and may lose the circular appearance, becoming diffuse and ill-defined.

Girth and shoulder/chest wall areas are common sites owing to infection from contaminated girths, riding boots, etc. (Fig. 7.5). Generalized infections are also common, particularly in younger horses (Fig. 7.6).

Different species of *Trichophyton* such as *T. verrucosum* and *T. mentagrophytes*, which are respectively usually derived from infected cattle and rodents, show some differences in the type of lesion (Fig. 7.7 and Fig. CD7 • 1A–D).

Lesions are pruritic only in the early stages of infection; however, the horse can be irritable if lesions are ‘picked’ with a fingernail (when performing this test, due hygiene precautions must be taken for self-protection). This response persists even 5–10 days after treatment if the lesion is still infected and is a useful aid to diagnosis when the hair has been shed and scale and crust are still present on the lesion.

The healing lesions are usually markedly alopecic, smooth and silvery in colour. There may be secondary infection under the shedding scab with accumulation of purulent material.

**Differential diagnosis**

- *Microsporum* infection: this is a similar condition epidemiologically and clinically but tends to involve only a proportion of the hairs in an infected area; plucking of the hair is therefore more difficult and is sometimes resented.
- *Dermatophilosis (Dermatophilus congolensis)*: characteristic bacteria and epidemiology; tends to infect the back and lower limb lesions mostly and lesions are more purulent.

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**Figure 7.2** Ringworm. Removal of the hair in a mat at 10 days after natural infection with *Trichophyton equinum* var. *equinum*.

**Figure 7.3** Ringworm. The typical silvery, slightly scaly appearance of a lesion due to *Trichophyton equinum* var. *autotrophicum* infection. The hair loss in this case occurred naturally and infection was considered to have arisen some 14 days previously.

**Figure 7.4** (A) This 5-year-old Warmblood had a very subtle, slightly pruritic raised lesion with elevated hairs on his nose (arrow). (B) By 7 days later a more typical ringworm lesion had developed and several smaller, slightly later lesions were developing on the muzzle.
develop secondary trichophytosis due to impaired local immunity.

- Actinic dermatitis: restricted to white regions, particularly on the nose, face and distal limb regions.
- Mercurial poisoning: history of applications.

**Culicoides allergy/hypersensitivity** (sweet itch): severe localized pruritus mainly centred on the mane, tail and in some cases the ventral abdomen, associated with seasonal exposure to *Culicoides* spp. in particular.

- Insect bites: defined localized swellings associated with oedema and a central haemorrhagic spot.
- Mite or louse infestation: obvious parasites and more pruritus; localized to limbs and head and tail; no defined lesions usually present.
- Pemphigus foliaceus: this can be very similar clinically and even histologically and it may require special stains to identify the fungal elements.
- Sarcoïdosis: generalized exfoliative seborrhoeic disease with little similarity; some generalized forms of trichophytosis can be similar but they tend to self-cure and mycology and skin scrapings are diagnostic.
- Granulomatous enteritis syndrome: systemic involvement with prominent scaling.
- Alopecia areata: similar circular areas but with characteristic absence of any inflammatory responses and sterile cultures; characteristic histology.
- Anhidrosis: single cases affected with typical history of a move to a warm climate; affected cases may
Figure 7.8 Hair plucking being taken from one of many circular alopecic, scaling lesions on a pony mare. Note that gloves are worn when handling the case and that hairs are plucked using artery forceps from the margin of the lesion.

- Wound/exudate/lacrimal scalding: history and obvious clinical evidence; may be secondarily affected with trichophytosis due to skin damage.

**Diagnostic confirmation**

- Characteristic clinical signs and history of contact with infected horses (or other species).
- Hair plucking from the margins of fresh lesions (Fig. 7.8) can be examined microscopically (possibly after clearing with chlorolactophenol or 10% potassium hydroxide solution). Hyphae and relatively few large endothrix spores will be seen.
- Staining with lactophenol cotton blue can assist the recognition of the hyphae.
- Infected hairs do not fluoresce under ultraviolet light.
- Culture of hairs plucked from the margins of lesions on Sabouraud’s fungal medium. Medium with added phenol red provides an early indicator of dermatophytosis. Culture permits easy identification of the typical macroconidia.
- Skin biopsy is usually diagnostic of dermatophytosis but does not help to establish the species concerned (upon suspicion the pathologists should be informed so that special stains can be used to confirm the hyphae and spores).

**Note**

It is not easy to be certain of the species of fungus involved without culture. Cultures of hair plucking on commercially prepared Sabouraud’s agar at 25°C show characteristic colonies and change in colour of medium. The species can be confirmed from the colony and conidial spore characteristics.

For Trichophyton equinum vitamin enrichment is important and this can be achieved by the addition of two drops of injectable vitamin B complex to the medium.

**Treatment**

Most cases will resolve spontaneously after 6–12 weeks (particularly if the horses are in sunshine) (Pascoe 1973a).

