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J VET Diagn Invest 2012 24: 405 originally published online 7 February 2012
DOI: 10.1177/1040638711435808

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Alopecia areata in two black Angus cows

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Danny W. Scott, William H. Miller

Abstract. Adult onset progressive alopecia and leukotrichia developed in 2 registered black Angus cows, aged 6 and 7 years. Histopathologic findings in skin were prominent melanin clumping and degeneration of matrix cells with formation of giant multinucleate cells within hair bulbs, accompanied by peribulbar melanin incontinence and fibrosis and dystrophic hair shafts. Intrabulbar and peribulbar lymphocytes were cluster of differentiation (CD)3-positive T cells. Findings were characteristic of alopecia areata.

Key words: Alopecia areata; cattle; dermatosis; immunohistochemistry.

Alopecia areata (AA) is a nonscarring form of inflammatory dermatopathy causing hair loss in human beings and animals.^{1,3} Alopecia areata is considered to be an autoimmune disease directed at unique hair follicle antigens,^{1,3} and targeting of melanocyte-associated autoantigens has been proposed.¹ Dystrophic hairs and follicles, plus infiltrates of T cells surrounding hair follicle bulbs (“swarm of bees”), within hair bulbs, and within deep follicular walls are characteristic histologic features.^{1,3} Affected follicles are almost exclusively growing (anagen) follicles engaged in active melanogenesis.¹ In human beings, dark hairs are more likely to be affected than white hairs.¹ In affected people with hair regrowth, dark hairs often regrow white.³ In animals, AA has been described in rats, mice, dogs, cats, horses, nonhuman primates, and cattle.^{3,5–8} Bovine AA has been reported in 2 black and white Holstein cows^{5,7} and in 20 Eringer cows, which are a black-haired breed.⁸ The current report describes AA in 2 American black Angus cows.

Case 1 involved a 6-year-old registered black Angus cow from ranch 1, with an approximately 3–4-month history of patchy hair loss. Lesions began in the axillary area and spread to involve skin of the neck, head, and thorax. Pruritus was not apparent, and the skin surface was normal to slightly crusted. When hair regrew, it was white (Fig. 1). Skin biopsy samples were obtained from multiple sites including affected and nonaffected skin. Skin biopsy samples were also obtained from a clinically normal black Angus cow on the same ranch. Additional biopsy samples were obtained from affected skin 3 months later. Clinical follow-up obtained 10 months later indicated persistent hair loss, although the few hairs that had regrown were white. Alopecia was bilaterally symmetric, sparing the dorsum, tail, and lower limbs. The hair loss was most severe on the face and neck, which were essentially bald.

Case 2 involved a 7-year-old registered black Angus cow from ranch 2, with a 1-year history of approximately 10–12 round patches of hair loss up to approximately 10–13 cm in



Figure 1. Six-year-old black Angus cow with alopecia areata. The photograph was taken at the time of biopsy, approximately 3–4 months since the onset of clinically apparent skin disease. There is patchy alopecia of the head, neck, and trunk with areas of regrowth of white hairs.

diameter on the head, neck, and trunk. Some regrowth of hair that was often white had occurred in the center of some lesions. Pruritus was not reported. Skin biopsy samples from affected areas were obtained. Clinical follow up obtained 8 and 11 months later indicated progression of the hair loss and leukotrichia, with severe hair loss on the face and neck (Fig. 2). Additional skin biopsy samples from affected areas were obtained 21 months after initial sampling.

All biopsy samples were obtained in the months of June through September, which are typically months with biting insects. Findings in hematoxylin and eosin–stained sections

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Figure 2. Seven-year-old black Angus cow with alopecia areata. The photograph was taken 8 months after biopsy diagnosis of alopecia areata, approximately 18 months since the onset of clinically apparent skin disease. Progressive disease has led to severe alopecia of the head, neck, and trunk with extensive regrowth of white hairs.

of affected skin were similar in both cows and were characterized by a small number of intrabulbar and peribulbar lymphocytes with associated peribulbar fibrosis. Degeneration of matrix cells within follicular bulbs with formation of giant multinucleate cells (macrophages, presumptive) containing fine melanin pigment was detected within most hair bulbs, accompanied by prominent melanin clumping and peribulbar melanin incontinence (Fig. 3). Hair shafts were often curled, twisted, or maloriented. Additional findings were moderate perivascular to interstitial eosinophilic dermatitis with mucinous edema and multifocal pustular epidermitis. Mild perivascular to interstitial eosinophilic dermatitis was also present in normal skin areas from cow 1 and in the skin sample from the clinically normal cow. Perifollicular tissue, hair bulbs, follicular walls, and hair shafts were normal in unaffected areas of cow 1 and in the normal cow.

