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Canine Panosteitis (1-Jan-1985)

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Canine panosteitis is a spontaneously occurring, self-limiting disease of young, large breed dogs involving both the diaphyseal and metaphyseal areas of the tubular long bones. It is characterized by medullary fibrosis (enostosis), as well as by both endosteal and subperiosteal newbone deposition; affected animals are generally afflicted with an obscure, intermittent lameness affecting one or more limbs either simultaneously or sequentially.(14)

The disease was first described in the European veterinary literature in 1951 by Gratz(19) and Bauman(2) as a chronic osteomyelitis (or eosinophilic panosteitis) of young German shepherd dogs. First described in the United States in 1960, the disease was soon noted epidemiologically to spread from New York through New England and into the Southeast,(1) probably as a result of a newly heightened practitioner awareness and unrelated to the spread of any etiologic agent. Originally believed to be a purulent hematogenous osteomyelitis caused by a streptococcus,(1-3,7,9,22) panosteitis was initially reported to be associated with tonsillitis and episodes of fever,(1,2,9,22,25) neither of which has been substantiated in subsequent investigations. The disease has been referred to in the veterinary literature as juvenile osteomyelitis,(2) enostosis,(5) eosinophilic panosteitis,(14) and canine panosteitis.(4) It appears to have no exact counterpart in humans.

CLINICAL PRESENTATION

Canine panosteitis is a disease that affects only large or giant breeds, most often the German shepherd, although it has been reported in the basset hound, Scottish terrier, Great Dane, St. Bernard, Doberman pinscher, German shorthaired pointer, Irish setter, Airedale, golden retriever, Labrador retriever, Samoyed, and miniature schnauzer.(1,4,9,10,12,14,17) Males are affected more commonly than females(1,5,10) (reported range 67%(6) to 84%(10)). The disease cycle in the long bones of males is more predictable and repeatable. The female usually has her first episode in association with her first estrus.(20)

The average age at presentation is 5 to 12 months,(4-6,10,22,24) although German shepherds have been documented to have the disease as young as 2 months of age and as old as 5 years.(4) In one study, 20% of the animals were 18 months old at initial presentation.(4)

The initial presenting complaint is usually an acute onset of lameness persisting for 2 to 14 days(1,4,20) with no current history of trauma. The disease begins in the bones of the forelegs, with the ulna being affected most often (42%), followed by the radius (25%), humerus (14%), femur (11%), and tibia (8%). The severity of these attacks becomes reduced and the interval between successive episodes increased with advancing age.(22) The degree of lameness usually increases during the first few days of an attack, remaining unaffected by either rest or exercise.(5) Periods of lameness are often accompanied by anorexia and lethargy. There may be a spontaneous regression of signs within 3 to 4 days with or without therapy,(22,23) however, more commonly the lameness is noted to shift from one limb to another every 2 to 3 weeks,(2,9,23) with occasional lapses of one month between episodes.4 In general, the pattern is from front limb to hind limb to recur again in the forelimb.

(20) Recurrence of the disease in a previously affected bone is seemingly rare;(2,22) however, in chronic cases the repeated occurrence of lesions can be found in the radius, followed by the ulna, with fewer repeats in the humerus and femur. The length of the cycle of disease is 90 days, but in some cases it extends to 160 to 190 days.(5,24) The interval between each skeletal cycle is 160 to 180 days. In one study, 53 of 100 dogs manifested multiple bone involvement, while 49 of 100 dogs showed multiple limb involvement on initial presentation.(4) As many as seven bones in various stages of disease have been observed to be affected during an episode in one dog. Clinical signs persist on an average of 2 to 9 months,(5,24) with the disease generally disappearing when the dog reaches 18 to 20 months of age.

Physical examination is usually nonremarkable, since there is seldom an elevation in body temperature, muscle atrophy, or local hyperthermia. There is, however, pain upon firm palpation of the diaphyses of affected long bones; the pain is believed by some to be directly proportional to the amount of periosteal reaction present.(10) The degree of pain manifested clinically may range from slight to exquisite, with lameness being minimal to nonweight-bearing in severity. Since the pathologic bone formation occurs haphazardly in both time and location, multiple bones of the same limb or multiple limbs may be affected simultaneously; also, different phases of the disease may be occurring concurrently in any given animal.(4,11) Radiography is generally necessary to distinguish panosteitis from other disease conditions; however, the radiographic changes in early and late phases are subtle and far less distinct than those of mid-disease. It is generally rewarding to radiograph multiple long bones in an attempt to reveal more pronounced lesions.(4) It is important to realize, however, that generally there is no distinct relationship between the severity of radiologic changes, the amount of pain elicited on palpation, and the degree of lameness the animal manifests clinically (4,5,22)