Treatments do not shorten the course but may limit the spread of infection and limit the extent of environmental contamination. Subsequent immunity can last for an extended period but some cases can re-emerge following partial elimination from hair follicles.

Treatment is directed at the use of fungicidal treatment of the horse and sporicidal treatment of the environment. The infected areas should be clipped (taking care to disinfect the clippers at regular intervals and particularly thoroughly after each horse). All horses in contact should be considered for treatment at the same time and access to sunlight should be encouraged. The horse(s) may be washed with a fungicidal wash such as enilconazole or natamycin. Proprietary washes of these compounds are widely available. A 2% miconazole–2% chlorhexidine shampoo applied twice weekly has been shown to be effective in reducing the infectivity and so limiting an outbreak (Paterson 1997). Some tertiary amine surgical scrub solutions have a strong antifungal (but limited sporicidal) effect. Spot treatment of lesions (with the above solutions or miconazole) is probably not very useful in the horse in view of the rapid spread across the horse.

Individual lesions and the immediate surrounding hair may also be scrubbed for 1–2 minutes daily for 7–10 days with one of the following treatments:

- 10% povidone-iodine solution
- 2.5% lime sulphur in water
- 10% thiabendazole in water
- 2.5–10% tincture of iodine (painted on, not scrubbed)
- 0.3% Halamid
- tertiary amine disinfectant scrub solution.

Oral griseofulvin may be administered daily for 15–60 days (Hiddleston 1970) but the results of this alone are very variable. There are no reports of its efficacy and many specialists consider that it is of no material help. In any case, it probably does not reduce the infectivity of the spores and fungus-laden hairs. It should therefore not be used alone except perhaps in grazing horses. The drug is teratogenic and must not be used in pregnant mares.

**Control**

Prevention of spread between horses is important. All scabs and infected hairs should be carefully removed and burned.

Appropriately diluted washes of antifungal drugs such as natamycin, potassium monopersulphate and enilconazole are particularly useful as a spray (or fumigant) for the environment and infected equipment – most have strong sporicidal effects and this will reduce the chances of reinfection or infection of unaffected horses.

A number of modern disinfectants, including in particular the halogenated tertiary amines and inorganic peroxycgen compounds, have potent antifungal effects and some are sporicidal. These should probably not be used on the horse unless appropriate instructions from the manufacturer are available.

The stable environment can be effectively disinfected by ‘fogging’ with potassium monopersulphate (using an industrial or horticultural fogging machine) or enilconazole distributed in the same fashion (Desplenter 1989).

Contaminated tack and other equipment may be washed in suitable fungicidal disinfectants; modern halogenated...
peroxy-oxide compounds have a strong sporicidal and anti-
fungal effect, but these can be unreliable. Preferably, all tack
and equipment should be fumigated with formaldehyde
gas (see p. 86).

Vaccination is available in some European countries to
some species of dermatophytes including \( T. \) verrucosum.
The vaccines rely upon common antigens in the various
species of dermatophyte but, because natural immunity is
short-lived, the vaccine is unlikely to induce a better immu-
nity. Repeated vaccinations are therefore required. In spite
of some reports of severe local reactions (including swel-
ling, pain and abscessation), its efficacy is suggested as
being good. There are few studies to support its use but
ings developing in a contaminated environment and limit
the severity of the diseases. Its major value probably lies in
stables where repeated infections have occurred and where
contamination is widespread and uncontrollable.

Microsporosis

Profile

Ringworm due to Microsporum gypseum, \( M. \) equinum or \( M.
\) canis (microsporosis) is less common than trichophytosis
(see above). This disease is also highly contagious, being
spread by direct and indirect contact with infected horses
or through contaminated equipment or environment. It can
also be spread by biting insects and skin abrasion (Pascoe &
Connole 1974).

The organism can frequently be isolated from the soil
or bedding over 6–12 weeks after infected horses have had
access to it. The spores are probably very resistant to envi-
ronmental conditions and may survive for years.

The pathogenesis is probably indistinguishable from tri-
chophytosis (see p. 168).

Clinical signs

Small alopecic areas, most commonly on the face and legs,
develop but lesions may also follow the distribution of
insect bites elsewhere (Fig. 7.9). The lesions may be mildly
exudative and many have an oedematous (urticaria-like)
plaque within the affected skin.

Not all the hairs in a particular area will be equally
affected and so, when a lesion is plucked, not all the hairs
are shed. Plucking of the lesion is therefore more difficult
and is often resented by the horse (Fig. 7.10).

This effect is fairly characteristic of microsporosis and is
quite different from trichophytosis.

Some lesions are very inflamed and more pruritic than
others (Fig. 7.11 and Fig. CD7 - 2). Where pruritus devel-
ops it is likely that some hypersensitivity is involved and
of course this will result in localized spread of the infection
and environmental contamination.