Immunohistochemical studies utilizing antibodies to cluster of differentiation (CD)3 and CD79 α ^a were performed on all sections. Bovine lymph node served as the positive control, and skin biopsy sections incubated with nonimmune serum served as negative controls. Affected skin contained many more lymphocytes than were appreciated with hematoxylin and eosin stain. Almost all perivascular to interstitial lymphocytes and all of the intrabulbar and peribulbar lymphocytes were CD3+ T cells (Fig. 4). CD79 α + B cells were rare in all sections except for interstitially in the first biopsy from case 2. A comparison of skin from a clinically normal black Angus cow from ranch 1 and from unaffected skin from the affected cow on ranch 1 indicated that perivascular to interstitial CD3-positive cells were present in both affected and normal cattle, but that the number was increased in affected cattle. Intrafollicular CD3-positive lymphocytes were not present in normal skin from the affected cow or in skin from the normal cow. Comparison of initial biopsy samples and repeat samples in affected cows indicated that all changes were persistent, with an increase in perifollicular fibrosis and a mild decrease in lymphocytic infiltration over time. The diagnosis in both cows was AA.

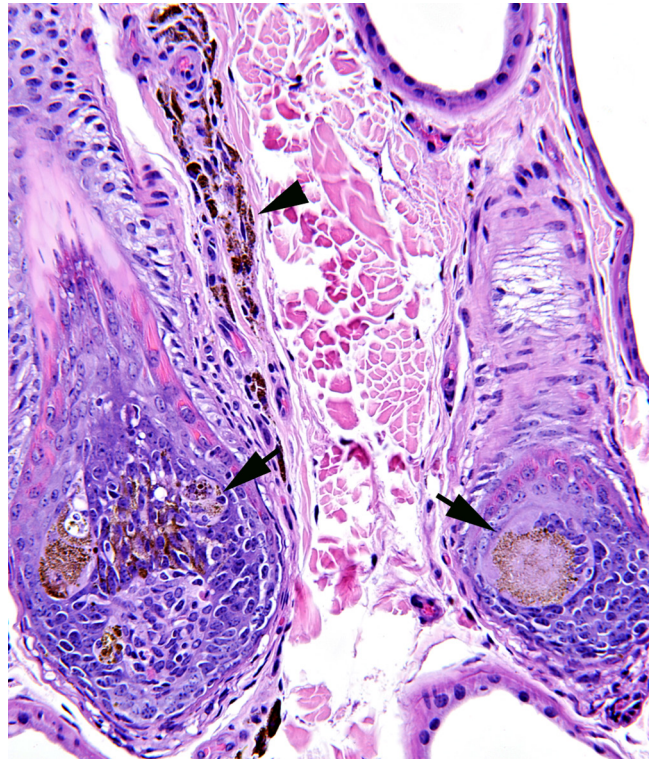


Figure 3. Skin; 6-year-old black Angus cow with alopecia areata. Photomicrograph of 2 hair follicle bulbs with melanin clumping and formation of multinucleate giant cells (macrophages, presumptive; arrows). There is melanin incontinence within the adjacent dermis (arrowhead), and there are a small number of peribulbar inflammatory cells. Hematoxylin and eosin. Bar = 100 μ m.

Based on these 2 cases, it is clear that detection of peribulbar and intrabulbar lymphocytes can be difficult in hematoxylin and eosin-stained sections of AA-affected cattle. One report of adult onset alopecia in a 7-year-old registered black Aberdeen-Angus cow, with histologic findings of dystrophic hairs with pigmentary clumping and incontinence within hair bulbs but with no description of lymphocytic infiltration, is also likely to be a case of AA.² Biopsy samples in that case were obtained approximately 1 year after onset, and the lack of obvious lymphocytic infiltration is consistent with late-stage AA, in which the characteristic lymphocytic infiltration within and around hair bulbs can be markedly reduced.³ Immunohistochemistry for CD3-positive T cells might have revealed lymphocytes not detected in routine stains. Perivascular dermatitis with eosinophils, as seen in the normal Angus as well as in affected cattle in the current report, is a common finding in bovine skin and was described in control Eringer cattle as well as in Eringer cows with AA.⁷ Reaction to biting insects, as previously described in cattle skin,⁶ is suspected.

