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Case Report Open Acces

Systemic Lupus Erythematous like Condition in a dog with Leishmaniosis

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ABSTRACT

Background: Canine leishmaniasis is endemic in Spain and should be included in the differential diagnosis of almost all sick dogs in endemic areas.

Clincial summary: This report describes an atypical case of systemic lupus erythematosus-like (SLE-like) condition in an 8-year-old male mixed-breed dog due to Leishmania infection. All clinical signs and clinical-pathological alterations remitted with anti-leishmania treatment, except for skin lesions that required subsequent immunosuppressive therapy for complete resolution.

Conclusions and clinical importance: This is the first documented case of a SLE-like condition in a dog with leishmaniosis to the best of the author´s knowledge.

Keywords: Canine leishmaniosis; Ulcerative dermatitis; Systemic lupus erythematosus; Humoral immune response; Interface dermatitis; Immunosuppressive therapy

Introduction

Canine leishmaniosis (CanL) is a zoonotic and chronic disease, endemic in Mediterranean countries [1]. Clinical outcome in CanL is dependent on the host immune response; a cell-based immune response (Th1) controls the Leishmania infection whereas a humoral response (Th2) leads to the clinical disease with the production of a massive amount of antibodies, the formation of immune complexes (IC) and their deposit in different organs and tissues [1-3]. A similar pathogenic mechanism with T-cell dysfunction, polyclonal B-cell activation and production of autoantibodies against different tissue antigens occurs in systemic lupus erythematous (SLE), a severe, autoimmune and multi-systemic disease [4]. A systemic clinical picture with fever, skin lesions, polyarthritis, polymyositis, glomerulonephritis, anemia, leukopenia, thrombocytopenia and gammopathy can appear in both diseases [2-4].

The diagnosis of SLE requires the presence of autoimmune disease in at least three different organs or systems or the involvement of two organs or systems linked to the presence of antinuclear antibodies [4]. The diagnosis of CanL in endemic areas is complex since the clinical spectrum and the range of clinicopathological abnormalities are wide and it is necessary to differentiate active disease from infection [5].

Case Report

An 8-year-old male mixed-breed dog with a presumptive diagnosis of Leishmaniosis was referred to a specialised dermatology service due to persistent ulcerative-crusting skin lesions that didn't respond to the conventional treatment against Leishmaniosis.

Two months before the dog was presented to the general practitioner with fever, weight loss, joint pain and skin lesions. The clinicopathological alterations were anaemia, leukopenia and polyclonal gammopathy. Leishmania IFAT was positive with a low level of antibodies, and the dog was treated with oral allopurinol (10 mg/kg; Zyloric®; Faes Farma, Vizcaya, Spain) twice daily for two months and meglumine antimoniate, (70 mg/kg, subcutaneous; Glucantime®; Merial Barcelona, Spain) once daily for 38 days. At the time of referral, his general condition had improved but not his skin.

The dermatological examination revealed multiple ulcerative lesions and dry crusts accompanied by alopecia on the face, trunk, and extremities (Figures 1-3). The hair was easily plucked off in strands with the crusts (Figure 4), causing alopecia in the affected areas. The differential diagnoses included leishmaniosis, SLE, ischemic dermatopathy, vasculitis and erythema multiforme.