Lesions may be exudative and may even be overtly
purulent as a result of secondary bacterial folliculitis. A few
cases initially present with an urticaria-like wheal that has
some exudation and crusting. Lesions are not pruritic but
are positive to a scratch test, i.e. the horse will respond to
gentle scratching of the area (when performing this test,
due hygiene precautions must be taken for self-protection).

In donkeys infected with Microsporum gypseum, there
may be significantly more hair loss and a much more
aggressive verrucose nature (Fig. CD7 - 3).

Note

It is always worth asking about possible infections amongst
human contacts – the presence of typical lesions in human
contacts (see Fig. CD7 - 1C-E) may be incidental of course
but it could be helpful also.
Part II Disease profiles • Section A Infectious disorders

Insect bite hypersensitivity (Culicoides spp. hypersensitivity/sweet itch): characteristic moderate–severe seasonal pruritus that is worse when outside than inside; this can be complicated by secondary dermatophytosis due to the rubbing on poles and walls, etc.

Mange mites: localized pruritic disorder with scale and crust in some cases; identifiable mites found in skin brushings.

Lice infestation, biting or sucking species: identifiable parasites and mild–moderate pruritus with a moth-eaten hair coat.

Onchocercal dermatitis: facial and ventral midline alopecic grey scaly areas; negative on fungal cultures.

Occult sarcoid: can be very difficult clinically but protracted static course and other forms of the condition on the same animal are helpful to diagnosis.

Pemphigus foliaceus: this can be very similar clinically and even histologically and it may require special stains to identify the fungal elements.

Chemical irritation: history of exposure.

Alopecia areata: benign static alopecic disorder with variable areas involved; very characteristic histology.

Diagnostic confirmation

Clinical appearance of lesions in patterns according to the transmission method.

M. equinum and some M. canis isolates may fluoresce under ultraviolet light (Wood’s lamp). M. gypseum does not fluoresce.

Hair samples should be examined after clearing with warm chlorolactophenol or 10% potassium hydroxide. Single or chains of ectothrix spores and hyphae may be seen.
Culture of hair plucking on a commercially prepared indicator (Sabouraud’s agar) shows characteristic colonies and conidial spores with characteristic colour change.

**Treatment**

Treatment is as for trichophytosis (above) but the response to both systemic (oral) griseofulvin and topical antifungal/fungicidal washes is significantly slower and more variable. However, again the disease will usually resolve spontaneously in 4–12 weeks.

**Control**

As for trichophytosis (above).

Removal of affected horses from contaminated yards after treatment is probably advisable, but the organism appears to have less resistance to environmental factors and so, by contrast to trichophytosis, may not appear annually.

Disinfection of the stable and all equipment and tack is important and again formaldehyde gas is probably the most reliable method. Clippers used for several horses are a common source of infection – skin abrasions are almost inevitable. Therefore fungicidal disinfectant trays should be used repeatedly during clipping.

**Malassezia dermatitis**

**Profile**

This is probably the most superficial mycosis in horses and is caused by *Malassezia pachydermatis*. The organism has recently gained increased importance in equine dermatology (Nel & Bond 2002). The organism is probably found as a natural commensal of the equine skin and gains significance when there is local or systemic immunocompromise. It may become significant in persistent damp or exudative skin and so is most often identified on the foot and distal limbs of horses with pastern dermatitis (see p. 471).

**Clinical signs**

The most significant syndrome is perineal and ventral abdominal pruritus due to intermammary debris (White 2005). *Malassezia* can usually be found here but it may not be the primary cause of the problem (Fig. 7.12). Secondary *Malassezia* spp. may also be identifiable in the exudate from some cases of pastern dermatitis.

**Differential diagnosis**

- Perineal pruritus due to *Oxyurus equi* infestation: eggs are easily identified with adhesive tape tests on the skin around the anus.
- Perineal or hind quarter/tail head pruritus due to insect bite hypersensitivity (*sweet itch*/*Culicoides* hypersensitivity): characteristic seasonality and epidemiology.
- Pastern dermatitis: *Malassezia* spp. infection is invariably secondary (see p. 471).

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**Key points: Malassezia dermatitis**

1. *Malassezia pachydermatis* is a natural commensal of equine skin occurring at low levels in normal skin. Local or systemic immunocompromise can predispose to overgrowth of the organism.
2. Mares have the only apparent primary Malassezia dermatitis in a syndrome in which the intermammary skin debris becomes infected with the organism. Perineal pruritus is the main sign. Otherwise the signs are usually related to a primary skin disease or damage and pastern dermatitis seems the commonest secondary syndrome associated with detectable forms of *Malassezia*.
3. Diagnosis is simply made on the identification of the organism in smears and cultures.
4. Treatment is simple with miconazole washes. Restoration of skin health is important.
5. The prognosis is excellent but recurrences can occur unless the underlying disorder is managed correctly.
Diagnostic confirmation
Direct smears and culture are required for confirmation.

Treatment
The infection is probably benign.