RADIOGRAPHIC FINDINGS

One or a combination of four radiographic abnormalities may be seen in panosteitis of the long tubular bones of the appendicular skeleton. The most common abnormality is an increased intramedullary radiopacity that may or may not have well-defined margins (Fig. 49-1). The increased opacity is usually hazy, demonstrating either a granular appearance or loss of the normal trabecular bony pattern. The increased medullary opacity usually is not prominent in the area of the nutrient canal of the bone. Additional radiographic findings that may be present with or without the medullary density include an endosteal bone thickening and a periosteal reaction. The periosteal new bone is usually smooth or laminar (Fig. 49-2). The least common radiographic finding is an increased radiolucency to the medullary portion of bone; this is the earliest radiographic abnormality that may be seen.



FIG. 49-1 Medial-lateral radiographs of a humerus (A) and femur (B) demonstrate characteristic areas of increased medullary opacity.

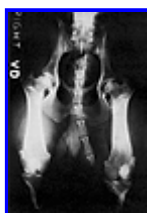


FIG. 49-2 Cranial-caudal radiograph of a femur demonstrates periosteal elevation, sometimes seen with panosteitis.

Panosteitis can often be seen concurrently with other disease processes. A differential diagnosis should include hypertrophic osteodystrophy, ununited anconeal/coronoid process, osteochondritis dissecans, hip dysplasia, trauma, osteomyelitis, nutritional secondary hyperparathyroidism, bicipital tenosynovitis, and olecranon bursitis.

ETIOLOGY

Although initially termed eosinophilic panosteitis by early authors(1,2,6,22) subsequent studies have failed to document consistent eosinophilias in affected animals during the acute phase of the disease (range 0%-50%).(1,4-6,9,10,15,20) Complete blood counts are generally within normal limits, as are levels of alkaline phosphatase, serum calcium, and serum phosphorus. Most dogs screened showed negative results when tested for intestinal parasites.

The few direct marrow and blood cultures that have been reported have uniformly been negative for aerobic and anaerobic bacteria, and histopathology has consistently failed to implicate any etiologic agent. The overall numbers of animals for whom these parameters have been published still remains low; however, such negative findings would seemingly be real in light of the usual absence of fever and normal white blood cell counts. The disease process and bony lesions have been believed by some to be indicative of an atypical viral osteomyelitis. One researcher stated that in preliminary studies, inocula had grown on bovine kidney cell culture. Other authors have purportedly accomplished artificial transmission of the disease via the intramedullary injection of both an unfiltered(10,22,24) and Seitz-filtered 10% suspension of affected bone marrow into young German shepherd dogs.(22,23) Controls were minimal, no generalized disease process reminiscent of canine panosteitis was produced, and only focal bone reactions resulted. More recently, an absence of cytopathic effect was noted when buffy coat and bone marrow inocula were taken from animals with active lesions and introduced into kidney cell lines; the same author also found an absence of second-degree intradermal delayed hypersensitivity reactions to the injection of biopsy material.(18)A genetic etiology would seem to be a factor since the German shepherd breed is disproportionately represented (86 of 100 animals in one study).(4,5,22) One investigator has reared German shepherd pups and English pointer pups together in the same pen, with only the German shepherds routinely manifesting the disease. He postulates a genetic predisposition with stress induction.(20) Other possible causes include transient vascular abnormalities,(18)allergy,(8) a metabolic phenomenon (tubular long bones of growing males),(4,8) hyperestrinism(16), parasite migration, and possibly an autoimmune reaction following viral infection.(1)

PATHOBIOLOGY

Canine panosteitis is a disease of the adipose bone marrow; it is often cyclic, and each episode is characterized by degeneration of medullary adipocytes followed by stromal cell proliferation, intramembranous ossification, removal of the medullary trabecular bone, and regeneration of the adipose bone marrow. While the etiology of the disease is unknown, stress can precipitate an episode of the disease. Radiographically, the lesion of canine panosteitis begins in the vicinity of the nutrient foramen of a long bone and is recognized by decreased medullary density (Fig. 49-3).



