CHAPTER 37
OSTEOMYELITIS

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The term osteomyelitis by definition implies inflammation of the bone and its marrow contents regardless of the cause. The purpose of this chapter is to describe osteomyelitis caused by known infectious agents. The two major classifications are bacterial osteomyelitis and fungal osteomyelitis.

BACTERIAL OSTEOMYELITIS

The study of bacterial osteomyelitis includes two distinct clinical forms of the disease. Acute osteomyelitis and chronic osteomyelitis represent only one disease state, each phase of which may be seen at different time periods. Acute osteomyelitis presents the clinical picture of infection in its early stage and usually includes systemic effects. Chronic osteomyelitis, on the other hand, shows the continuation of this infection after the acute stage has passed. Exacerbation of signs can occur at any time in the course of chronic osteomyelitis and mimic acute osteomyelitis. It must be remembered that the treatment of osteomyelitis may produce remission and healing, but that exacerbations can be seen after many years; hence, healing should never be thought of as a cure.(5) Bacterial osteomyelitis may be caused by any pathogenic bacteria and occasionally by bacteria not regarded as pathogenic. The literature seems to suggest that similar organisms cause osteomyelitis in both dogs and humans. One report lists Staphylococcus as the most commonly cultured organism, followed by Streptococcus, Escherichia coli, and Proteus species.(6) Most other series do not list Streptococcus with such prominence.(29) Often cultures may contain more than one organism, and the presence of a gram-positive and gram-negative organism is quite common. Each hospital will have its own spectrum of offending organisms that should be documented to allow for proper treatment and prophylaxis.

ACUTE HEMATOGENOUS OSTEOMYELITIS

Acute hematogenous osteomyelitis, an endogenous form of the disease, is seen infrequently in the dog. It is seen most often in the neonatal dog and recognized as joint ill. The septicemia is initiated from a focus of infection of the umbilicus, which allows the infective emboli to enter the nutrient arteries of the long bones. Following the normal circulation in the young animal, these emboli are trapped in small end-arteries and capillaries that are located in the metaphysis at the level of the epiphyseal plate. In the dog these emboli then cause arterial thrombosis with resulting loss of vascularity and necrosis. Hyperemia, produced by this necrosis, leads to the establishment of infection at the level of the physeal plate. Migration of leukocytes into the area and their destruction lead to formation of purulent material, which under pressure travels in planes of least resistance. Where the joint capsule is above the epiphysis, the infectious process may sequestrate the bony cortex or lift the periosteum of the metaphysis with advancement toward the diaphyseal portion of the bone. When the joint capsule is attached to the metaphysis below the epiphyseal area, this infection can extend into the joint, yielding a septic joint (Fig. 37-1). Any skin wound or infection can lead to acute hematogenous osteomyelitis in the puppy. Caywood and co-workers(6) reported acute hematogenous osteomyelitis in four dogs over 2 years of age. In the adult dog this condition is rare; I have not seen any cases.

Classically, endogenous forms of osteomyelitis are associated with infection of the cortex and marrow with periosteal reaction resulting from lifting of the periosteum. The sequestered bone may be resorbed by osteoclastic activity, and healing (resolution of all clinical signs) may take place at any stage. Because of the inadequate resorption mechanisms associated with necrotic bone, new-bone formation can occur with incorporation of this necrosed bone in the healing callus. This incorporation of necrotic bone leads to a focus of continuing infection, the reason that osteomyelitis is such a difficult condition to treat.(5,11,30,31) Sometimes, in areas of minimal destruction, the exudate is resorbed and new bony trabeculae are formed. Sometimes this persistent abscess may be walled off by a ring of dense sclerotic bone. Infection may be reactivated at some time later or the cavity may become filled with serous fluid or connective tissue, resulting in the classic Brodie's abscess.