The organism probably has little primary significance; the only primary condition is intermammary syndrome of pruritus and tail rubbing, which is easily treated by removal and miconazole/chlorhexidine washes.

Cases of pastern dermatitis in which the organism is identified should be washed once with a miconazole wash.

Alternariasis

Profile
Environmental fungal organisms do occasionally infect the skin of horses. Alternaria spp. dermatitis (alternariasis) occurs in more temperate countries and is an occasional pathogen infecting mildly damaged epidermis and dermis. The organism is a commensal on the skin and is more often found in spoil and stable environments.

Infection may be gained via open skin abrasions or biting insects may be involved.

A more severe form is possible if the animal is immunocompromised.

Clinical signs
The nodules are usually very slow growing and are neither pruritic nor painful. The ears and breast are more often involved but they can occur at any site (Fig. 7.13). More extensive nodular and dermal serpiginate thickening can occur in sites where skin trauma from pruritus has occurred (Fig. 7.14).

Differential diagnosis

1. Cutaneous eosinophilic necrogranuloma with collagen degeneration (collagenolytic granuloma); usually develop along the back, trunk and neck, often multiple and usually very hard, small and commonly have a central calcified core under a small crust; usually very benign.

2. Zygomyces/Basidiobolus infection (Basidiobolus haptosporus); geographically restricted to warm humid climates; ulcerative, granulomatous skin disease with interlinking cutaneous and subcutaneous cording nodules.

3. Sporotrichosis (Sporothrix schenckii): multinodular, ulcerating disorder with characteristic geographic distribution and histopathology.

4. Nodular sarcoid: invariably other types of sarcoid lesions are present and the distribution of lesions is

Figure 7.13 These small nodules developed slowly over some months in this Shetland pony’s ears. The non-pruritic, non-painful nodules were biopsied and special stains and cultures from the central parts of the nodules confirmed Alternaria spp.

Figure 7.14 This horse developed a localized pruritus over the hind quarter following a pelvic fracture. This was assumed to be a result of neuritis but biopsies of the nodules revealed Alternaria spp. fungal elements in granulomatous nodules. They were surgically removed following partial response to oral potassium iodide.
significantly different; much longer and more severe course in most cases.

- Melanoma: characteristic black nodules mostly encountered in grey horses in the perineum, preputial skin, eyelids and lip; biopsy is diagnostic.

Diagnositic confirmation
Biopsy and culture are required for confirmation.

Treatment
Where possible surgical removal is curative but recurrence can occur locally if any infection is left. Extensive areas are much more problematic. Cryosurgical necrosis and thermocautery can be used. Oral potassium iodide (5–10 g twice daily by mouth) for 3–6 weeks may improve the condition. Antibiotics are ineffective and systemic antifungal drugs are prohibitively expensive and only marginally effective.

The condition is very benign and so once the diagnosis is established a decision to leave the lesions alone can be justified.

Phaeohyphomycosis

Profile
This is a chronic subcutaneous and systemic fungal disease with small, multiple subcutaneous nodules, caused in tropical countries by Drechslera spicifera (Kaplan 1975). It differs from mycetoma and alternaritasis (above) in that the hyphae remain discrete and do not aggregate into nodules. The fungus gains access to the skin via wounds. In temperate climates a similar condition (which may be indistinguishable from phaeohyphomycosis) can occur (most often on the head and ears) from deep and chronic infection with a variety of less pathogenic fungi including Alternaria spp. (see p. 176).

Clinical signs
Small, black or darkly coloured, denuded plaques and nodules primarily containing papules and pustules or multiple fibrotic subcutaneous nodules present on the sides of the neck, body (Fig. 7.15) and limbs. The lesions can, however, occur at any site.

Differential diagnosis
- Eosinophilic dermal necrogranuloma with collagen degeneration (collagenolytic granuloma): similarly benign nodular disease with almost no pathognomonic signs but with characteristic biopsy features.
- Molluscum contagiosum: geographically restricted and can look very similar clinically; biopsy is diagnostic.
- Mixed and nodular sarcoïd: biopsy is diagnostic and usually other sarcoïd types are present.
- Cutaneous lymphosarcoma: larger, more extensive nodules and cording of cutaneous lymphatics; occasional ulceration.
- Melanoma: characteristic black colour and definitive biopsy and fine needle aspirate; usually multiple and most often in the perineum of grey horses.
- Insect bite reactions: transient nodules with urticaria-like reactions; some cases can be more persistent.

Diagnostic confirmation
- Biopsy is essential (fungal elements may be identified directly on sections).
- Culture and identification of Drechslera spicifera from the deepest regions of the biopsy specimen on Sabouraud’s dextrose agar (without antibiotic additions). Initially the colony grows rapidly and is white-grey but it soon changes to a brown-black colour.

