Eosinophilic Panosteitis of Young Dogs

By Dean H. Riedesel*

Eosinophilic panosteitis is a self-limiting disease affecting young dogs from three to 18 months of age. The disease was initially called hematogenic purulent osteomyelitis when it was first described in Europe in 1951. At that time the etiology was thought to be streptococci bacteria. Since 1951 the disease has been described using other names including osteomyelitis of young German Shepherd dogs, juvenile osteomyelitis, enostosis of young dogs, and eosinophilic panosteitis of young dogs. The latter name seems to be preferred by most authors.

Eosinophilic panosteitis is seen most often in young German Shepherd dogs, but it has been diagnosed in Basset Hounds, Doberman Pinschers, Airedales, Golden Retreivers, Great Danes, Labrador Retrievers, and Saint Bernards.[1]

The affected animals usually have a history of an intermittent shifting-leg lameness which cannot be associated with any previous trauma. The initial attack of lameness occurs when the dog is approximately three to seven months old. This lameness will spontaneously subside in three to 14 days without therapy. The acute lameness may recur in two weeks to three months after the initial attack. Radiographs show that there is usually more than one bone involved and the amount of pain expressed by the dog varies from none to severe. In the latter case the dog will not put weight on the affected leg and will resent deep palpation of it. A few days after the lameness appears the muscles of the affected limb atrophy. The dogs body temperature ranges from high normal to 104° F., and occasionally there will be heat and swelling of the affected limb.

The radiographic changes characteristic of this disease might be seen in any of the following long bones: humerus, radius, ulna, femur, or tibia.[3] The lesion is usually observed in the diaphyses, but the metaphyseal region occasionally shows radiographic changes. The radiographic lesions may disappear from a bone then reappear in the same bone or in another one during the course of the disease. Thus, there is a shifting leg lameness seen clinically. Early in the acute phase of this disease radiographs will show an increase in the density of the medullary canal and an increase in the cancellous trabecular

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Figure 1. A radiograph showing an increased density in the medullary cavity of a dog with eosinophilic panosteitis.

pattern. Three to five days later there will be infiltration of the medullary canal resulting in an increased density (Fig. 1). This infiltration usually begins at the nutrient foramen (Fig. 2). Two to eight weeks after the acute phase of the disease there will be migration of the densities within the medullary canal (Fig. 3). Finally two to three months after the initial signs there will be resolution of the lesion and the bone will again appear normal radiographically. Periosteal proliferation may be seen radiographically (Fig. 4) a few days after intramedullary lesions, but it also will be resolved in a few months. Researchers have shown histologically that in the area of increased density there is an increase of histocytes and plasma cells.[1]

Previous literature reports that one-half of the cases of eosinophilic panosteitis show relative and absolute eosinophilia.[1] There is an accumulation of eosinophils in the marrow of affected bones late in the course of the disease. The total white count, however, remains in the high end of the normal range. There is not a specific anemia present, but target cells and hypochromic erythrocytes are occasionally observed. The serum levels of calcium, phosphorus, and alkaline phosphatase are all within the normal range. Bone marrow smears reveal an increased number of myeloid precursors, histiocytes, and plasma cells with a definite lack of megakaryocytes.

The specific etiology of this disease is yet unknown, but *Clostridium striatum* is a common isolate. The disease has, however, been artificially transmitted to German Shepherd pups using bacteria-free and cell-free Seitz filtrates obtained from the bone marrow of spontaneously occurring cases. Nutritional causes have been excluded because all known cases have been fed adequately.

Presently there are no therapeutic regiments which affect the course of the disease. Treatment is, therefore, primarily symptomatic using analgesics and glucocorticoids in cases where there is severe pain. The duration of clinical signs is usually self-limiting being resolved in one week to eight months and most commonly in two to three months. Thus, since this is a chronic disease, steriod therapy should be used only in those dogs which show extreme pain and discomfort.

INCIDENCE

There have been three cases of eosinophilic panosteitis presented to the Stange

Memorial Clinic. One was a six month old St. Bernard in 1966; the other two were an 11 month old Basset Hound and a 12 month old German Shepherd dog in 1968.

CASE REPORT I

On December 4, 1968, a 12 month old male German Shepherd was admitted to the Stange Memorial Clinic with a history of a shifting-leg lameness. This periodic

