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Therapeutic management of canine atopic dermatitis in a German shepherd dog: A case report

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Abstract

A German shepherd dog having severe itching, hyperpigmentation, alopecia and diffuse purulent lesions was presented at the Govt. Veterinary polyclinic, Bundi. The common causes of dermatitis condition *viz*. mange/mite, were screened through laboratory examination and ruled out. As per the history and ruling out the other infective agents for causing the dermatitis, the case has been diagnosed as atopic dermatitis with secondary lesion develop due to bacterial and fungal infection. Anti-allergic/antihistaminic drugs along with the administration of corticosteroids and nutritional supplement of omega fatty acid had showed marginal recovery in the dog.

Keywords: Alopecia, itching, diffuse purulent, canine atopic dermatitis

Introduction

Canine atopic dermatitis (CAD), although not completely understood, seems to result from a combination of genetic and environmental factors that induce skin barrier dysfunction, immune dysregulation, skin microbiota dysbiosis (Marsella 2021; Hensel et al. 2015) [1, 2]. Canine atopic dermatitis (CAD) is estimated to affect 15% to 30% of the canine population (Scott and Miller 1999) [15] and in most cases, a life-long disease. The exact pathogenesis of CAD is not yet completely established, but it is thought to involve immunoglobulin (Ig) Emediated immediate and late-phase hypersensitivity reactions to environmental allergens (Scott et. al. 2001) [14]. Dogs with atopic dermatitis have a defect with their skin's natural protective barrier that cause itching in these dogs (Rebecca et al., 2021). This itching support the skin microbiome as the secondary source of infections which intensify the severity of CAD (Santoro et al. 2015) [20]. Microbial culture-based studies in dogs showed that the most prominent bacterium on lesional skin of dogs with AD is Staphylococcus pseudintermedius, whereas Malassezia pachydermatis is the main fungal representative (Miller et al., 2013) [3]. Clinical signs appears usually between 6 months and 3 years of age. The first symptom is generally persistent itching, followed by erythema, papules, and pustules, defined as "primary skin lesions" (Griffin and De-Boer, 2001; Favrot et al., 2010; Eisenschenk, 2020) [6, 4, 7]. There are "secondary skin lesions" in chronic CAD, such as alopecia, cutaneous lichenification, and often bacterial infections (DeBoer and Griffin, 2001; Eisenschenk, 2020) [5,7].

Materials and Methods Case History and Observation

A 3-year-old, male, German shepherd dog, with body weight of 30 kg was presented with history of chronic pruritus. The onset of pruritus started 6 month ago with scratching the area near neck and hind limb.

General physical examination

The patient appeared dumb but responsive. The heart rate was found to be normal 80 bpm, the respiratory rate was normal 20 brpm and the rectal temperature was high 103.5 °F.

Dermatological examination

During the consultation the patient occasionally scratched his ears and tried to reach the paws and theme-dial aspect of the thighs bilaterally through the muzzle. On otoscopic examination, both auricles had no lesion. The external auditory canals appeared moderately erythematous. There were no signs of purulent otitis. The clinical examination of the lateral aspect of the

Corresponding Author Munesh Kumar Pushp Teaching Associate, Pashu Vigyan Kendra, Kumher, Bharatpur, Rajasthan, India thighs and neck revealed spotty hyperpigmentation, lichenification, alopecia and diffuse purulent lesions showing pyodermatitis over the skin. No ectoparasites were seen. There were no other primary or secondary dermal lesions on the rest of the body.

Diagnosis

CAD is also a diagnostic challenge. Pathognomonic signs or

specific biomarkers have not been identified. The diagnosis is generally made to exclude other diseases with similar symptoms, such as ectoparasitic infestations (Hill *et al.*, 2006; Hensel *et al.*, 2015) ^[2, 8]. Allergy tests used are the evaluation of skin reactivity by Intra Dermal Testing or the detection of IgE by Allergen-Specific IgE Serology (ASIS) test (Hill *et al.*, 2006; Hensel *et al.*, 2015) ^[2, 8], not conducted in this case.





Picture of German shepherd dog having hyper pigmentation, alopecia and diffuse purulent lesions showing dermatitis.

Treatment / Management

The symptomatic treatment of CAD, consists of the administration of topical or systemic glucocorticoid, antifungal, implementation of a flea control regime, dietary supplementation with essential fatty acids, antibiotic treatment and frequent shampoos (Olivry Saridomichelakis, 2013; Olivry et al., 2015; Santoro, 2019) [9, ^{10, 11]}. As Malassezia pachydermatis (M. pachydermatis) is commensal yeast that is commonly found on mammalian skin and has been recognized as a very common cause of dermatitis in dogs (Santoro et al. 2015) [20] and the relative abundance of the Staphylococcus genus on the skin of AD dogs compared to healthy controls (Bradley et al. 2016; Bierre et al. 2017) [18, 21]. Thus, our treatments need to target various areas concurrently to correct or decrease the negative effects of an excessive self-perpetuating inflammatory response.

The dog was treated with injection of antihistaminics (Chlorpheniramine maleate) for five days intramuscularly (i/m), corticosteroid (Prednisolone @1mg/kg B.W.) for five days i/m, anti-fungal drug (Ketoconazole @ 10 mg/kg B.W.) orally bid for 7 days, antibiotic (Ceftriaxone-Tazobactum antibiotic @ 25 mg/kg B.W.) i/m bid for 5 days, Ivermectin @ 0.2 mg/kg B.W. S/C once, anti-fungal dusting powder containing chlorhexidine applied locally for 7 days. On supportive therapy, vitamin supplement containing omega fatty acid, was given orally for two weeks. Owner was advised to keep the body dust free and moist free. After a week owner brought back the dog and showed slight improvement. Though some improvement noticed, but the dog was not totally free of pruritus.

Result, Conclusion & Discussion

The Atopic dermatitis is a genetically predisposed inflammatory and pruritic allergic skin disease. Alterations in epidermal barrier function is a common factor that contribute to the occurrence of disease (Tarpataki, 2006) [16]. Standard therapeutic protocols of canine atopic dermatitis include the use of glucocorticoids, antihistamines, omega-6/omega-3 fatty acid supplements, topical antipruritic agents, antibiotics, antifungal and combinations thereof (Scott *et. al.* 2001) [14]. Specific skin test could not be performed in this case and the specific immunotherapy was not tried. Glucocorticoid was

used traditionally and antihistaminics used also act as synergestic to reduce the doses of glucocorticoids (Christopher et. al. 2004) [12]. The responses to antihistamines in dogs with CAD are notoriously individualized and unpredictable (Scott et. al. 2001, Scott and Miller 1999) [15]. Effective control of pruritus was achieved by using chloropheniramine in a percentage of dogs with CAD (Scoot and Miller, 1999) [3]. Decreased biodiversity of the microbiome and an increased presence of Staphylococcus has been reported in atopic dogs and associated with clinical flares of the disease (Rodrigues, 2017) [17]. Longitudinal studies in dogs with CAD showed that antipruritic treatments restored biodiversity and normalized skin barrier parameters (Bradley et al., 2016) [22]. Topical antimicrobial therapy has also been reported to increase biodiversity on the skin in atopic dogs (Chermprapai et al., 2019) [19]. The responses of treatment though reflected well but not achieved successful in this case. Tarpatki N (2006) also reported that no single treatment is universally effective in treating canine Atopic Dermatitis.

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