FIG. 49-3 The radius and ulna of both forelimbs of a 5-month-old male German shepherd. The radius on the left is in the initial stages of panosteitis while the medullary cavity of the adjacent ulna is totally affected and the secondary periosteal reaction is evident on the caudal border. The medullary cavity of the right ulna appears to be clearing while the medullary cavity of the right radius is becoming increasingly radiodense and the periosteal reaction is evident.

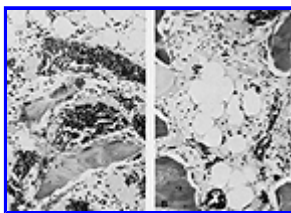


FIG. 49-4 (A) Passive hyperemia in the bone marrow of a 6-month-old male German shepherds with panosteitis. Note the accompanying osteoclasts of the trabeculae. (H&E x 316) (B) The medullary edema and capillary distension that accompany the reaction in A are thought to cause the pain associated with panosteitis. (H&E x 316)

Initially the area of the lesion is extremely sensitive to digital pressure. Histologically, there is an eosinophilic granular degeneration in the cytoplasm of the medullary adipocyte with eventual loss of the cell, leaving the latticelike stroma (Fig. 49-4). Next, there is a proliferation of the stromal and adventitial cells of the bone marrow in which intramembranous ossification occurs. Radiographically, this is recognized as a nidus and occurs 10 to 14 days after the initial clinical signs. The nidus enlarges, becoming attached secondarily to the endosteal surface and subsequently extending toward each metaphysis. The leading or peripheral edge of the nidus is composed of proliferating stromal cells and a few vessels behind which fibrous bony trabeculae are forming, while the center of the nidus is composed of lamellar bone trabeculae. The cells of the bone marrow of the nidus disappear, and the vascularity appears to be only capillaries and large venous sinuses. The ossification spreads linearly along the central medullary artery and vein with outlying bony satellites becoming incorporated into the central nidus. A periosteal reaction of linear trabeculae arranged perpendicularly to the long axis of the cortex and enclosing a vascular connective tissue appears secondary to the medullary osseous reaction (Fig. 49-5). By 20 to 30 days, the medullary diaphyseal density has reached the metaphysis; it has never been observed to extend into the epiphysis. During the osteogenesis of the medullary density, the fibrous bony trabeculae are replaced by lamellar bone trabeculae, the vascularity of the bone marrow is reestablished, and the bone marrow becomes hemopoietic. The cortical bone becomes more callus of midshaft fractures is the reaction of the injured adipose bone marrow. Research on the regeneration of bone marrow

following complete removal(13) or irradiation,(19) shows a proliferation of stromal cells, trabecular bone formation, reestablishment of the medullary vasculature resulting in a periosteal bone formation and enlarging of the osteons, and restoration of the histology of the original bone marrow type. Not only is the tissue pattern the same as in panosteitis but the chronology (90 days) is similar.

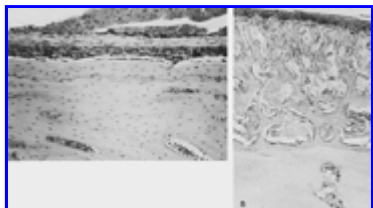


FIG. 49-5 (A) The normal periosteum of a young dog. Note the cellular or osteogenic layer adjacent to the cortical bone (H&E x 200) (B) The secondary periosteal reaction of canine panosteitis. The hyperplasia of the cellular layer of the periosteum is evident as well as the newly formed bone arranged perpendicularly to the cortex. By comparing the osteon of the cortex with one in A, its enlargement by cutting bones is readily evident. (H&E x 90) (Van Sickle DC: Panosteitis. In Selected Orthopedic Problems in the Growing Dog. American Animal Hospital Association, 1975)

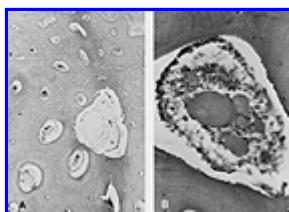


FIG. 49-6 (A) The intense osteonal remodeling of the cortex that accompanies panosteitis. Note that the outer 25% of the cortex (next to the periosteum) is fiber bone resulting from the periosteal addition while the inner 50% is a mixture of lamellar and woven bone and the deepest 25% is all lamellar bone. (H&E x115) (s) An enlargement of an osteonal canal from the cortex seen in A. The vessels are congested and form a canalicular pattern around the canal. This latter characteristic is evidence of osteocytic osteolysis occurring during the course of panosteitis.(H&E x416)(VanSickle DC: Panosteitis. In Selected Orthopedic Problems in the Growing Dog. American Animal Hospital Association, 1975)

TREATMENT

Therapeutic regimens comprising antibiotics, vitamin and mineral supplements, dietary changes, and irradiation of diseased bones and adrenal glands have all been tried with little success.(1-21) More commonly, analgesics or antiinflammatories such as salicylates or phenylbutazone remain the first line of defense against pain. When these fail, corticosteroids (prednisolone) seem to have a beneficial effect in relieving pain and decreasing lameness in a fair number of animals.