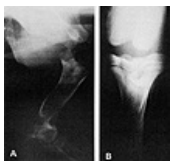

FIG. 37-1 (A) Brucella canis osteomyelitis in the shoulder of an English mastiff. The shoulder joint was completely destroyed by the infection. Multiple other joints were affected as well. (B) Radiograph shows the results of the infection in the growth plate where most hematogenous infections begin. Note the difference between the joints where the joint capsule attaches below the physis (A) and where the attachment is above the physis (B). This anatomical difference predicts the incidence of joint

infections.

The clinical signs of acute hematogenous osteomyelitis are those of fever, malaise, and disuse of the involved extremities. The puppy may present as an extremely sick animal or as an animal that is lame in one leg. The wide variation in presentation should not preclude an adequate workup and diagnosis. Radiographs may show only soft tissue enlargement at this time. Elevation in white blood cell count is common. Fluctuant swellings are common only when the purulent material has broken through the periosteum or into the joint. Multiple sites of infection are possible, and patients should be followed very closely to avoid missing concurrent areas of infection.

TREATMENT

If the animal is systemically ill and running a high fever, blood cultures may be taken. If a fluctuant area is palpated, aspiration may be used for bacterial identification and sensitivity testing. Extension of infection into the joint represents an orthopaedic emergency and should be dealt with as outlined in Chapter 86. If the fluctuant swelling is not within the joint, it should be opened, drained, and flushed to remove all of the exudative material to try to reestablish normal continuity of soft tissue surrounding bone. Antibiotics should be administered immediately after collection of material for culture, using high levels of bactericidal antibiotics for approximately 3 to 4 weeks following the cessation of signs. If treatment is instituted quickly, prognosis in the dog seems to be good. Following decompression of the wound, most wounds are closed immediately and systemic antibiotics are administered to prevent further accumulation of purulent material. Immobilization of body parts can be considered if it does not interfere with joint motion in the young puppy. It is probably not wise to immobilize joints for long periods but better to confine the entire dog to a small cage or pen. Without knowing prior bacterial sensitivities, my preference as the antibiotic of choice is one of the cephalosporins.

POSTTRAUMATIC OSTEOMYELITIS

Posttraumatic osteomyelitis usually represents a form of exogenous infection in which the bacteria are initiated through a traumatic wound or surgical incision.(5) This introduction of infection can occur after simple wounding in which the formation of hematomas under the skin may allow bacterial contamination and infection to develop. These types of infections may spread to bone, often without resultant systemic signs. Posttraumatic osteomyelitis is rather avascular, and the infection of bone, although probably developing through vascular occlusion, is localized. Infection may also be introduced by penetrating wounds, such as may be caused by a bullet, nail, or other foreign body. Bullet wounds are discussed in Chapter 36.

Posttraumatic or iatrogenic infections caused by the open treatment of closed fractures represent the most common forms of osteomyelitis seen in small animals. When the skin is devitalized to the point that bacteria contamination is possible, infection of bone is an ever present danger. Although no reliable figures are available for the veterinary literature, clinical experience would indicate that the incidence of bone infection following open fractures parallels that of humans.(5,11,2l) In acute hematogenous osteomyelitis, infection is from within (endogenous). With posttraumatic osteomyelitis, the infection comes from without (exogenous). Therefore, the course of the disease is somewhat different. In exogenous forms of osteomyelitis the clinical picture may change depending on the mode of infection and methods of treatment.

ACUTE

In posttraumatic osteomyelitis, the level, mode, and time of infection are more easily understood than in acute hematogenous osteomyelitis. If after open reduction of a closed fracture infection should intervene, the usual course of acute osteomyelitis shows an elevation in temperature and white blood cell count. The surgical area is swollen, warm, and reddened; the animal will be non-weightbearing, and the area of infection will be tender to the touch. At this point it can be assumed that an infection is present, although it may be only a deep wound infection at this time. Radiographic examination will show only soft tissue swelling.

