Case Report

Progressive Generalized Leukotrichia with Discoid Eruption on the Planum Nasale of a Border Collie

進行性汎発性白毛症と鼻鏡に円板状の皮疹を認めた ボーダー・コリーの1例

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Abstract: A 1.3-year-old, female Border collie presented with a 2-month history of erythema, progressive scaling and hypopigmentation on the dorsal planum nasale extending to the muzzle and eyelids. Histopathological analysis of the nasal lesion revealed interface dermatitis. Erythema improved after the administration of oral tocopherol acetate and topical betamethasone for 2 weeks. However, the nasal lesion showed a discoid appearance with loss of its normal ‘cobblestone’ architecture, and progressive generalized leukotrichia was recognized. Ophthalmologic examination revealed no evidence of uveitis. A serum antinuclear antibody test was negative. Based on these findings, the present dog was considered to have vitiligo-like leukotrichia with discoid eruption.

Key words: erythematosus, leukotrichia, vitiligo

要 約: 1.3歳齢, 雌のボーダー・コリーに, 2カ月前より始まった鼻背面および眼瞼における紅斑, ならびに進行性の過角化と白斑が認められた。鼻背面の病理組織学的検査では境界部皮膚炎を認めた。紅斑は酢酸トコフェロールの内服および吉草酸ベタメサゾンの外用後2週間で改善したものの, 鼻背面に‘敷石状’の外観を失った円板状皮疹を認めたとともに, やがて白毛が全身へ拡大した。眼科検査によりぶどう膜炎は認められなかった。血清抗核抗体は陰性であった。以上より, 自験例を白斑様の白毛症および円板状皮疹を合併した症例と考えた。

キーワード: エリテマトーデス, 白斑, 白毛症

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Introduction

Hypopigmentation is a term describing a lack or decrease of pigment in the skin (leukoderma) or haircoat

(leukotrichia)⁸⁾. Acquired hypopigmentation can be the result of many factors causing melanocyte destruction, dysfunction or dislocation. In dogs, acquired nasal hypopigmentation is recognized in association with age,

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trauma, severe inflammation (*e.g.* infections, post-inflammatory hypopigmentation), irradiation (*e.g.* sunburn), immune-mediated disorders (*e.g.* pemphigus erythematosus, lupus erythematosus, cutaneous vasculitis, uverdermatologic syndrome, vitiligo), or tumors (*e.g.* epitheliotropic lymphoma, squamous cell carcinoma)^{1-3, 6-8)}. Meanwhile, acquired leukotrichia is rarely seen in dogs and is associated with drugs (*e.g.* vitamin E), or immune mediated disorders (*e.g.* uverdermatologic syndrome, vitiligo)^{2, 3, 7, 8)}. A previous report described skin lesions consistent with cutaneous lupus erythematosus in a human⁴⁾ with vitiligo. However, similar case reports have not appeared in veterinary literature. This report describes a Border collie with a discoid eruption on the dosal planum nasale progressing to generalized leukotrichia.

Case Report

A 1.3-year-old, female Border collie presented with a 2-month history of non-pruritic, progressive scaling and hypopigmentation on the dorsal planum nasale and eyelids. The dog was kept indoor, but went outdoors twice daily. The dog had no history of clinical signs except for skin lesions or heat stroke. The dog also had no episode of drug administration except for prevention of heartworms.

On the initial visit, the dog seemed to be healthy except for the skin lesions. The lesions consisted of symmetrical erythema, hypotrichosis, leukoderma on the dorsal planum nasale and muzzle (Fig. 1A) and eyelids (Fig. 1B). Scaling was also recognized on the planum nasale. The dog had no ocular changes. Careful taken skin scrapings yielded no fungi or ectoparasites. Cytology of the nasal lesions revealed mild parakeratosis, but did not show marked overgrowth of bacteria or *Malassezia* sp. Fungal culture yielded a negative result. Complete blood count and serum biochemistry revealed unremarkable results. From the clinical and laboratory findings, discoid lupus erythematosus (DLE), vitiligo, drug eruption and vasculopathy were considered as differential diagnoses.

With the owners' consent, a skin biopsy of the perilesional area of the planum nasale was performed under general anesthesia. A histopathological examination revealed interface dermatitis with vacuolation of keratinocytes at the stratum basale, hydrophilic degeneration at the stratum basale and dermo-epidermal junction, moderate infiltration of lymphocytes (arrow) and melanophages (arrowhead) (Fig. 2). Apoptosis of keratinocytes was unremarkable in the

Fig. 1. Clinical features of the present case on the initial visit. (A) Note the leukoderma with atrophic skin on the planum nasale. (B) Leukoderma is also seen on the eyelids.

Fig. 2. Histopathology of the perilesional area of the nasum in the present case. (A) Infiltration of mononuclear cells was recognized exclusively at the dermo-epidermal junction and superficial dermis. (B) Note the vacuolation of keratinocytes at the stratum basale, intense hydrophilic degeneration at the stratum basale and dermo-epidermal junction, and lymphocyte infiltration (arrow) and melanophages (arrowhead).

Fig. 3. Clinical course of the present case. (A and B) Eight months after the initial visit (2 years old). (C and D) Two years and 8 months after the initial visit (4 years old). Note that leukotrichia developed from the head and became generalized with the course of time.

epidermis and hair follicles. Atrophy of the hair follicles was recognized in the muzzle (data not shown). Histopathological changes suggesting vasculopathy (*e.g.* pale-stained collagen and dermal blood vessels, dermal edema) were not seen in the specimen.