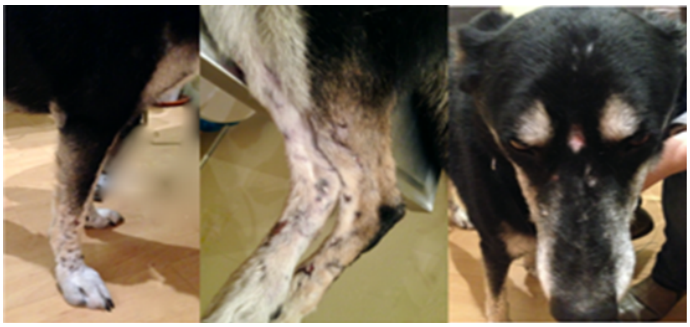

Figure 1: Alopecia and ulcerative lesions in face and extremities

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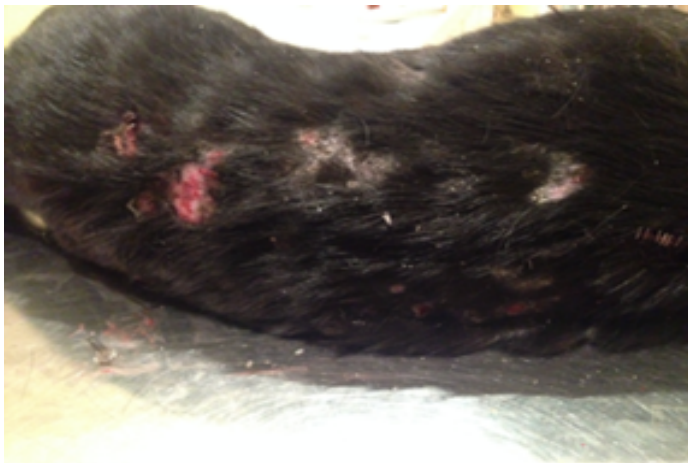

Figure 2: Alopecia, crusts and ulcers on the trunk

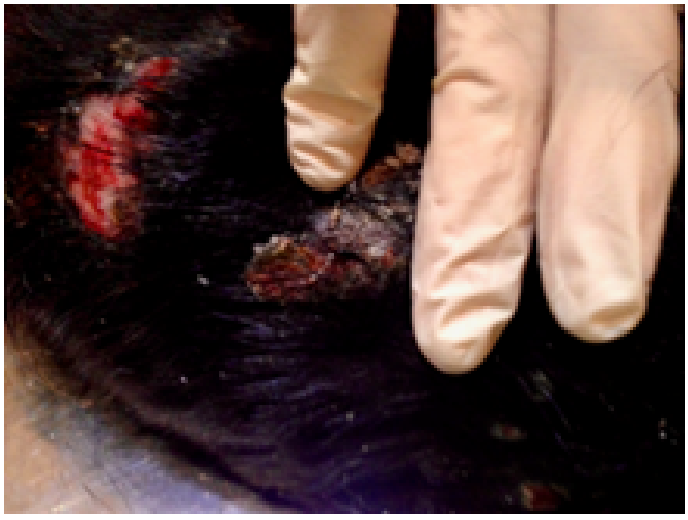

Figure 3: Detail of ulceration under the crusts

Figure 4: Strands of hair with crust easily plucked off

CBC, blood biochemistry and serum protein electrophoresis showed no abnormalities at this time.

Histological review of five skin biopsy specimens revealed a mild hyperplastic epidermis with hydropic degeneration of the basal cells (Figure 5) and the presence of few isolated apoptotic cells in the basal cell layer. Foci of dermo-epidermal splitting with the formation of suprabasilar vesicles were present in the epidermis and follicular wall. A perivascular to diffuse mononuclear infiltrate was present in the superficial dermis, more intense and diffuse immediately under the epidermis. It extended around the hair follicles to the deep dermis and was mainly integrated by lymphocytes accompanied by few plasma cells and macrophages and some eosinophils (Figure 6). All these findings have been described in SLE. There were few apoptotic cells, no atrophic follicles, no dermal pallor and no vascular lesions that could suggest an ischemic dermatopathy or vasculitis. The histopathological lesions were also no suggestive of erythema multiforme.