Since rapid treatment of acute osteomyelitis most often leads to a satisfactory outcome, it is important to institute treatment immediately. This treatment consists of surgical decompression and debridement. It is important to evacuate all the hematoma, fluid, and purulent material from the wound. Culture of the fluid obtained during surgical debridement is appropriate to determine the antibiotic for continued treatment. Following surgical decompression the wound may be closed primarily using a suction drain if a large amount of dead space is present. is The animal should be placed on sufficient levels of systemic antibiotics at this time. The choice of antibiotic is related to the hospital environment and the known susceptibility of the most commonly isolated organism in that environment;(20,32) my choice is one of the cephalosporins, sometimes in combination with an aminoglycoside. If following decompression and drainage little dead space is present, there is no need to use a suction drain. If suction drains are unavailable or not desired, the wound may be packed open for a delayed primary closured Following treatment of the wound, the skin and soft tissues should be immobilized, often using a Robert Jones dressing. The gentle pressure of this type of bandage will help eliminate dead space and reapproximate soft tissues next to the bone or implant.

Antibiotics should be continued for a prolonged time even after wound healing has occurred. The exact length of time is not well documented. One human study shows the rate of recurrent infection to be markedly diminished when treatment continues for over one month following wound healing.(21) If suction drains are in place, aseptic technique must be used in their upkeep, and they are usually removed within 3 to 5 days in cases of acute osteomyelitis. The choice of antibiotics is very important, since the first 2 days of treatment may determine the clinical outcome of the patient. Drug dosage is dependent upon circulation. Higher levels of antibiotics will be needed when blood perfusion is poor or in animals in whom there may be clustered bone fragments or soft tissue edema and vascular stasis around the fracture. Following culture and sensitivity testing, antibiotics may be changed if necessary, taking into consideration both the sensitivity report and the clinical course of the patient under treatment. Radiographs may be taken at 10-day intervals to ascertain the bony reaction associated with the infection. Since the radiographs record events that happened at least 10 days previously, short-term radiographic progression of the osteomyelitis can be ignored if the patient is responding to treatments The radiographs will also help determine the status of the internal fixation device, if one has been used, and the immobility of the fracture site.

Stability of the fracture site is one of the most important means of controlling infection in bone.(24) It is extremely important to monitor the stability of the fracture and to maintain stability during treatment (Fig. 37-2).

FIG. 37-2 Radiograph of a closed fracture of the tibia (A) that was treated by open reduction and internal fixation (is), after which the animal developed a deep wound infection (C). The infection was treated as described and the osteomyelitis resolved. The implants were left in during this period and all drainage stopped. (Nunamaker DM: Management of infected fractures. Vet Clin North Am 5:259, 1975)

CHRONIC

Following inadequate treatment of acute osteomyelitis, the condition may become chronic. Chronic osteomyelitis represents an infection that is well established in bone and has been present for several weeks, months, or years. Chronic infection can present after bony union has occurred or prior to union of the fracture. If the infection occurs before bone union, the treatment of the condition is made more difficult by the presence of the non-union or delayed union. If, on the other hand, union has been established and stability achieved at the fracture site, the treatment of osteomyelitis can concentrate on the soft tissues and bone, preserving the stability that is already present. An infected non-union is the worst of all possible problems; generally, treatment is directed first to heal the fracture and then to heal the infections (Fig. 37-3). One must remember that when dealing with chronic osteomyelitis healing may allow long remissions of signs but the condition is never considered cured.

DISEASE PROCESS

Chronic osteomyelitis is a surgical disease. The incorporation of infected bony sequestrum in remodeling callus and cortex provides a nidus for continuing infection. Futhermore, the walling off of dead infected bone (infected sequestrum) makes the treatment of such cases with antibiotics alone impractical.