REFERENCES

1. Barrett RB, Schall WD, Lewis RE: Clinical and radiographic features of canine eosinophilic panosteitis. *Anim Hosp* 4:94, 1968
2. Baumann R, Pommer A: Die chronische Osteomyelitis der jungen Schaferhunde. *Wien Tierarztl Monatsschr* 38:670, 1951
3. Berta S, Lance E: Mit verinehrung der eosinophilen zeu einhergehende Osteomyelitis junger Hunde. *Academiae Scintorum Hungorica Acta Vet* XI:367, 1961
4. Bohning R, Suter PF, Hohn RB et al: Clinical and radiologic survey of canine panosteitis. *J Am Vet Med Assoc* 156:870, 1970
5. Burt, JK, Wilson, GP: A study of eosinophilic panosteitis (enostosis) in German Shepherd dogs. *Acta Radiol (suppl)*319: 7, 1972
6. Cotter SM, Griffiths RC, Leav I: Enostosis of young dogs. *J Am Vet Med Assoc* 153:401, 1968
7. Evers WH: Enostosis in a dog. *J Am Vet Med Assoc* 154:799, 1969
8. Gartner K: Klinische Beobachtungen an der eosinophilen Panosteitis der Junghunde. *Kleintier-Prax* 1:71, 1956
9. Gratzl E: Die eosinophile Panosteitis der Junghunde (Osteomyelitis der jungen Schaferhunde). *Wien Tierarztl Monatsschr* 38:629, 1951
10. Hardy WD, Stockman WS: Clinicopathologic conference. *J Am Vet Med Assoc* 154:1600, 1969
11. Kaastrom H, Olsson S-E, Suter PF: Panosteitis in the dog. *Acta Radiol (suppl)* 319:15, 1972
12. LaCroix JA: Diagnosis of orthopedic problems peculiar to the growing dog. *Vet Med* 65:229, 1970
13. Pratt HM, Maloney MA: Reconstitution of bone marrow in a depleted medullary cavity. In Stohlman F Jr (ed): *Hemopoietic Cellular Proliferation*, pp 5666. New York, Grune & Stratton, 1970
14. Riedesel DH: Eosinophilic panosteitis of young dogs. *Iowa St Univ Vet* 31:29, 1969
15. Schalm O: *Veterinary Hematology*, 2nd ed. Philadelphia, Lea & Febiger, 1965

16. Sprinkle TA, Krook L: Hip dysplasia, elbow dysplasia, and eosinophilic panosteitis: Three clinical manifestations of hyperestrinism in the dog. *Cornell Vet* 60:476, 1970
17. Tandy J, Haywood S: A case of panosteitis. *Vet Rec* 100:287, 1977
18. Turnier JC, Silverman S: A case study of canine panosteitis: Comparison of radiographic and radioisotopic studies. *Am J Vet Res* 39:1550, 1978
19. Van Dyke K, Harris N: Bone marrow reaction to trauma. *Blood* 34:257, 1969
20. Van Sickle D: Canine panosteitis. In *Selected Orthopedic Problems in the Growing Dog*, pp 20-28. Monograph, South Bend, American Animal Hospital Association, 1975
21. Whorton S. Eosinophilic panosteitis in the dog. *Am J Vet Clin Pathol* 2:241, 1968
22. Zeskov B: A contribution to eosinophilic panosteitis in dogs. *Zentralbl Vet Med* 7:671, 1960
23. Zeskov B: A contribution to eosinophilic panosteitis in German Shepherd dogs. *Vet Archiv* 32: 146, 1962
24. Zeskov B: Prilog eozinofilnom panositisu u. njemackih ovcara. Doctoral Thesis, Veterinarskiarhiv, Zagreb Kryiga XXXII, Svezak 4-6, 146-149, 1962
25. Zeskov B. Marzan: Kronichni osteomielitis mladih njemackih ovacava (eosinofilni panostitis mladih pasa). *Vet Archiv* 27:129, 1957

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