http://dx.doi.org/10.4322/rbcv.2014.074

Black hair follicular dysplasia in a Brazilian Terrier: a case report

Displasia follicular dos pelos pretos em Terrier Brasileiro: relato de caso

Lílian Sayuri Tatibana,* Daniella Naves Paglioni,** Adriane Pimenta da Costa Val***

Abstract

Black hair follicular dysplasia (BHFD) is a rare genetic disease in dogs. A black and white female Brazilian Terrier (one year and three months old) was referred with a history of progressive hair loss only in black hair areas. Following anamnesis, clinical examination, trichographic analysis of hairs and histopathological examination of skin fragments, the animal was diagnosed with BHFD. This report describes for the first time a case of BHFD in a pure bred Brazilian Terrier.

Keywords: dog diseases, hypotrichosis, alopecia.

Resumo

A Displasia Folicular dos Pelos Negros (DFPN) é uma doença genética rara em cães. Um cão Terrier Brasileiro, fêmea, de um ano e três meses de idade foi apresentado com histórico de perda progressiva de pelos, apenas em áreas de pelos negros. Após anamnese, exame clínico, tricografia e exame histopatológico de fragmentos da pele, o animal foi diagnosticado como portador de DFPN. Este trabalho descreve pela primeira vez um caso de DFPN em um Terrier Brasileiro pura raça.

Palavras-chave: doenças do cão, hipotricose, alopecia.

Introduction

Skin and hair colour is determined primarily by the occurrence, quantity and metabolism of melanin, the most common forms of which are eumelanin, responsible for black to dark brown pigments and pheomelanin, from which yellow to red pigments are dependable. The production of melanin is genetically determined and controlled by various enzymatic steps, giving rise to a large variation in skin and hair colours (Scott et al., 2001).

The melanogenesis and storage of melanin occurs within membrane-bound organelles, the so-called melanosomes originating from the Golgi apparatus, that are present in melanocytes located in the epidermis and hair follicles. The melanocytes of the hair follicle are, however, only active during the anagen phase of hair growth. The melanosomes undergo various stages (I – IV) of development, following which they are transferred to the tips of dendrites and subsequently to adjacent keratinocytes (or other types of cells) through a process termed cytocrinia (Scott et al., 2001). As the keratinocytes ascend through the epidermis, melanin is released from the transferred melanosomes. Skin colour therefore depends on the size, rate of transfer and degree of dispersion of melanosomes (Selmanowitz et al., 1977).

Canine follicular dysplasias (CFDs) are a class of hair and hair follicle abnormalities that frequently have a hereditary basis. CFDs are relatively uncommon and may not be associated with irregularities in hair colour, as seasonal flank alopecia and follicular lipidosis. Colour-associated CFDs include colour dilution alopecia (CDA) and black hair follicular dysplasia (BHFD). The former is found in dogs bearing the diluted coat colours blue or fawn (dilution of black and brown respectively) and is characterised by poor quality hair and varying degrees of alopecia (Gross et al., 2005). BHFD is characterised by focal or generalised loss of black hairs and is typically found in multicoloured dogs (Scott et al., 2001).

This paper presents the first report of BHFD in a pure bred Brazilian Terrier, formerly known in Brazil as Fox Paulistinha. The peculiarities of the case are discussed in the light of existing knowledge about the disease.

Case report

A tricolour (mainly black and white with a small patch of yellow hair on the head) female Brazilian Terrier (Fig. 1), of age one year and three months, was referred to the Dermatology Service of the Veterinary Hospital of Universidade Federal de Minas Gerais, Brazil, with an 11 month history of permanent alopecia in the dorsal region. The dog had never shown signs of pruritus. Clinical examination revealed alopecia and hypotrichosis, restricted to the black-haired areas, and discreet floury-type desquamation (Fig. 2A). No dermatophytes could be detected by visual or mycological analysis of the hair or flaky skin. Other skin or endocrine diseases that could be presented as alopecia and hypotricosis were ruled out by serum thyroid hormones quantification and by

*Programa de Especialização em Residência em Medicina Veterinária, Nível II, Hospital Veterinário, Escola de Veterinária.

**Veterinário Autônomo.

***Escola de Veterinária da Universidade Federal de Minas Gerais. Autor para correspondência: Av. Antônio Carlos, 6627, 31270-901, Escola de Veterinária, Departamento de Clínica e Cirurgia Veterinárias, adriane@ufmg.br.

