

COM October 2026

Case history and Signalment

A 19-year-old Arabian gelding from Iowa presented to the ISU Equine Clinic with a progressive hair loss over the last couple of weeks. Physical examination showed moth-eaten alopecia with mild scaling and hyperpigmentation all over the body involving the nostrils, periocular, neck, thorax, and appendages. These areas of patchy alopecia range from 0.5-2.5 cm in diameter. The animal showed significant pruritus throughout the examination. Three punch biopsies were taken from the left neck.

Figure 1. Full body.



Figure 2: Right side of the neck.



Figure 3: Left side of the face.



Figure 4: Histopathology. 4X.

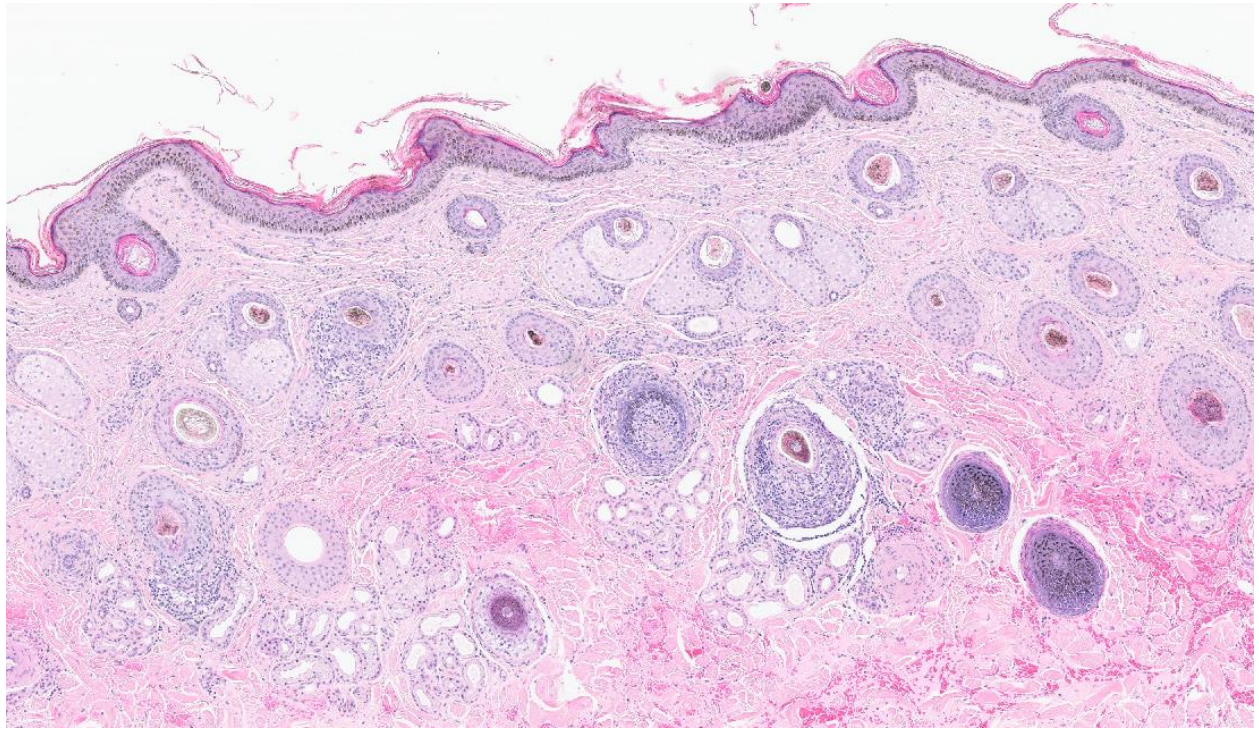


Figure 5: Histopathology. 10X.