The dog was treated with sun-avoidance and topical betamethasone valerate mixed with gentamicin sulfate (Rinderon-VG ointment, Shionogi & Co., Ltd., Osaka, Japan) on the nasal lesion, twice daily for 2 weeks. The therapy was discontinued because erythema had improved, and the dog showed no pruritic behavior on the skin lesions. Oral tocopherol acetate (Juvela, Eisai Co., Ltd., Tokyo, Japan) was also administered at a dose of 50 mg/head once daily for 4 months. However, the nasal lesion showed a discoid appearance with loss of its normal ‘cobblestone’ architecture and well-circumscribed atrophic skin. In addition, leukoderma extended towards almost the entire planum nasale, eyelid, lip and oral mucosa. Thus, the administration of oral tocopherol acetate was discontinued. Moreover, leukotrichia was recognized on

the head, and gradually extended towards the entire body surface (Fig. 3) until the dog became 4 years of age. The nasal lesion with discoid appearance had not improved at the time when the dog exhibited generalized leukotrichia. Careful ophthalmologic examination was performed, but revealed no evidence of uveitis (Fig. 4), and the dog seemed to have normal eyesight. Complete blood count, serum chemistry and urinalysis yielded unremarkable results. A serum antinuclear test was negative. After 4 years of age, the leukotrichia gradually improved from the caudal side of the body toward the cranial site (Fig. 5), and pigmented spots returned to the planum nasale, lip and oral mucosa without any specific therapies (data not shown).

Discussion

In the present case, leukoderma and leukotrichia developed at less than 3 years of age, and gradually extended to the entire body surface and oral mucosa. The abnormalities gradually improved from 4 years of age.

Fig. 4. Clinical features of the eyes at 4 years of age. Uveitis is unremarkable both in the left (A) and right (B) eyes.

The clinical history of the present case completely fitted that of dogs with vitiligo, in which leukotrichia is commonly recognized and the affected areas may repigment^{2, 8)}. However, the nasal lesion of ‘discoid’ appearance with histopathology of moderate interface dermatitis is not a typical finding in dogs with vitiligo. The discoid appearance with loss of its normal ‘cobblestone’ architecture is a common clinical feature of dogs with DLE^{1, 6)}. However, the histopathological findings of the skin lesion in the present case did not strongly support a diagnosis of DLE, as there was no intense, lichenoid-interface dermatitis. The histopathology of cell-poor interface dermatitis is recognized in dogs with drug eruption, erythema multiforme, ischemic dermatopathy, lupus erythematosus, and uveodermatologic syndrome^{1-3, 6-8)}. However, we think that the clinical findings of the present case are not typical of any of these diseases, as the dog had no target lesions or erosions/ulcers, and no edematous changes in the extremities, or ocular or systemic signs apart from the skin lesions.

Fig. 5. Clinical features of haircoats at 5 years of age. Pigmented hairs were observed on the trunk and hindlimbs. Pigmentation was also recognized on the planum nasale, except the dorsal aspect.

The exact pathogenesis of vitiligo in humans and dogs is still unclear. Marked breed predilection suggests that vitiligo in the dog has a mode of inheritance. However, vitiligo in humans may have a complex pathogenesis, involving the lack of normal protective mechanisms to eliminate toxic melanin precursors, neurochemical mediators that inhibit melanogenesis or destroy melanocytes, and induction of autoantibodies against antigens in the melanogenic system^{2, 5)}. Histopathologically, hypopigmented lesions in dogs with vitiligo typically exhibit marked reduction or complete absence of epidermal melanocytes with minimal or no histopathological changes at the dermo-epidermal junction^{2, 8)}. However, the histopathology of the present case was characterized by moderate interface dermatitis with intense hydropic degeneration and moderate lymphocyte infiltration, which is not supportive of a histopathological diagnosis of vitiligo. The exact mechanisms underlying the interface dermatitis on the planum nasale were unclear. In addition, the exact mechanisms underlying transient leukoderma and leukotrichia in the present case were also unclear, although transient melanocyte destruction, dysfunction or dislocation of the skin and hair bulbs were likely associated with the leukoderma and leukotrichia.

Dogs with DLE commonly have skin lesions on the planum nasale and muzzle^{1, 6)}. Collies are known to be predisposed to DLE. Histopathological findings of dogs with DLE are characterized by intense, band-like or lichenoid dermal infiltrate and apoptosis of basal keratinocytes. The apoptotic keratinocytes are sometimes

sparse in the lesions of DLE¹⁾. While the clinical findings in the nasal lesion in the present case were lupus-like, the intensity of dermal infiltration did not strongly support a diagnosis of DLE. It was also considered whether the discoid, nasal skin lesion was a non-specific inflammatory pattern which is seen in collies. Lupus-like drug eruption against topical ointment application and topical corticosteroid reaction should also be considered if the lesions disappear after discontinuing topical drugs¹⁾. However, the nasal lesions of the present case persisted for over 2 years after topical drug application was discontinued. Thus, adverse drug reactions against the topical drug were unlikely in the present case. It has been reported that leukotrichia, which may be associated with vitamin E therapy, was recognized in a collie with LE⁸⁾. Association of tocophenol acetate with leukotrichia was unlikely in the present case, as leukotrichia progressed for over 2 years after discontinuing the vitamin E therapy.

In summary, the clinical findings of the present case suggested vitiligo-like leukotrichia and discoid lupus-like eruption, while the histopathological pattern of the nasal lesion was not supportive of a diagnosis of DLE. Further studies with additional cases will be needed to understand the pathophysiology of the nasal lesions with discoid appearance seen in collies.

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