FIG. 37-3 Radiographs of a Doberman that sustained closed fractures of both front legs. The left radial and ulnar fracture was treated in a cast and healed uneventfully. The right radial and ulnar fracture was presumed to be a segmental fracture, and an intramedullary pin was used for fixation following open reduction. The animal was brought for treatment several months later with this open, draining nonunion (A). Following sequestrectomy and debridement of the area, a dynamic compression plate was used along with a bone graft to try to consolidate the fracture (B). Radiographs 8 weeks later showed consolidation of the radial nonunion (C) The ulna healed at a later date. It was interesting to note that the bone graft used in this case failed to be incorporated owing to the infection and lack of adequate vascularity at the time of the initial surgery. The graft underwent coagulation necrosis and was expelled from the wound through copious drainage. The implant was left in place and the fracture was considered healed al 12 weeks. Continued intermittent drainage necessitated the removal of the plate and screws (D). (Nunamaker DM: Management of infected fractures. Clin North Am 5:259, 1975)

While acute infection may be associated with good vascularity, most chronic infections are associated with avascularity and scar formation. The process of continued inflammation and occlusion of small arteries renders the bone dead and avascular. This avascular bone may be resorbed through osteoclastic activity when attached to living bone and may, in fact, be remodeled by creeping substitution over a long period of time if the dead bone remains attached. Very often these dead segments may fragment off from the major structure of bone, forming floating sequestra that are walled off with sclerotic bone, leaving this dead calcified infected structure to perpetuate the infection. The presence of a sequestrum can usually be appreciated radiographically by the appearance of a radiodense fragment of bone surrounded by an area of lysis and then a sclerotic bony margin (Fig. 37-3 A). This bony margin may form an involucrum of the entire shaft or may wall off just an individual sequestrum. The increased density of the original bone is apparent because of vascular resorption and rarefaction of living bone that surrounds it. The sequestrum itself does not become denser, but the surrounding bone becomes less dense because of its vascular supply.

The diagnosis of chronic osteomyelitis is usually not difficult. Pain, disuse atrophy, tenderness, and drainage from the area constitute the hallmarks of chronic osteomyelitis. At any stage in the natural history of chronic osteomyelitis an acute exacerbation may occur that mimics the signs of acute osteomyelitis, with systemic effects as well as local ones. Usually chronic osteomyelitis is limited to local effects, and the patient is not systemically ill.

Radiographically, osteomyelitis must be differentiated from tumors or other osteoproliferative, sclerosing, or lytic diseases. Radiographic pictures can vary with the stage of development and acuteness of the condition. In general, acute osteomyelitis shows delayed periosteal proliferative disease with little actual bone resorption. The chronic form of osteomyelitis may yield areas of sequestration with dead bone lying in a pocket of cellular debris outlined by the sclerotic border, periosteal proliferative activity, modeling of the entire cortex and endosteum, and areas of bone lysis.

TREATMENT

Treatment of chronic osteomyelitis is a combination of antibiotics and surgery.(30-32) The first step in treatment is the identification of the organisms present and determination of their susceptibility to antibiotic treatment. As soon as proper antibiotics have been chosen, a thorough surgical debridement of the area should be performed. This debridement should include excision of all avascular tissue as well as dead bone. It is wise, whenever possible, to leave an intact bone intact and not create instability via a new fracture. If the infection goes into the medullary cavity, the cavity should be opened and

debrided (Fig. 37-4). The removal of dead bone should be carried back until there is active bleeding from the cortical bone. This debridement must include all of the sequestrum. Any remaining fragments may act as a nidus for further infection, yet too vigorous removal of bone may lead to fracture and instability. This represents one of the major problems in dealing with chronic osteomyelitis. The greatest chance for the healing of the infection is through massive removal of dead bone; the greatest chance for nonunion or refracture is also related to massive removal of bone. Therefore, following debridement and sequestrectomy, stabilization of the fracture must be achieved. This can be accomplished with internal fixation such as plates and screws or with external skeletal fixation. The use of external skeletal fixation has achieved wider acceptance in treating chronic osteomyelitis with instability in humans. The use of plate and screws has been shown by Rittmann to be compatible with bone healing.(24) It appears that the stability at the fracture site is more important than the device used to achieve that status. When a plate and screws are applied to an infected fracture, the implant itself can act as a nidus for continuing infection. Thus, the goal is to heal the fracture so that the implant can be removed to help heal the infection.