Figure 1: Brazilian Terrier presenting black hair follicular dysplasia

trichography and histopathological examinations, as described as follows:trichography of plucked white hairs revealed a normal architecture with no observable anomalies (Fig. 2B), whereas the black hairs presented irregular hair shaft outlines and bulges containing an excessive accumulation of melanin (Fig. 2C and 2D). Histopathological analysis of skin fragments revealed pigment-related follicular dysplasia in black-haired areas with alopecia and those containing depigmented hairs, whilst fragments collected from white-haired areas presented a normal aspect. Based on the history, clinical findings and complementary tests, the dog was diagnosed with BHFD and was prescribed a treatment involving an antiseborrheic shampoo and a moisture product. The owners were informed about the hereditary and incurable nature of the disease and advised not to employ the dog for breeding purposes.

Discussion

Selmanowitz et al. (1977) described BHFD in mongrel dogs, but the disease has since been identified in pure breeds including Border Collie, Bearded Collie, Jack Russell Terrier, Salukis (Schmutz et al., 1998), Chihuahua (Delmage, 1995), Cavalier King Charles, Basset Hound, Dachshund, Yorkshire Terrier, Doberman (Cunha et al., 2005), Cocker Spaniel, Pointer, Setter Gordon, Papillon, and Schipperke (Lewis, 1995). The present report constitutes the first published case of the disease in a pure bred Brazilian Terrier.

According to Schmutz et al. (1998) and Bomhard et al.(2006) colour-associated CFDs generally result from the accumulation of large numbers of stage IV melanosomes in the melanocytes of the epidermis and hair bulb accompanied by no, or insufficient, transfer to the keratinocytes. Although the specific causes of BHFD have not been completely elucidated, the disease is known to be associated with genetic abnormalities of the hair follicle (Miller, 1991). There is evidence that BHFD is determined

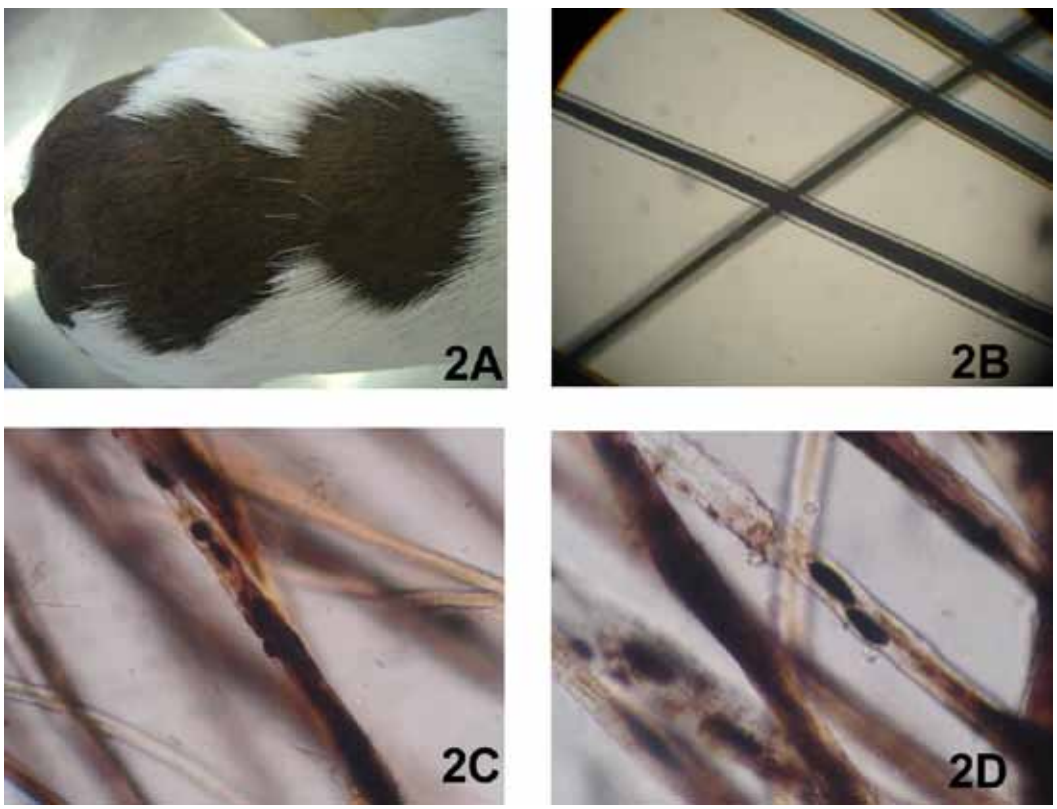

Figure 2: Closer view of a Brazilian Terrier presenting black hair follicular dysplasia: (A) hypotrichosis restricted to the black-haired areas; (B) photomicrograph of the normal adjacent white hair shaft presenting no pathological alterations; (C) photomicrograph of the black hair shaft exhibiting protrusions of macromelanosomes distributed in the hair cortex and loss of differentiation between the hair cortex and medulla; and (D) photomicrograph of the black hair shaft revealing agglomerates of melanin distributed unevenly in the cortex and medulla together with an irregular and undefined hair shaft outline.