Treatment
Treatment is likely to be prolonged and even in mild cases only marginally effective. Systemic iodide therapy (sodium iodide at 40 mg/kg as a 20% solution by slow intravenous

Key points: Phaeohyphomycosis

1 Rare nodular skin disease usually associated with Drechslera spicifera and other ‘black’ moulds occurring in tropical countries in particular.
2 Signs restricted to dark, non-painful, multifocal, firm–solid, cutaneous nodules with overlying hair loss. Some are pustular and can ulcerate. Most lesions occur on the face.
3 Histological examination of excised lesions and stained smears from aspirates may reveal fungal hyphae and a granulomatous inflammation.
4 Treatment is limited to surgical removal of the nodules but iodosides and topical antifungal disinfectants and conazoles may be helpful.
5 Prognosis is fair with some cases resolving spontaneously.

Figure 7.15 Phaeohyphomycosis. Multiple fibrotic subcutaneous nodules on the sides of the body. Drechslera spicifera was isolated from an excised lesion.
injection once daily for 3–5 days then orally until cured or 5–10 g twice daily of potassium iodide by mouth until signs of iodism appear or a cure is achieved) may be partially effective. Amphotericin B or fluconazole therapy may also be useful but there are no studies on the efficacy. Topical application of etisazole in dimethyl sulphoxide (DMSO) can be effective (Evans 1990).

**Mycetoma**

**Profile**

True eumycotic mycetomas are caused by fungal contamination of wounds by free-living (soil and plant) fungi. The fungi cause chronic subcutaneous infections characterized by tumour-like lesions with extensive sinus tracts and fistulas. They commonly have granular components (so-called ‘kunkers’). The most common fungi are *Curvularia geniculata* and *Pseudoallescheria boydii*. The former produces a very dark lesion known as a black-grained mycetoma (Boomker 1977, Miller 1980), while the latter produces a white-grained mycetoma. *Alternaria* spp. are also liable to produce small granulomas at the sites of contaminated skin injuries which could be termed mycetoma (see p. 176).

Actinomycotic mycetomas are due to such bacteria as *Actinobacillus* spp., *Nocardia* spp. and *Actinomyces* spp. and so are not strictly mycetomas.

**Clinical signs**

Ulcerating nodules occur on limbs, head or ventral abdomen with cording of lymphatic vessels. Lesions show a chronic seropurulent discharge from granulating ulcers. The condition is accompanied by mild pruritus. Some asymptomatic nodules, similar in appearance to papilloma but black or dark in colour, covered by ulcerated hairless skin may be present (McEntee 1987). Biopsy of the excised lesion readily identifies the unusual but characteristic colour and consistency of the lesions (Fig. 7.16).

**Differential diagnosis**

- Pythiosis (*Pythium* spp.): geographically restricted severely invasive fungal infection of wounds.
- *Basidiobolus* infection (*Basidiobolus haptosporus*): nodules are very similar.
- Sporotrichosis (*Sporothrix schenckii*): rare, geographically restricted nodular skin disease with lymphatic tracking.
- Glanders/farcy (*Burkholderia* (*Pseudomonas*) *mallei*): severe geographically restricted ulcerative nodular disease affecting the respiratory tract and skin in areas of skin trauma.
- Nodular sarcoid: slowly expanding lesions with nodules more common on the body and several different types present on the same horse; biopsy is characteristic.
- Melanoma: characteristic black nodules sometimes with an ulcerated surface predominately affecting grey horses.

**Diagnostic confirmation**

- Restricted geographical area.
- Physical examination is suggestive.
- Biopsy with special staining methods is required for confirmation.
- Isolation and culture of *Curvularia geniculata* or *Pseudoallescheria boydii* from deep within nodules or from biopsy.

**Treatment**

Some cases can be resolved by aggressive surgical removal of affected area, but recurrence is common. Oral potassium iodide (5–10 g twice daily by mouth) for 3–6 weeks may improve the condition.

Antibiotics are largely ineffective and systemic antifungal drugs are prohibitively expensive and only marginally effective.

**Sporotrichosis**

**Profile**

This is a chronic, progressive, sporadic infection of skin and subcutaneous/lymphatic tissue caused by *Sporothrix*...
S. schenckii. The organism is a saprophytic free-living dimorphic fungus. Infection is usually introduced through small skin wounds (Evans 1990). The primary site is usually associated with a small wound on the distal limb region.

The disease is a zoonosis so care should be taken when handling suspected or proven cases.

Clinical signs
The primary lesion is a firm, nodular swelling at the site of a previous (usually) small wound, often at or below the fetlock. Over some weeks or months, corded lymphatics and painless, non-pruritic subcutaneous nodules (1–5 cm diameter) develop running proximally. Lesions can develop on the upper limb regions and body trunk.

Ulceration of surface of nodules sometimes occurs with creamy, white-grey, purulent discharge and surface encrustation and scabbing (Fig. 7.17). The cardinal signs are gradual extension from a single nodule or small cluster of nodules.

