



Research article

Fungal diseases of horses

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ABSTRACT

Among diseases of horses caused by fungi (=mycoses), dermatophytosis, cryptococcosis and aspergillosis are of particular concern, due their worldwide diffusion and, for some of them, zoonotic potential. Conversely, other mycoses such as subcutaneous (i.e., pythiosis and mycetoma) or deep mycoses (i.e., blastomycosis and coccidioidomycosis) are rare, and/or limited to restricted geographical areas. Generally, subcutaneous and deep mycoses are chronic and progressive diseases; clinical signs include extensive, painful lesions (not pathognomonic), which resemble to other microbial infections. In all cases, early diagnosis is crucial in order to achieve a favorable prognosis. Knowledge of the epidemiology, clinical signs, and diagnosis of fungal diseases is essential for the establishment of effective therapeutic strategies. This article reviews the clinical manifestations, diagnosis and therapeutic protocols of equine fungal infections as a support to early diagnosis and application of targeted therapeutic and control strategies.

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1. Introduction

Over the last two decades, the number of fungal and fungal-like diseases of plant and animals in both natural and controlled systems has increased, most likely as a consequence of environmental changes (Fisher et al., 2012). Similarly to other mammals, horses may be affected by several fungal diseases, although only some (i.e., dermatophytosis, pythiosis and epizootic lymphangitis, and aspergillosis) are well described (Al-Ani, 1999; Chermette et al., 2008; Gaastra et al., 2010; Cafarchia et al., 2012b). Most reports of fungal diseases are available as sporadic clinical cases, and comprehensive reviews on the topic date back to the 80s (Blackford, 1984). This article aims at summarizing the clinical manifestations, diagnosis and therapies of equine fungal infections, and at supporting clinicians and their efforts to diagnose the infections and set out targeted therapeutic and control strategies.

In the present review, equine fungal diseases are classified into: (i) superficial mycoses, caused by

pathogens confined to the *stratum corneum* except hairs; (ii) cutaneous mycoses, by pathogens invading keratinized tissues (including hairs, horns and skin); (iii) subcutaneous mycoses and; (iv) deep mycoses which affect the upper and/or lower respiratory tracts, as well as internal organs (de Hoog et al., 2000).

2. Superficial mycoses

Superficial mycoses are caused by facultative or commensal pathogens, which are responsible for mild inflammatory, usually benign infections, generally associated with underlying immunodepressive conditions in the mammalian host. Yeasts belonging to the genus *Malassezia* are the most frequent agents of superficial mycosis in horses (Nell et al., 2002; White et al., 2006; Kim et al., 2011).

2.1. *Malassezia* infections

Malassezia yeasts have recently been under the spotlight in equine dermatology since they cause dermatitis in immunocompromised individuals (Nell et al., 2002; White

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et al., 2006; Kim et al., 2011). These yeasts can be isolated from the axilla, interbulbar region, groin and anus of healthy horses (~60%) (Nell et al., 2002), the intermammary region of mares and the preputial fossa in geldings (White et al., 2006). Out of the 14 species ranked within the genus, only *M. furfur*, *M. slooffiae*, *M. obtusa*, *M. globosa*, *M. restricta*, *M. equina* and *M. pachydermatis* have been isolated from horses (Cabañes et al., 2007), in which they cause benign infections of limited or no significance. However, perineal and ventral abdominal exudates associated with pruritus, and alopecic areas without inflammation, exudates or crusts have been described (White et al., 2006). The diagnosis usually requires cytological and cultural examinations and/or histology. For cytology, samples are usually collected by sticking tape strips on the skin of the infected animal; subsequently, the material collected is stained with May–Grunwald Giemsa (Nell et al., 2002; Kim et al., 2011). Samples are considered positive if large populations of *Malassezia* cells are counted at 40× magnification (cf. Cafarchia et al., 2005). Histological examinations of the skin of *Malassezia*-infected horses have previously led to the observation of superficial hyperplastic dermatitis with a predominance of lymphocytes and macrophages (Kim et al., 2011). Because of the lipophilic properties of these yeasts, fungal media supplemented with a range of lipid sources are suitable for their culture (e.g., Dixon's medium). Treatment protocols of the clinical disease are reported in Table 1.