Following stabilization of an unstable fracture with a plate or external skeletal fixation, a closed or open irrigation system is established. Local antibiotics are used as described in the chapter on open fractures (Chapter 36). In the dog the usual method involves the use of a single irrigation tube with intermittent instillation of antibiotics followed by continuous suction. If a large skin defect is present, suction will not be able to be maintained.

Difficulties may be encountered in hooking a mobile animal to a stationary continuous suction device. If the wound can be closed over the suction irrigation system, intermittent irrigation and continuous suction is used as described above. If the wound cannot be closed, it is usually packed open with Betadine-soaked sponges and/ or Vaseline gauze to prevent fluid accumulation within the wound. If at the time of initial exploration vascularity of the fracture site appears to be adequate, a cancerous bone graft will help fill the defect and speed consolidation of bony union. (1,16) If a cancerous graft is used, continuous suction may be applied, but irrigation with antibiotic solution is not used because it may damage the graft. If, in fact, at the time of surgical debridement the wound is still avascular, irrigation of the wound by suction drainage techniques may establish sterility and allow vascularization of the wound bed with granulation tissue formation. In this case, a delay of 10 to 14 days may be necessary, after which the wound is reexplored and debrided. At this time, a large cancerous bone graft may be used as described in Chapter 39 (bone grafting). Most wounds in the dog will epithelialize and close spontaneously. Occasionally, sliding skin grafts or full-thickness grafts may be applied to cover a defect of long standing. Skin grafting should not be contemplated until the wound appears to be free of any drainage and is covered by good granulation tissue. Muscle flaps, although not used commonly in veterinary surgery, can allow a better bed for skin grafts as well as fill large bony cavities.

Occasionally during debridement of a wound the sinus tract seems to disappear. This problem can be overcome by the instillation of 1% methylene blue solution into the sinus tract just prior to surgical exploration. This vital stain will mark the sinus tracts, usually to the level of the sequestra, so that their removal can be more easily carried out (Fig. 37-4). The procedure is initiated at the time of surgical scrub prior to debridement. Approximately 5 ml to 10 ml of 1% methylene blue solution are injected through the sinus tract using a small syringe.

The stabilization of the fracture following debridement is important. It is necessary to provide reasonable anatomical reduction of the fracture to prevent any sequela of malunion or deformities, as well as immobilizing the soft tissue so that vascularity can proceed across the fracture site. The type of stabilization used is also important. Most cases of chronic osteomyelitis represent a localized infection in a part of the bone. For this reason, intramedullary pinning is not used, since this technique has the propensity for spreading infection along the entire medullary cavity, hence the entire bone. The only two fixation methods contemplated are those of screw fixation supplemented with plates or external skeletal fixation (Fig. 37-5).

Only cancellous bone grafts should be used to fill in defects and to provide stability of unstable fractures with chronic osteomyelitis. Justification for transplanting cancellous bone into areas of infection has been shown by Matti.(16) For further discussions of bone grafting see Chapter 39.

Osteomyelitis is a very difficult disease to treat. Acute osteomyelitis is treated by immobilization of bone and soft tissues, systemic antibiotics, and wound drainage. Chronic osteomyelitis is a surgical condition that may require extensive debridement and careful attention to bone and soft tissue vascularity and fracture stability.