Repeated episodes over some months result in lymphatic cording (lymphangitis) in some cases only – medication with corticosteroids may result in recurrences and exacerbation of the condition. A few cases develop isolated nodules and so when nodules are investigated this condition should be considered.

Remarkably, the regional lymph nodes are seldom involved.

Differential diagnosis
- Ulcerative (bacterial) lymphangitis (Corynebacterium paratuberculosis).
- Epizootic lymphangitis (Histoplasma farciminosum): geographically restricted, debilitating, cording and ulcerating nodular disease affecting the skin of horses, donkeys and mules; organism can be identified in purulent discharges.
- Glanders (Burkholderia (Pseudomonas) mallei): geographically restricted, severely debilitating respiratory and cutaneous fungal nodular disease with ulceration and discharge of a honey-coloured seropurulent discharge.
- Mycetoma: isolated but often complex and slowly expanding cutaneous/subcutaneous nodules that seldom have lymphatic cording.

Diagnostic confirmation
- Characteristic cording and nodules on lymphatic vessels.
- Gram stain of exudate from impression smear identifies the causative organism.
- Culture on Sabouraud's agar.
- Biopsy is not usually diagnostic.

Treatment
Iodine therapy is the only available and useful medical approach.
Systemic iodide therapy (sodium iodide at 40 mg/kg as a 20% solution by slow intravenous injection once daily for 3–5 days then oral 10 g daily of potassium iodide until cured or 10 g of potassium iodide daily by mouth until signs of iodism appear.
Organic iodides such as ethylene diamine dihydroiodide administered orally can be useful but prolonged courses are essential (White 2005).

Histoplasmosis (epizootic lymphangitis)
Profile
Epizootic lymphangitis is an important, chronic, highly contagious disease of Equidae caused by Histoplasma capsulatum var. farciminosum. It is also known as ‘African farcy’ or pseudoglanders. It is currently restricted to some localized...
Key points: Histoplasmosis

1. A serious debilitating cutaneous fungal infection of horses, donkeys and mules in limited areas of North Africa, Asia and the Far East caused by *Histoplasma capsulatum* var. *farciminosum*. Altitude is a significant factor with cases encountered within a narrow altitude band.

2. Early cases show localized dermal or subcutaneous nodules which track along lymphatics and ulcerate. Severe forms show coalescing masses of ulcerated nodules with a creamy yellow purulent discharge. Ocular forms are also encountered.

3. Diagnosis is relatively easy in endemic areas but the signs can closely resemble glanders. Culture and smear characteristics from the pus are diagnostic.

4. Treatment is tedious and involves the individual incision and iodine flushing of each infected nodule. Oral and systemic iodides, flucytosine and topical fungicidal disinfectants can be effective for some cases. Severe cases are probably not treatable.

5. Vaccines are not available at present. Prognosis is good if treatment can be instigated in the earliest possible stages but hopeless in advanced cases.

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**Figure 7.18** A severely affected, debilitated horse with epizootic lymphangitis. Note the most severely affected regions are the limbs, lower trunk and head.

**Figure 7.19** An early case of epizootic lymphangitis. The nodules are prominent but have not yet burst out. Note that this mule in Ethiopia had a wound over the withers region also that was treated inappropriately.

The disease is characterized by suppurative lymphangitis primarily affecting the hind legs and neck, lips and other areas where harness abrasions occur such as the face, girth and hind limbs. Gross enlargement and inflammation of cutaneous (and other) lymphatic vessels with lymphadenitis is typical. Skin ulceration along the corded lymphatics occurs with a ‘mouldy’ odour from lesions. Often the lesions remain fairly localized in the earlier cases so that one region of tack contact becomes badly affected but the rest of the skin is unaffected (Fig. CD7 • 4).

The severe forms of the disease are more generalized and the extent of skin involvement can be extreme with all areas of the skin being involved to variable degrees. Even in this form the lesions themselves are seldom painful on palpation (Fig. CD7 • 5).

Most cases have no systemic signs, but the disease can affect the nasal cavity, lungs, pleural cavity, eyes and joints, which makes it clinically very similar to glanders. However, generally the absence of significant systemic signs and the definitive smear morphology of *Histoplasma farciminosum* readily differentiate this from glanders.
Ophthalmic/Ocular form  Ocular histoplasmosis affects the conjunctiva and skin of the periorbital region; it is commoner in donkeys than horses (Fig. 7.21). The condition in donkeys is somewhat different in that the lesions are more proliferative and tend to cause obstruction of the nasolacrimal duct (Fig. CD7•6).

**Differential diagnosis**
- Glanders: this is a very similar condition and can easily be mistaken for epizootic lymphangitis. There is more pulmonary involvement and serology and the ‘mallein’ (intradermopalpebral) test can be used to differentiate the two conditions.
- Ulcerative lymphangitis: sporadic localized ulcerative condition that has characteristic culture and smear identification of C. pseudotuberculosis.
- Sporotrichosis: sporadic geographically restricted disease with ulcerating nodules; can be similar to early cases of epizootic lymphangitis.
- Malevolent sarcoid: single cases with different implications and usually several different forms present on the horse.
- Cutaneous lymphosarcoma: sporadic, rare and individual condition without geographical restrictions; characteristic histology.

