Proceedings of the 12th International Congress of the World Equine Veterinary Association 
WEVA

November 2 - 5, 2011
Hyderabad, India

Reprinted in IVIS with the Permission of WEVA Organizers
Alopecic skin diseases without scaling:

**Telogen effluvium (TE)** is a syndrome of hair loss during the telogen or resting phase of the hair follicle. With TE, a stressful circumstance (e.g., fever, shock, severe illness, surgery, anesthesia) causes premature cessation of growth of many anagen hair follicles and synchronizes hair follicles in catagen (transition phase of the hair follicle) followed by telogen. Within 1-3 months after the insult, a large number of telogen hairs are shed as a new wave of hair follicle activity begins, leading to areas of both widespread and patchy alopecia.

**Anagen effluvium (AE)** is a syndrome of hair loss during the anagen or growth phase of the hair follicle. In contrast to TE, AE is generally caused by a severe infectious or metabolic disease or exposure to certain chemicals (e.g., antimitotic agents) that interferes with the anagen phase of the hair follicle and results in abnormalities of the hair follicle and hair shaft. Alopecia is usually abrupt and occurs within a few days of the insult.

The underlying skin appears normal in both diseases. If a skin biopsy is pursued, histologic examination reveals normal skin and hair follicle activity. The most useful diagnostic test is a trichogram with which shafts and roots of plucked hairs are examined under low and higher power microscopy: TE telogen hairs are characterized by a uniform shaft diameter that may taper at the distal end and a mildly clubbed, non-pigmented root end that lacks a root sheath; AE hairs are characterized by dysplastic changes: shafts may be focally narrowed and deformed leading to breaking when the hairs are plucked. In contrast to TE, hair roots in AE are thick and bulb-like. Both TE and AE are self-limiting disorders that resolve as new hair grows.

**Alopecia areata (AA)** is an immune-mediated disorder targeting the anagen hair follicle, leading to patches of well-circumscribed alopecia with normal underlying skin. In horses, antibodies directed against parts of the hair follicle have been identified. In addition, genetic, endocrine, and psychogenic (stress) factors are thought to play a role in human AA. Alopecia patterns vary from single to multiple, well-circumscribed areas of alopecia to generalized alopecia (alopecia totalis or universalis). With the latter, mane and tail hairs may also be lost. Pruritus is absent and affected horses are otherwise normal. Over time, the underlying skin often becomes hyperpigmented. Further support of an immunopathogenesis is detection of an accumulation of lymphoid cells around hair bulbs ("swarm of bees") with skin biopsies but these lesion are only present in the active phase of the disease. Later histopathological changes include prominent telogen hair follicles, peribulbar melanosis, follicular atrophy, and changes consistent with follicular dysplasia (dysplastic hair shafts, distorted hair follicle contours) with minimal evidence of inflammation. Collection of multiple biopsies from the leading edge of early lesions and serial sectioning of the biopsy specimen may be required to find the characteristic lesion. No curative treatment is available but lesions commonly undergo spontaneous remission over several months to a couple of years.

Alopecic areas should be protected to avoid sunburn by applying sunscreen daily or stabling the horse during daylight hours. In humans, glucocorticoids, cyclosporine, topical minoxidil and tacrolimus have resulted in varying degrees of hair regrowth. These treatments have not been reported to be effective in the horse.

**Alopecic diseases with scaling:**

**Dermatophytosis (Ringworm, Girth Itch)** is a common superficial skin infection especially prevalent in warm, humid climates where abundant biting insects act as vectors. In temperate climates the incidence increases in fall and winter months when horses are stabled with limited exposure to ultraviolet light. The disease is acquired from spores present in the environment or by transmission of spores on contaminated fomites (brushes, combs, clippers, blankets, bedding, tack,
fencing, transport vehicles, etc.). Hair shafts with arthrospores can remain infectious in the environment for many months to years. The incubation period varies from 1-6 weeks. Disruption of skin integrity appears to be crucial for invasion of actively growing hair follicles (anagen hair). Contagiousness varies from limited spread to explosive outbreaks, depending on previous exposure of resident horses to the particular type of dermatophyte. Clinical signs include focal to multifocal (rarely generalized) lesions with alopecia, scaling, and crusting although early lesions can appear urticaria-like (erected hairs). The classic lesion is a ring of alopecia with central healing and crusts/scales with a sharp margin at the periphery and easily epilated hair from the lesions. The most commonly affected areas are the face, neck, dorsolateral thorax, and girth but lesions can also be limited to the pasterns. Most horses are non-pruritic.