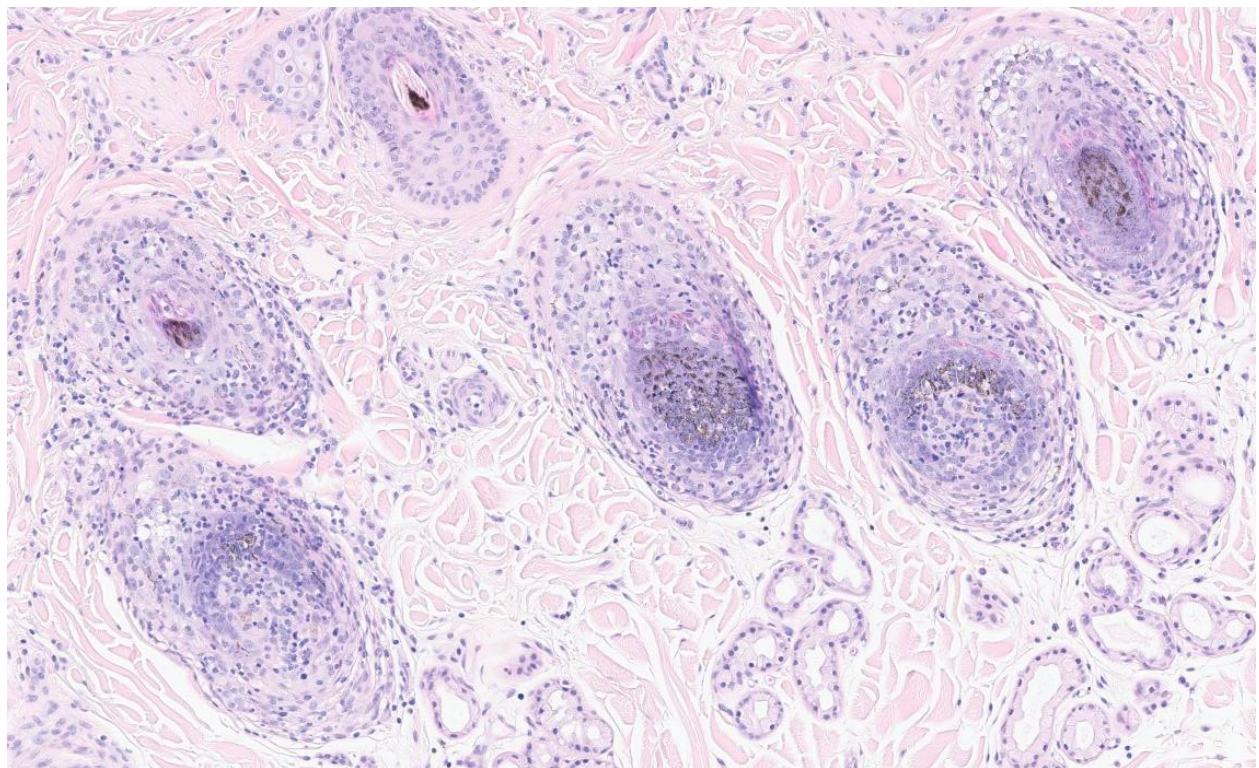
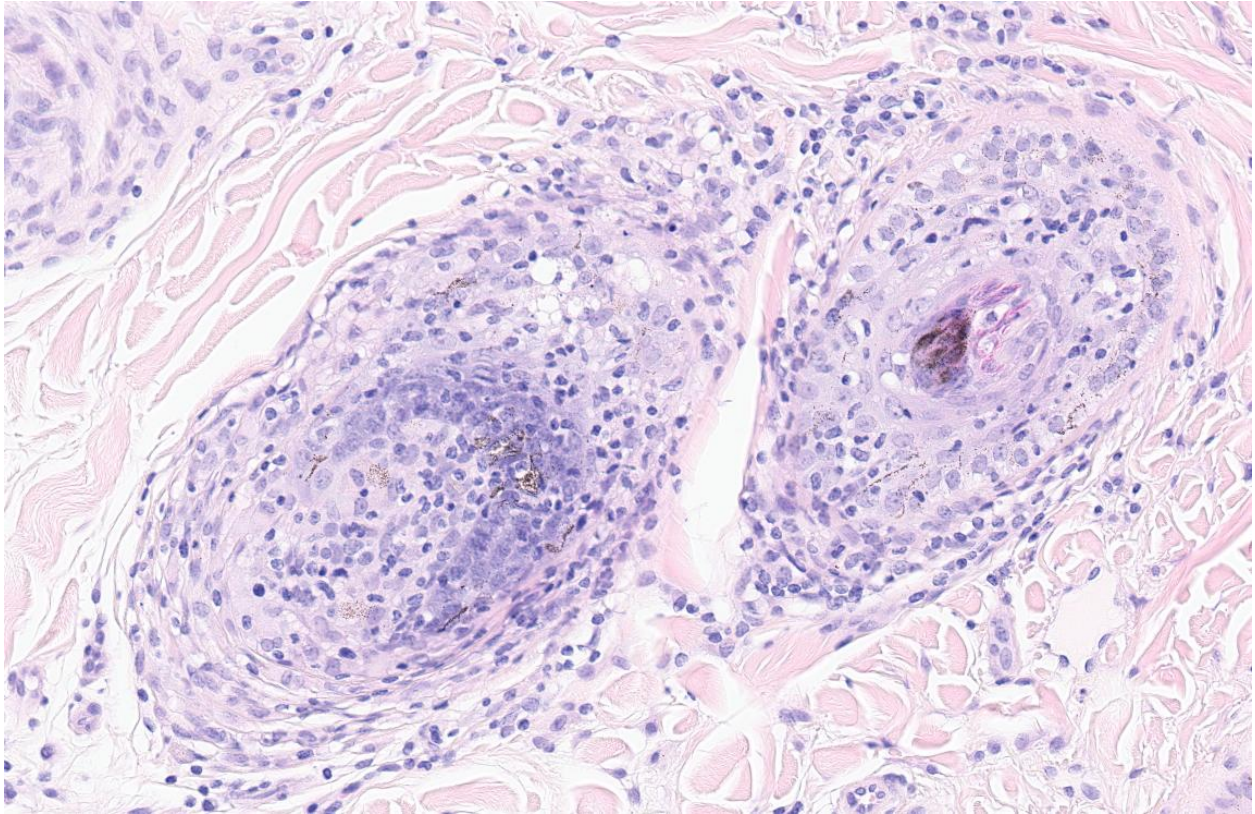