**Diagnostic confirmation**
- Direct impression smears from discharging lesions reveal the tissue form of the disease (Fig. CD7•7).
- Biopsy and culture from biopsy on Sabouraud’s agar can demonstrate the cultural forms of the fungus. However, growth is very slow; over 2–3 weeks is required.
- Serological testing is not yet established.

**Treatment**
Ideally the disease should be eradicated by an aggressive slaughter policy but, in most endemic areas, that is impractical. In endemic areas treatment is an important consideration.
Provided that treatment can be undertaken, early cases can be treated effectively but it does require considerable dedication and persistence. In theory at least the organism should be susceptible to amphotericin B, nystatin and possibly fluconazole. None of these are usually available in the areas where the disease occurs and in any case treatment would be prohibitively expensive. Other treatment options are limited by the access to the patients over several months or more.

In the early stages the nodules should be individually incised and flushed aggressively with strong (7.5%) iodine solution (Fig. 7.22). Repeated flushing with the iodine solution is required for up to six weekly treatments (and sometimes more). Persistence is the main factor involved in successful treatment — a single treatment is unlikely to help a great deal. Once the fungus is under control, granulation of the lesions occurs (Fig. 7.23) and the nodules resolve as small focal scars.

Some horses recover spontaneously and are then solidly immune. Animals that are treated effectively are probably also immune.

Control

Surgical drainage of the purulent material and naturally discharging nodules are laden with infective material. The organism can survive for many months in soil and in vegetable matter. Disinfection of the environment following treatment is essential — halogenated peroxygen and quaternary ammonia compounds, or other fungicidal disinfectants can be used.

In all cases fly control is a major aspect of control of spread but in endemic areas this is virtually impossible. Vaccination has been attempted but so far without success. Cases occurring outside endemic regions are usually slaughtered to prevent spread. Treatment is not usually attempted with slaughter policies in force to control the spread of the condition.

Cryptococcosis (European blastomycosis/torulosis)

Profile

This is a very rare, chronic subcutaneous and systemic fungal disease caused by an encapsulated yeast, *Cryptococcus neoformans*, with several different variants that are geographically restricted. *Cryptococcus* spp. organisms are free-living, saprophytic yeast-like fungi that have a close connection to wild bird faeces and the associated detritus of bird habitats.

The fungus gains access to the skin via wounds and possibly via the inhalation route. Immunocompromise is a major risk factor in all affected species including man.
The virulence of the organism relates to the specific polysaccharide capsule that prevents phagocytosis. Non-capsulated strains have much less virulence. Animals with a normal immune system seem unlikely to be affected. Some forms of immunocompromise may be involved in some cases although concurrent disease may not be obvious. In common with many other of the opportunistic deep fungal infections, concurrent corticosteroid therapy is known to exacerbate the infection. Some cases can lie dormant in the skin until some form of immunocompromise develops.

Clinical signs
Non-specific firm skin nodules, particularly in the lips and pinnae, that expand very slowly are typical. Ultimately these can ulcerate and produce a thick creamy pus. The surrounding tissue may be minimally inflamed (Fig. 7.24 and Fig. CD 7-8).

Differential diagnosis
- Eosinophilic dermal necrogranuloma with collagen degeneration (collagenolytic granuloma): benign nodular disease easily recognized and very common; seldom occurs on the ear flaps and mouth region; typical histology.
- Molluscum contagiosum: geographically restricted and usually multiple with no tendency to ulcerate; biopsy is diagnostic;
- Mixed and nodular sarcoid: biopsy is diagnostic and usually other lesions of sarcoid types are present.
- Cutaneous lymphosarcoma: larger, more extensive nodules and cording of cutaneous lymphatics; occasional ulceration.
- Melanoma: characteristic black colour and definitive biopsy and fine needle aspirate; usually multiple and most often in the perineum of grey horses.
- Insect bite reactions: transient nodules with urticaria-like reactions; some cases are more persistent.

Diagnostic confirmation
- Biopsy is essential (fungal elements may be identified directly on sections) (see Fig. CD 7-8).

Treatment
There are too few reported series on which to base any treatment advice (Chandra 1992). However, in other species fluconazole (and some other conazole medications) or amphotericin B therapy may also be useful but there are no studies on the efficacy, and dose rates are uncertain. Surgical removal of isolated nodules can be effective.

Note
Nodules can remain dormant or expand very slowly unless and until an immunocompromise develops or corticosteroids are administered.