by an autosomal recessive character that induces abnormal morphogenetic changes in the melanocytes and results in defective cytocrinia (Selmanowitz et al., 1977; Schmutz et al., 1998). This hypothesis is supported by the presence of only small amounts of melanin in the keratinocytes of dogs presenting BHFD (Bomhard et al., 2006), as found in this case. Another cause of BHFD has been attributed to a deficiency of melanocyte-stimulating hormone resulting in exposure of the hair bulb cells to toxic melanin precursors. This hypothesis has, however, yet to be confirmed (Schmutz et al., 1998). A recent study has revealed that a mutation either in the canine *Mlph* gene (encoding the carrier protein melanophilin that is responsible for transferring melanosomes to the actin cytoskeleton in melanocytes) or in a locus nearby, was responsible for the diluted coat colour phenotype observed in Dobermann Pinscher and German Pinscher dogs (Philipp et al., 2005). Other studies have also indicated that mutations in the *Mlph* gene may be responsible for colour-linked CFDs (Bomhard et al., 2006).

Although BHFD-affected puppies exhibit normal coats at birth, the first signs of the disease are detectable at about four weeks (Lewis, 1995; Medleau e Hnilica, 2003), but in the case here presented no signs could be observed before four months of age. By the other hand, all black hair abnormalities were similar to those described before: only the black-haired areas are affected, whereas the light-haired areas are preserved and the hair is of normal size, density and texture (Selmanowitz et al., 1977; Schmutz et al., 1998). Initially, in the case here presented, it was possible to observe progressive hypotrichosis of the black-haired areas, although pruritus is absent as described elsewhere (Miller, 1991). As in the literature, the black-haired areas became progressively dull, dry and weak, and the hair could be readily plucked (Scott et al., 2001; Gross et al., 2005).

Although CDA and BHFD are characterised by different clinical signs, the histopathological aspects are similar and it is believed, therefore, that they are different manifestations of the same disease (Gross et al., 2005). Hence, the differential diagnosis between BHFD, CDA and related diseases (i.e. dermatophytosis, demodicosis, superficial pyodermitis and endocrine alopecia) is very important (Selmanowitz et al., 1977; Medleau e Hnilica, 2003; Guaguère e Rubliales-Degorge, 2006).

Diagnosis of BHFD is based on anamnesis, physical examination, trichography and skin histopathology (Gross et al., 2005, Guaguère e Rubliales-Degorge, 2006). Trichography of the black hair reveals deformities in the hair shaft including unclear definition between the hair layers (medulla, cortex and cuticle), irregular outline of the hair shaft and bulges of macromelanosomes, indicating excessive accumulation of melanin (Gross et al., 2005). Indeed, in this case, trichography performed in the first examination lead to the suspicious of BHFD, confirmed by histopathological examination of skin fragments stained with haematoxylin eosin, which revealed irregular and dilated hair follicles that are filled with keratin, hair fragments and clumps of melanin, and the predominance of resting (telogen phase) hair follicles (Bomhard et al., 2006). The hair shafts are reduced or absent, typically fractured and containing large quantities of free melanin. The melanosomes predominate throughout the hair follicle, including the outer root sheath, bulb and hair shaft, whereas free melanin can be observed in the melanophages surrounding the bulb and follicle (Bomhard et al., 2006). Adjacent light-haired areas were also, however, totally normal, as described elsewhere (Medleau e Hnilica, 2003).

There is no cure for BHFD and treatment of the disease is limited to palliatives (Selmanowitz et al., 1977; Guaguère e Rubliales-Degorge, 2006). As described in literature (Morris, 2004) BHFD is not typically associated with problematic folliculitis or seborrhoea, which is common in CDA. So, the application of keratomodulating shampoos and moisturisers were recommended in the case presented here. Although alopecia is irreversible, the disease does not affect the quality of the tegument or life of the animal (Medleau e Hnilica, 2003) as it may occur in CDA (Morris, 2004). However, owing to the hereditary nature of the disease, affected dogs should not be employed for breeding purposes (Selmanowitz et al., 1977).

In summary, the present case report describes for the first time the rare occurrence of BHFD in a Brazilian Terrier and emphasises the importance of differential diagnoses in cases presenting loss of hair in focal areas and absence of itchiness. Moreover, the first symptoms of BHFD were not observed by the owners until the puppy was 4 months old, indicating that there is a possibility that the disease might emerge later than the 3 – 4 weeks that had been previously reported (Lewis, 1995; Medleau e Hnilica, 2003). However, it should be noted that information provided by anamnesis might not be totally accurate. Finally, it is important to highlight the value of trichography, a swift and straightforward technique that can be used by veterinarians in normal practice to assist in the identification of rare diseases such as BHFD.

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