A case of cutaneous sterile pyogranuloma/granuloma syndrome in a golden retriever

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Very little has been published on the clinical presentation of cutaneous sterile pyogranuloma/granuloma syndrome (SPGS) in the dog (1-3). We describe herein a case of SPGS in a dog with multiple cutaneous nodules, characteristic histopathological findings, and excellent response to treatment with glucocorticoids.

A six-year-old, neutered male golden retriever dog weighing 44 kg was referred to the Western College of Veterinary Medicine (WCVM) with a history of having had chronic skin problems for four months. The owner reported the appearance of skin "lumps" on the dog's back, beginning shortly after the dog had been in a boarding kennel for 10 days. Although the skin masses did not appear to bother the dog, the owner was concerned that they were spreading over the dog's back and onto the head and extremities. The dog had no history of travel. He was predominantly kept indoors and, when outside, was confined to the owner's property. There were no other pets in the household. Vaccinations were current and no previous health problems were reported.

On admission to the WCVM, the dog had a normal rectal temperature, pulse, and respiratory rate. Multiple skin papules/nodules, varying in size from approximately 2 mm to 2 cm in diameter, were palpated over the dog's entire body. Three nodules were palpated on the forehead and a few were found on the distal extremities. The majority of the masses were present on the back, lateral thorax, and abdomen. There was no pruritus, ulceration, alopecia, or discharge associated with any of the masses. They did not appear to bother the dog in any way. Some of the skin lesions had erythematous margins. Peripheral lymph nodes were not enlarged.

The dog was hospitalized. Complete blood cell count, serum chemistry profile, and urinalysis showed no significant abnormalities. Multiple skin scrapings from the masses and adjacent normal skin showed no evidence of ectoparasites. Fine needle aspirates were taken from several of the skin masses. The aspirates were mild to moderately cellular. The majority of the nucleated cells were classified as small to medium sized lymphocytes. A few large mononuclear cells were also seen. No etiological agents were identified. A tentative diagnosis of lymphocytic inflammation was made on the basis of the fine needle aspirates.

The dog was sedated with sufentanil citrate (Sufenta, Janssen Pharmaceuticals, Mississauga, Ontario), 2 μg/kg body weight (BW) IV, acepromazine maleate (Atravet, Ayerst Laboratories, Montreal, Quebec) 0.05 mg/kg BW IV and glycopyrrolate (Robinul Glycopyrrolate, A-H-Robins, Montreal, Quebec) 0.01 mg/kg BW IV, and multiple 6 mm punch biopsies were obtained. Several skin papules and nodules were removed in their entirety.

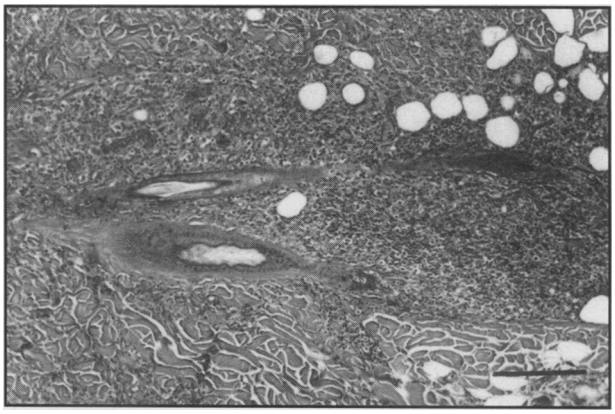

Figure 1. Photomicrograph of deep dermis showing a mixed infiltrate of macrophages, epithelioid, and lymphoid cells adjacent to and surrounding a pilosebaceous unit. Several large fat vacuoles are present. Bar = 200 μm.

The biopsies all showed a granulomatous, periadnexal or nodular to diffuse dermatitis consisting principally of epithelioid and histiocytic cells (Figure 1). Moderate numbers of lymphoid cells and a few neutrophils were also present, scattered throughout the granulomatous areas. Densely cellular, nodular masses of these cells were most common in periadnexal sites but often extended into the panniculus adiposus as well.