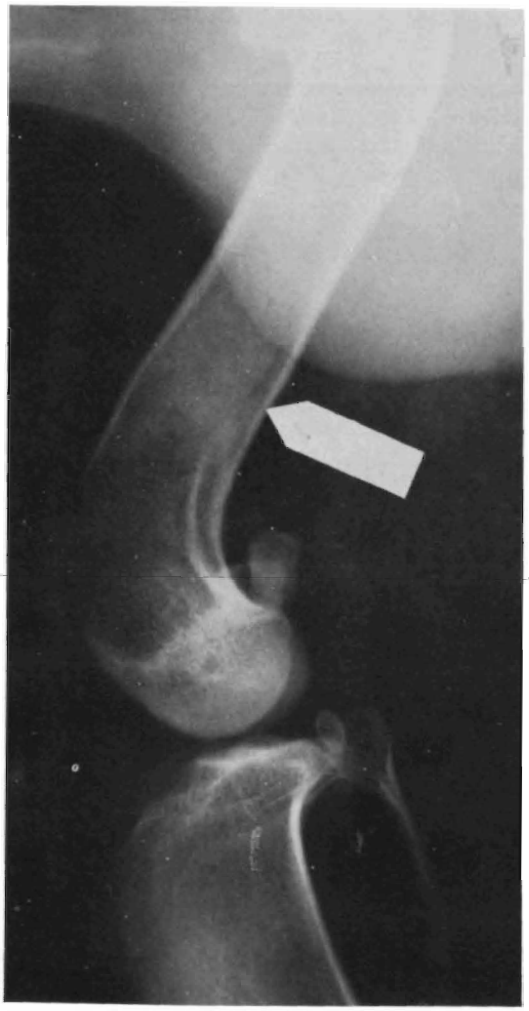

Figure 2. A radiograph of the right femur of an 11 month old Basset Hound with eosinophilic panoseitis showing an increased density n the region of the nutrient foramen.

Figure 3. A radiograph taken 2 weeks after Figure 2 showing that the lesion has migrated down the medullary cavity and is resolving in the region of the nutrient foramen.

TABLE I

	Shepherd German	Basset Hound
Blood Culture	Negative	-
Hemoglobin	16.9 gm%	19.4 gm%
Packed Cell Volume	48.0 %	55.5%
Total WBC/cmm	11,700	9,000
Eosinophils (%)	9	15
Bands (%)	0	1
Segmented (%)	59	53
Lymphocytes (%)	27	29
Monocytes (%)	5	2
Platelets	Adequate	Adequate
RBC Morphology	Normal	Normal
Total Eosinophils	277.5	-

Figure 4. A radiograph of the humerus of a 12 month old German Shepherd with eosinophilic panosteitis showing an increased density within the medullary cavity and periosteal proliferation.

lameness had been going on for the last six months, and presently the dog was lame in the right front leg. Pain could not be elicited by deep palpation of the lame leg. The dog had a temperature of 102.5° F., and the appetite was normal. Radiographs were taken of both front legs. The right humerus showed an increase in the density of the medullary cavity with some periosteal proliferation (Fig. 4 and 5). The left humerus showed an increase in the cancellous trabeculation within the medullary cavity. A hemogram was performed (Table I) and serum calcium, phosphorus, and alkaline phosphatase were determined (Table II). All of these determinations were essentially within the normal range.

From the history, clinical signs, radiographs, and blood chemistry a diagnosis of eosinophilic panosteitis was made.

CASE REPORT II

An eleven month old male Basset Hound was brought to the Stange Memorial Clinic on August 12, 1968. He had a history of persistent lameness in the right hind leg for the last 1½ weeks. The dog would not support any weight on his right hind leg, and pain was elicited while the right femur was being palpated.

Radiographs were taken which revealed an increased density within the medullary

Figure 5. A ventro-dorsal view of the same humerus shown in Figure 4.

Figure 6. A ventro-dorsal view of both hind legs of an 11 month old Basset Hound with eosinophilic panosteitis. Notice the increased density in the medullary cavity of the femur seen on the left as compared to the femur seen on the right.

cavity of the right femur (Fig. 2, 3, and 6). A blood sample was taken for a hemogram (Table I), which was essentially normal except for eosinophilia. A bone marrow smear from the affected femur showed a M:E ratio of 2.43:1 which is on the high side of normal. A diagnosis of eosinophilic panosteitis was made, and the dog was treated with corticosteroids for 4 days and then discharged. Since August the dog has had reoccurrences of lameness which is not uncommon in dogs with eosinophilic panosteitis.

SUMMARY

Eosinophilic panosteitis is a disease which affects young dogs between the ages of three and 18 months. It is most commonly observed in German Shepherds, but other breeds are also susceptible. The etiology is unknown, but the disease can be transmitted by injecting into the marrow cavity of susceptible dogs a bacteria-free and cell-free Seitz filtrate from the bone marrow of diseased dogs. The clinical signs are those of a shifting-leg lameness which are self-limiting, but they may recur in two weeks to three months. Diagnosis is made by the clinical signs, history, blood chemistry, and radiographs. Treatment is symptomatic with analgesics and corticosteroids for relief of the dog's discomfort. Two to three months after the initial attack there will be resolution of the lesion, and the bone will again appear normal radiographically.

REFERENCES

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TABLE II

	Serum Ca	Serum P	Alkaline Phosphatase
German Shepherd dog	11.5 mg%	5.0 mg%	2.52 Bodonsky Units
Normal[2]	9–11 mg%	2-5 mg%	3–6 Bodansky Units