FIG. 37-4 This Labrador presented with a history of previous fracture of the tibia that had been treated with an intramedullary pin and cerclage wire. The fracture had healed, but several months later a draining tract was noted just above the patella on the lateral side of the thigh. The tract had been explored several times but its origin could not be found. Following radiographs (A) and culturing of the wound, the tract was explored by injection with a l% solution of methylene blue. The tract could then be followed easily to the proximal tibia and the entrance of the intramedullary pinning. The medullary cavity of the proximal tibia was thoroughly debrided and a suction drain was inserted. (B) Radiograph taken on the day the drain was removed. It had already been partially removed by the dog. (C) Photograph shows the dog with the drain in place immobilized in a Robert Jones dressing. The dog had a good result with no further drainage reported. (Nunamaker DM: Management of infected fractures. Vet Clin North Am 5:259, 1975)

When dealing with an infected nonunion, although treating both the fracture and infection simultaneously, it is necessary to first heal the fracture and then the infection. Continuous suction drainage and cancellous bone grafts are useful techniques to help establish healing. When internal fixation is used in the treatment of chronic osteomyelitis, removal of the fixation devices will be necessary before healing of the infection can be completed. Continuous drainage and recurrence of infection necessitates continued aggressive treatment to solve the problems of osteomyelitis.

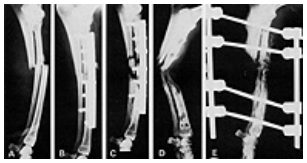

FIG. 37-5 Lateral radiographs of an open fracture of the radius and ulna (A) that was treated with a plate and screws (B). Infection and loosening of the implant necessitated several revisions of the procedure (c)+ The infected nonunion is seen about 6 months later (D). A full-frame external skeletal fixation was used in combination with multiple cancerous bone grafts to heal the fracture (E). This radiograph was taken 30 weeks after the fracture occurred. The frame was removed 53 weeks after the original injury; the result was a healed radial and ulnar fracture.

Amputation is reserved for animals for whom a reasonable hope of a functional result is lost. These cases usually exhibit loss of joint function, neural function, or soft tissue and muscle loss.

FUNGAL OSTEOMYELITIS

Depending on the geographic area in which one practices and the spectrum of patients, it may be possible to spend a lifetime in veterinary orthopaedics without encountering some of the following forms of osteomyelitis.

COCCIDIOIDOMYCOSIS

Coccidioidomycosis (Coccidioides immitis) is a regional mycotic infection that occurs in the southwest United States. The veterinary literature contains over 170 cases of coccidioidomycosis in the dog.(4,14,22) Such infections in dogs appear to be chronic in nature. A 2- to 5-month course has been reported as typical, with the infection usually beginning as a respiratory disease. A chronic cough, typically dry and nonproductive, may be the first sign. The distribution of lesions produced by coccidiodal infections involves many organs. Although the lungs and thoracic lymph nodes appear to be the sites most commonly affected, approximately 50% of the dogs will have bone involvement (Fig. 37-6). Although animals with respiratory infections may have constant elevated temperatures, dogs with bone or joint involvement may have only intermittent temperature elevations. Most bony lesions occur late in the course of infection and are characterized radiographically by increased bone density, lifting of the periosteum, and involvement of the joints themselves.

Diagnosis may not be easy. The fungus will grow readily at room temperature when cultured on Saabouraud's media. It is best to culture from granulomatous lesions. Coccidioidal sensitivity tests by way of intradermal inoculation of .5 ml of undiluted coccidioidin is positive in about 90% of the cases. Serologic tests, on the other hand, seem less helpful, with fewer than 50% of the animals being seropositive. Since this test is a complement-fixation test, part of the problem appears to be that many dogs with the disease may be anticomplementary. Biopsy procedures are useful in differentiating bony lesions from other diseases (osteosarcoma) but may also fail to prove coccidioidomycosis even after repeated attempts. Culturing bone biopsies may produce positive results. The combination of the proper environment, early respiratory disease, and long, chronic course of the disease associated with bone lesions helps make a diagnosis of exclusion when other diagnostic tests are not conclusive.