Black-haired cattle (black Angus, black Aberdeen-Angus, and Eringer) and cattle with areas of black hair (black and white Holsteins) appear to be predisposed to development of AA. In Holsteins, involvement of black-haired areas were

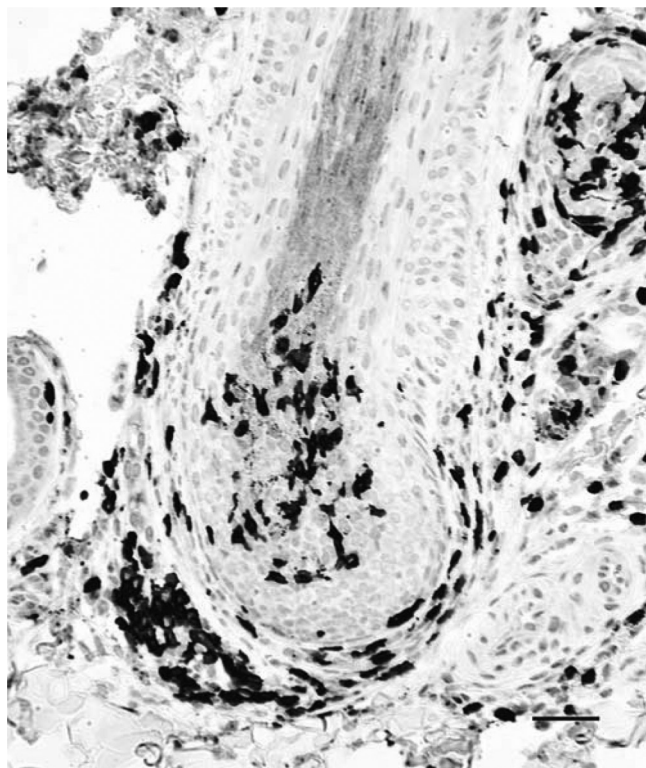


Figure 4. Skin; 7-year-old black Angus cow with alopecia areata. Peribulbar inflammatory cells (“swarm of bees”) and cells infiltrating the hair bulb are cluster of differentiation (CD)3-positive T lymphocytes. Nova red chromogen, hematoxylin counterstain. Bar = 100 μ m.

illustrated in a previous report,⁷ although targeting of white-haired areas of the face were illustrated in another affected black and white Holstein.⁵ Interestingly, AA in black-haired Angus cows and Eringer cattle, and in a case of AA involving black-haired areas of a black and white Holstein cow, has most often been progressive, whereas AA involving white-haired skin of a cow apparently resolved.⁵ In Eringer cattle, 40% were reported to improve with time, although hair regrowth was white.⁸

Genetic susceptibility to AA is suspected in human beings, but AA is poorly penetrant within families and is likely polygenic.^{1,3} Examination of pedigrees from the 2 registered black Angus cows indicated a bull common to the grandsire and great grandsire lines, but this is a popular bull in the lines of many registered black Angus cattle worldwide. Pedigree analysis of Eringer cattle revealed a stronger genetic relationship between AA-affected cattle than between normal cattle, but this difference was not statistically significant.⁷ If the pathogenesis involves melanocyte-associated autoantigens, then cattle with black hair may be predisposed to AA, but too few cases have been reported to draw such conclusions.

The primary differential diagnosis in cases of bovine AA involving black hair is black hair follicular dysplasia.⁴ Dystrophic hairs, melanin clumping, and melanin incontinence are features common to both AA and black hair

follicular dysplasia in cattle. Lymphocytic infiltration was not described in the black and white Holstein cow with black hair follicular dysplasia,⁴ nor would it be expected. However, biopsies were not obtained until the cow was 7 years of age,⁴ and lack of lymphocytes could still represent late-stage AA. The age of onset appears to be the major difference between AA and black hair follicular dysplasia. Follicular dysplasia is apparent soon after birth,⁴ whereas AA has an adult onset, with the youngest reported case occurring at 3 years of age.⁸ Similar to the black Angus cows described in the current report, the majority (89.4%) of affected Eringer cattle were greater than 5 years of age at the time of onset of AA.⁸

The diagnosis of AA in cattle can be suspected based on clinical and histopathologic findings. Immunohistochemical identification of CD3-positive lymphocytes within and surrounding hair bulbs is diagnostic. Immunohistochemistry for CD3 may be particularly useful in chronic cases in which T-lymphocyte infiltrates are often markedly reduced³ and where inflammation may be difficult to detect in routinely stained sections.

Acknowledgements

The authors thank Drs. Barry Cooper, Susan Piripi, and Scott Hendy, as well as Diane Swingley for initial workup of case 2.

Sources and manufacturers

a. Dako North America Inc., Carpinteria, CA.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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