3. Cutaneous infections

Cutaneous mycoses include fungal infections of keratinized tissues including hairs, horns and skin, which cause significant destruction of the keratinized tissues and induce variable (protective) immunological responses. Skin infections might also result from widely disseminated fungal infections; in these cases, a prompt observation of the clinical signs, followed by an appropriate diagnosis, is crucial for positive treatment outcomes.

3.1. *Chromoblastomycoses*

Chromoblastomycosis (Syn: *Chromomycosis*, *Cladosporiosis*, *Fonseca's disease*, *Pedroso's disease*, *Phaeosporotrichosis*, *Verrucous dermatitis*) is a slow-developing chronic granulomatous fungal infection, which results in the formation of pigmented hard yeast cells, known as "muriform cells" or "sclerotic bodies". Clinically, the disease consists in the development of verrucose, dyschromic, scaly plaques, as well as atrophic patches and ulcerative lesions of the skin (Abid et al., 1987; López and Méndez, 2007). This disease occurs sporadically in horses; only a few cases, caused by *Fonsecaea* spp., have been reported in animals from the United States and Canada, localized to areas characterized by heavy rainfalls (Abid et al., 1987; López and Méndez, 2007). The infection is acquired following the accidental inoculation of the etiologic agent into the skin or the subcutaneous tissue. Following penetration of the tissue, the fungus transforms from filamentous to parasitic stages, known as muriform

bodies, which are not destroyable by macrophages and polymorphonuclear phagocytic cells (López and Méndez, 2007).

At first, from one to two months after the infection, nodular granuloma-like lesions with no draining tracts appear (Table 1—Abid et al., 1987); these lesions are clinically indistinguishable from melanomas, foreign body granulomas, squamous cell carcinomas, habronemosis, onchocerciasis, mycetomas, phaeohyphomycosis and sporotrichosis (Abid et al., 1987). The laboratory diagnosis requires a direct cytological examination of the affected skin sample using potassium hydroxide (KOH) and/or histopathological examination using Hematoxylin–Eosin (H&E) or Periodic acid–Schiff (PAS) or Gomori's methenamine silver (GMS) stains, followed by fungal culture of scrapings or biopsy material (Tables 1 and 2—Abid et al., 1987). Culture on Sabouraud dextrose agar (SAB) for 10 days produces velvet colonies, which are initially deep green in colour, and black later. Since spontaneous recovery from the disease is rare, cases of chromoblastomycoses require adequate therapies. In horses, surgery has been proposed as the only definitive solution (Abid et al., 1987; López and Méndez, 2007).

3.2. *Dermatophytoses*

Dermatophytoses are superficial, cutaneous mycoses caused by dermatophytes. These diseases are considered zoonoses, since they can be transmitted from animals to humans. Dermatophytes are filamentous fungi which invade keratinized tissues of humans and animals, causing mild to severe, localized and/or diffuse infections. Zoophilic dermatophytes infect both animals and humans, whereas anthropophilic ones are mainly found on humans. Geophilic dermatophytes can cause disease in both animals and humans. Fungi of the genera *Microsporum* and *Trichophyton* cause animal dermatophytoses; amongst these, *Microsporum canis* and species of the *Trichophyton mentagrophytes* complex are also pathogenic to humans. *Trichophyton equinum* and *M. canis* frequently cause 'ringworm' in horses, particularly in young animals (Chermette et al., 2008). Other species such as *T. mentagrophytes* or *M. gypseum* have also been isolated from skin lesions, while *T. bullosum* and *M. praecox* from healthy animals and the surrounding environment (De Vroey et al., 1983). The latter two species have also been implicated in human cases (Alanio et al., 2011; Sitterle et al., 2012).

Dermatophyte infection is acquired by direct contact with diseased animals or asymptomatic carriers and/or from the environment. Generally, clinical signs include mild to severe alopecia associated with erythema (Table 1—Chermette et al., 2008). Lesions due to *T. equinum* or *M. canis* are typically dry, with thin powdery scales and hairs broken at their base (Fig. 1—Chermette et al., 2008). Lesions are usually not pruriginous, and kerion and miliary dermatitis may also occur which extends rapidly from the saddle and the girth through the body (Chermette et al., 2008). Infections are clinically indistinguishable from those by *Dermatophilus congolensis* (Chermette et al., 2008).

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