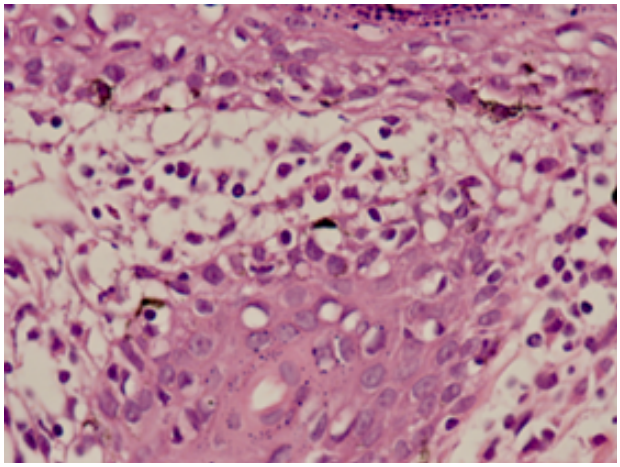

Figure 5: 40x Microscopic image of the dermal-epidermal junction showing hydropic degeneration of basal cells from the epidermis and the follicle wall

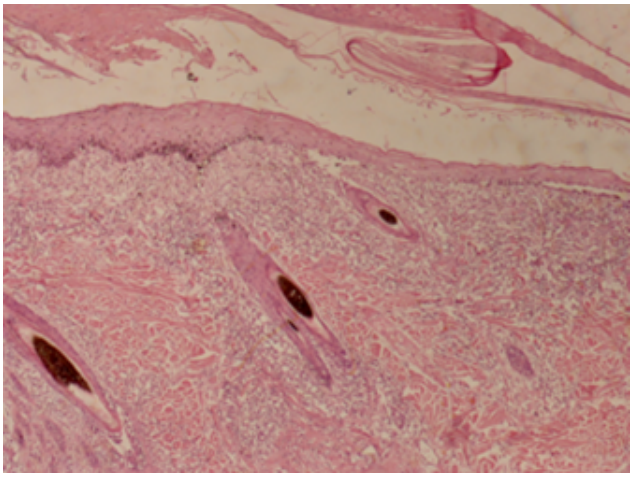

Figure 6: 10x Microscopic picture. Interface dermatitis with splitting foci of the dermal-epidermal junction. A lymphocyte-rich inflammatory infiltrates forming a continuous layer under the epidermis and extending around the hair follicles to the deep dermis is evident

A quantitative PCR (RT-qPCR; Laboklin®, Madrid, Spain) for Leishmania sp. was performed on the tissue with negative results.

Immunosuppressive treatment with prednisone (Dacortin®; ERN, Barcelona, Spain) 1 mg/kg PO twice daily was prescribed. Ten days after starting the therapy, the lesions had significantly improved. The dose of prednisone was progressively reduced over three months until it was completely withdrawn. The animal remains in complete clinical remission three years later.

Discussion

The general systemic picture, with the affection of more than three organ or systems, fever, joint pain, ulcerative dermatitis, anaemia and leukopenia was compatible with the diagnosis of systemic lupus erythematosus. ANA titer was not investigated in this case. The histopathological lesions were also consistent with SLE, as it is described in the literature [1,6]. The resolution of most clinical and clinicopathological signs, except for skin lesions, with anti-leishmania treatment, suggests in this case, a Leishmania infection as a trigger of the condition [2]. The absence of a large macrophage component and the negative RT-qPCR Leishmania rules out a direct action of the parasite as a pathogenic mechanism of skin lesions [7]. It suggests that excessive antibodies/autoantibodies production as the principal cause of the clinical signs, dermatological lesions and clinicopathological alterations in this animal. Once established, the exacerbated and aberrant humoral immune response may be self-perpetuating after eliminating the infectious agent. The administration of immunosuppressive treatment was necessary to resolve the skin lesions as in autoimmune disease (SLE) [2,3].

The absence of relapse, once the lesions were resolved, rules out the existence of a pure and permanent autoimmune disease and supports the hypothesis of the development of an SLE-like picture as a result of Leishmania infection.

The use of immunoregulators/immunosuppressants in leishmaniosis is controversial [7]. However, their use should be assessed whenever the response to anti-leishmania treatment is not adequate, and the clinical picture or lesions are related to the exacerbated and aberrant immune response, similar to an autoimmune disease [2,3].

Conclusion

The authors consider that cases of systemic lupus erythematosus-like due to leishmaniosis can be relatively frequent, although, to the best of the author´s knowledge, this is the first documented case.

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