Figure 6: Histopathology. 20X.



Histopathologic Description

Three sections of haired skin were evaluated; and all sections have similar histologic findings. Anagen bulbs are swarmed and infiltrated by moderate numbers of lymphocytes, as well as a few melanomacrophages. Dermal papillae are also infiltrated by similar inflammatory cells; the inflammation slightly extends up to follicular epithelium at the level of isthmus in some of the affected hair follicles. The epidermis is mildly hyperplastic with the stratum corneum expanded by mild orthokeratotic hyperkeratosis. The epidermis is also heavily pigmented. All hair follicles are noted to be in anagen phase.

Morphologic Diagnosis

Haired skin: Moderate lymphocytic bulbitis

Questions

What is the most likely diagnosis of this skin condition?

A) Insect Bite Hypersensitivity

B) Dermatophytosis

C) Alopecia Areata

D) Cutaneous lymphoma

E) Multisystemic Eosinophilic Epitheliotropic Disease

Comments:

Alopecia areata (AA) has been reported in a range of species, including dogs, cattle, horses, and mice, as well as humans. It is classified as an inflammatory alopecia in which inflammation targets the hair bulbs of anagen follicles. Typical histologic findings across species characteristically reveal lymphocytic infiltration targeting the anagen bulb, a pattern commonly termed “bee swarming.” Although the specific target antigen has not been fully elucidated, AA is recognized as an autoimmune disease involving autoantibodies against hair follicles and autoreactive T cells; CD3+ and CD8+ lymphocytes predominate within the inflammatory infiltrate.

As the name implies, the primary clinical sign is alopecia. Additionally, nail, claw, or hoof dystrophy has been reported. Pathognomonic features such as “exclamation mark hairs” or “Pohl-Pinkus constrictions” are well-documented in human patients. While these specific hair-shaft anomalies have not been described in the veterinary literature, leukotrichia and a dry, brittle hair coat were noted in one horse in a retrospective study of 15 animals. Interestingly, pruritus has also been recognized across species, though its severity varies considerably.

The age of onset in equine AA ranges from 3 to 15 years, with a median age of 9 years. Lesion distribution varies by species: the face is the most common site in dogs, whereas the scalp is primarily affected in humans. In equine AA, hair loss is typically patchy, predominantly affecting the mane, tail, and face, though it can progress to generalized alopecia.

Because AA is an autoimmune condition, the primary therapeutic goal is immunomodulation. Equine treatment protocols are not supported by randomized, placebo-controlled treatment trials. Evidence is instead largely anecdotal, with mixed

outcomes reported for therapies like 2% minoxidil, topical calcineurin inhibitors, and both systemic and topical steroids. Although alopecia areata is non-scarring, complete hair regrowth is not consistently achieved even in human medicine.

In this patient, because the condition is primarily cosmetic and not life-threatening, no treatment was pursued. Multifocal alopecia persists along the muzzle, neck, dorsum, and trunk, though partial hair regrowth was documented at the 10-month follow-up visit.

References:

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