It is unusual for a healthy horse to develop dermatophytosis a second time unless a different dermatophyte species is involved. Horses suffering from severe, chronic, or recurrent dermatophytosis may have immunosuppressive disorders, are being treated with glucocorticoids, or inhabit moist, crowded environments. Dermatophyte infections in healthy horses are usually self-limiting over a 2-3 months period when hair shafts stop growing and enter telogen. When clinicians rely on clinical signs alone, dermatophytosis is commonly overdiagnosed. The diagnosis can be confirmed by collecting hair plucks from the edge of the lesions and finding hyphae and arthrospores (trichogram). The most reliable diagnostic test is fungal culture. After gentle cleansing of the area with soap and water or wiping with 70% alcohol, hairs should be plucked from the edge of several recently formed alopecic, crusty, and scaling lesions. Dermatophyte test medium (DTM) has frequent false-positives, making microscopic exam of macroconidia from culture essential. For these reasons the author prefers to send samples for dermatophyte culture directly to a reliable laboratory that can also identify the species of dermatophyte responsible for the outbreak. A skin biopsy can find spores and hyphae in follicular lumens, hair shafts, or crusts, but special stains (PAS) may be needed. Caution is necessary for T. mentagrophytes dermatophytosis because it can produce acantholytic keratinocytes with neutrophils in a surface crust, a lesion that may be indistinguishable from pemphigus foliaceus.

Dermatophilosis (Rain scald, Rain rot, Mud fever, dew poisoning) is another common disease of horses with a worldwide distribution. Dermatophilosis is caused by a gram-positive, facultative anaerobic branching actinomycete: *Dermatophilus congolensis*. Chronically affected, but asymptomatic carriers are thought to be the primary source for outbreaks and the organism is transmitted by flies and ticks. The infection can only develop when the skin is exposed to chronic moisture. Clinical signs are more common during the rainy or wet seasons and have a predominantly dorsal distribution. Lower extremities can also be affected (pastern area/"grease heel") when horses are kept in a damp environment (dew at pasture or muddy paddocks). Normal, dry skin cannot be infected. In the early stages, crusts and papules/pustules are much easier to feel than to see. Lesions coalesce and become exudative, matting hair together (paintbrush effect). The hair comes off with the crusts and under the surface is a thick, creamy, white/yellow/greenish exudate. Active lesions are often painful but rarely pruritic. Healing lesions (more chronic stage) usually have dry crusts, scaling and alopecia (ringworm-like). The diagnosis may be made by impression smears taken directly from the underside of fresh crusts to look for coccoid cells arranged in 2-8 parallel rows (railroad tracks). Unfortunately, in the chronic healing or dry stage, cytology is rarely positive. Histopathology may reveal suppurative luminal folliculitis, intradermal pustular dermatitis and most characteristically palisading crusts of hyperkeratosis alternating with dried serum, degenerative neutrophils, and the visible branching organism. However, dermatophilosis cannot be ruled out until negative culture results of crusts are returned (tell the laboratory that you suspect dermatophilosis because the organism can be difficult to grow). Most affected horses recover spontaneously when conditions improve by providing shelter from rain or turn out only when the dew has dried. Grooming and bathing to remove the crusts is also important but sedation might be necessary. Effective topical medications include: iodine shampoo/spray, 5% lime sulphur (LymDyp), or 4% chlorhexidine shampoo/spray applied daily for 3-5 days, then weekly until healing. For severe cases systemic antibiotic treatment may be necessary: penicillin (short-term) or sulfonamides (long-term for more chronic cases) past resolution of all active lesions. Other important management recommendations include insect control, improved hygiene, and improved nutrition. Unfortunately, horses do not develop significant immunity against the organism; thus, reinfection may occur.

*Pemphigus foliaceus* (PF) is the most common autoimmune skin disease of the horse and is likely to be seen by most equine practitioners. Horses with PF may crusting papules over the head, neck, and shoulders and may also have systemic
signs, including decreased appetite, limb and ventral edema, and mild fever. Skin biopsies are often diagnostic as they reveal subcorneal or intraepidermal vesicopustules with marked acantholysis, produced by autoantibodies against cell adhesion proteins, specifically desmosomal proteins. Treatment with corticosteroids (dexamethasone, 0.1 mg/kg, PO, q 24 h for 5-10 days, followed by decreasing the dose and prolonging dosing interval to q 48-72 h) and gold salts has often been effective. Azathioprine is used as a steroid sparing drug but bioavailability of this drug is low (4 ± 3%) after oral administration at this dosage.