Fungal granuloma/pythiosis (phycomycosis/basidiobolomycosis/bursatti)

Profile
This is a chronic, subcutaneous, fungal, ulcerative, granulomatous skin disease caused by Pythium insidiosum occurring in subtropical and tropical areas and affecting horses of all types and ages and both sexes. The disease is also called bursatti, Florida horse leeches and swamp cancer. The fungi are aquatic and so wetting and contact with infected plant material in water is essential. The fungi are actively drawn to sites of tissue damage (skin wounds are probably a prerequisite for infection) and skin wetting/maceration. Most cases therefore occur during warm wet seasons. Horses kept in persistently warm wet conditions such as swamps and low-lying water courses are more often affected. Most cases have been reported in Australia, the Gulf states of the USA including particularly Florida, and the Indian subcontinent. Lesions most commonly occur on the limbs, abdomen, neck, lips and nasal margins and cases rarely show systemic involvement even when the extent of damage is severe.
Basidiobolus haptosporus causes a similar disease to pythiosis but is confined to head and abdomen; no leg lesions have been recorded.

Clinical signs

Pythiosis Ulceration of skin or wound with pruritus manifest as biting and kicking at affected area(s). Dense granulation tissue containing masses of yellow-grey gritty masses (‘kunkers’). The fibrinous exudate hangs from the wound (giving it the name ‘Florida leeches’).

Diagnosis is by biopsy, smear recognition and culture of the organisms responsible. Recent ELISA and PCR methods have improved diagnosis significantly.

Treatment is limited to wide surgical excision combined with immunotherapy. Lodges are not as effective as for other fungal skin infections.

Vaccination holds some promise of effective prevention.

Differential diagnosis

- Sarcoid (particularly if recurrent interference): localized tumour condition with characteristic histology.
- Habronema musca infestation in wounds: this can be very similar but tends to be less destructive and is far easier to treat. The larva can be identified in washings from the lesion.
- Neoplasia (including cutaneous lymphosarcoma): sporadic, non-infective and usually slow to expand. Systemic signs are common and histology is diagnostic.
- Mycetoma: probably similar but much less aggressive.
- Botryomycosis (staphylococcal pyogranuloma): biopsy and culture of S. aureus (usually) are diagnostic.

Exuberant granulation tissue (from any cause) or indolent or non-granulating: can be difficult to differentiate if the granulation tissue is infected.
Diagnostic confirmation
• Pythiosis usually has a history of access of horses to water-logged pasture or lagoon creeks; basidiobolomycosis occurs more in dry conditions with the organism probably living in the soil.
• Clinical appearance of characteristic mucopurulent strings and ‘kunkers’ in exudate or in cut surface of lesion.
• Biopsy (collected into 10% formol saline) shows increased collagen and intense eosinophilic infiltration.
• Fungal culture on Sabouraud’s agar (using a fresh sample sent on ice) from early lesions identifies organisms responsible.
• ELISA and PCR methods hold promise of an improved diagnosis.

Treatment
Early treatment is essential as the prognosis is much poorer in chronic cases.

Surgical excision of the tumour under general anaesthesia is the most common and most successful treatment, particularly in chronic cases (Bridges & Emmons 1961, Restrepo et al 1973, Pascoe 1989, Moriello et al 1998). Complete removal of all affected tissue is essential if recurrences are to be avoided. Surgery should be performed again as soon as any new lesions develop (usually manifest as 1–5 mm diameter dark red or black spots). Surgery requires extreme caution on the limbs in particular and iodine-soaked pressure bandages should be applied after excision is complete. Repeat surgery is commonly required when the lesions occur around tendons and joints.

Intravenous sodium iodide (Pascoe 1973b) is a useful adjunct to surgery. Amphotericin B may be administered intravenously once daily and topically (McMullan et al 1977). For intravenous administration, amphotericin B is available as a vial containing 50mg lyophilized amphotericin B. The required dose is dissolved in 1 litre of saline and slowly infused intravenously over 1 hour. Intravenous treatment is well tolerated at an average daily dose starting at 0.3 mg/kg rising to 0.6 mg/kg on day 3 and then every other day for the required time (10–80 days). For local treatment of the wound site or fresh lesions, 50 g amphotericin B in 10 mL sterile water + 10 mL dimethyl sulphoxide (DMSO) on a gauze dressing is applied daily for at least 1 week.

Significant benefit may be obtained by surgically removing the bulk of the fungal growth, followed by daily intravenous and topical administration of amphotericin B, with periodic extirpation of small necrotic tracts as necessary. Some strains of Pythium have shown resistance to amphotericin B (Eaton 1993).

Vaccination may provide therapeutic and prophylactic effects (Miller 1981, Newton & Ross 1993, Mendoza et al 2003) but vaccines are not manufactured or available commercially and need to be prepared by a suitably qualified laboratory.

Prognosis
Successful treatment depends on the age and physical condition of the horse, previous treatment, age, size/site of the lesion and whether there is bony involvement. Young fresh lesions of 2 weeks’ duration respond well to immunotherapy alone. Older lesions respond poorly and all reported cases with bone involvement have died (Alfaro & Mendoza 1990).

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