Special stains (periodic acid-Schiff and Grocott's methenamine silver for fungi, Fite's (acid-fast) for mycobacteria, Gram's for bacteria) and immunohistochemical stains for staphylococci and mycobacteria failed to reveal any etiological agent.

Based on the clinical presentation and histological appearance of the skin masses, a diagnosis of SPGS was made. The dog was treated with prednisone at a dosage of 2.2 mg/kg BW, q12h per os for one week. This resulted in rapid resolution of all but three small nodules on the dog's forehead. The dosage of prednisone was reduced to 2.2 mg/kg BW q24h for one week. Total resolution was apparent at that time and the biopsy sites had healed well. The dosage of prednisone was further reduced to 1.1 mg/kg BW q24h for one week. As the dog remained asymptomatic, the dosage of prednisone was tapered to alternate day therapy (2.2 mg/kg BW alternate days for one week, then 1.1 mg/kg BW alternate days for one week and, then 0.5 mg/kg BW alternate days for two weeks). No further problems were encountered.

The clinical and pathological lesions in this dog are consistent with a syndrome called SGPS or idiopathic

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periadnexal multinodular granulomatous dermatitis (1-3). This syndrome has been reported in several breeds of dogs with the collie, boxer, Great Dane, weimaraner, golden retriever (1,2), English bulldog, Doberman pinscher, and dachshund (2) being predisposed to the syndrome. Both sexes and all ages may be affected and no seasonality has been associated with the onset of the disorder (2,3). No consistent clinicopathological abnormalities have been noted in association with this syndrome (3). The lesions are usually multiple, consisting of haired, nonpainful, nonpruritic papules, nodules, or plaques, varying in size from 0.5-5 cm in diameter (1-3). These lesions can grow quickly and coalesce (3). The hair follicles can become involved resulting in alopecia, and, occasionally, the lesions become ulcerated and secondarily infected (2). The skin masses start on the head and are typically found on the head and extremities of an affected dog (1-3). This dog was somewhat unusual as the lesions began on the back and then extended to the head region, but only a few lesions were found on the head and extremities. Regional lymphadenopathy is not a common finding in SPGS (1,3).

Histologically, the SPGS lesions are characterized by multifocal, nodular to diffuse, granulomatous to pyogranulomatous dermatitis. In early lesions, a characteristic vertical orientation of oblong perifollicular granulomas or pyogranulomas is often present (1-3). Hair follicles are initially spared, as the cellular infiltrate favors a periadnexal pattern at the level of the sebaceous glands (2,3). Extension of the lesion into the subcutis and through the panniculus adiposus has been reported in more advanced cases (3), and it occurred in our case. The inflammatory infiltrate is composed of a variety of cell types with macrophages, epithelioid lymphocytes, and neutrophils present in most cases. Plasma cells and eosinophils are occasionally seen (3). Mitotic figures, evidence of thrombosis and hemorrhage, and areas of tissue necrosis have been reported (3), but they did not occur in this case.

A variety of agents including bacteria, fungi, pathogenic algae, parasites, and foreign bodies are capable of provoking a granulomatous reaction in the skin (3,4). No etiological agent has been found in dogs with SPGS despite exhaustive searches involving aerobic, anaerobic, and fungal cultures; special stains; light and electron microscopy; and immunofluorescent and peroxidase-antiperoxidase techniques (1,3).

Although clinical lesions may spontaneously resolve in some cases of SPGS, immunosuppressive dosages of corticosteroids (2.2-4.4 mg/kg BW daily) are recommended (3). Lesions generally regress within 7-14 days, and once this has occurred, alternate-day steroids may be employed (3). It has been suggested that most dogs require prolonged alternate-day glucocorticoid therapy (3). Azathioprine (Imuran, Burroughs Wellcome Inc., Kirkland, Quebec) has been recommended in refractory cases (3).

Failure to demonstrate a causative agent, absence of response to antibiotics, and favorable response to steroids suggest an immune-mediated process in this disorder (1,3). Based on clinical and histopathological findings, a type IV hypersensitivity is likely.

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