FIG. 37-6 Coccidioides immitis osteomyelitis in the proximal tibia (A) and distal humerus (B) of two dogs. A blastic response is seen in the tibia (A) and a lytic and blastic response is seen in the distal humerus (B). Differential diagnoses would include a tumor. (Brodey RS, Roszel JE, Rhodes WH et al: Disseminated coccidioidomycosis in a dog. J Am Vet Med Assoc 157:926,1970)

Treatment of coccidioidomycosis seems to be limited to use of amphotericin B. although recent experience with ketoconazole may add another drug to the armamentarium. No accurate survival rate is given in the literature. Coccidioidomycosis is found in the southwest, almost exclusively in the lower Sonorian area. Infection is acquired by inhalation of dust containing arthrospores of the fungus, which may propagate in the soil of the area. There appear to be three highly infective areas in this region. These include south Texas, south central Arizona, and the San Joaquin valley of California.

BLASTOMYCOSIS

North American blastomycosis is a chronic systemic or cutaneous infection caused by Blastomyces dermatitidis.(5) It is characterized by suppurative granulomatous lesions especially of the skin, lungs, and bone. It has been confined to the United States and Canada and appears to be one of the most important systemic fungus infections of humans and animals. The etiologic agent is identified as a large spherical thick-walled yeast cell.

It forms figure-of-eight patterns in infective tissue as the yeasts are piled on top of one another. This fungus usually enters the organism through the respiratory tract and may be disseminated throughout the body. Occasionally localized lesions may appear as papules or nodules in the skin. Treatment is usually confined to excision of local lesions or treatment of systemic disease with amphotericin B or ketoconazole. Two hydroxystilbamidines have also been used for treatment. Both have nephrotoxicity.(2) Prognosis is poor in systemic disease.(21) In humans it has been reported that the incidence of bone or joint lesions may reach 50%. A 32% incidence of bone lesions has been reported in the dog(25) (Fig. 37-7). The endemic zone for blastomycosis reaches from Wisconsin to Louisiana and across Kentucky to the Carolinas.

The radiographic features of blastomycosis are similar to those of any chronic pyogenic infection of bone.(17,23) There is usually rather marked destruction with or without surrounding sclerosis. The diagnosis relies on the direct microscopic examination of pus or other secretions. Identification is best confirmed by culture. The organisms can usually be found in bone biopsy material, and most dogs will react positively to the gel immunodiffusion test. One report shows a total of 25 bone lesions observed in nine dogs.(25) Solitary lesions were seen in six dogs, and 23 lesions involved the appendicular skeleton while only 2 lesions were found in the axial skeleton. Radiologically, most lesions in this study appeared to be

osteolytic, while a few were osteoblastic and some were mixed. Differential diagnosis includes bacterial osteomyelitis as well as malignant neoplasms.

HISTOPLASMOSIS

Histoplasmosis (Histoplasma capsulatum) is a granulomatous disease that affects the reticuloendothelial system. The organism seems to be endemic to the Ohio and Mississippi River valleys but has been recognized throughout the world. The disease appears to be caused by inhalation or ingestion of macroconidia, after which the organism spreads through the blood stream to organs with many reticuloendothelial cells. Disseminated disease may be presented Bone lesions are commonly reported in histoplasmosis in humans and have been reported in the dog (Fig. 37-8). Radiographic features of histoplasmosis in humans include subperiosteal cortical thickening, osteoporosis, and irregular widening of the medullary cavity. Cortical bone destruction and osteoblastic activity are all part of this varied picture. The infection may involve the joints. Diagnosis is made through histoplasmin tests or culture of the organism.

FIG. 37-7 Blastomyces dermatitidis osteomyelitis in the proximal tibia (A) and distal tibia (B) of two dogs. A predominantly lytic response is seen in A while that in B is predominantly biastic. (Courtesy of J. E. Cartels)

PATHOGENIC ACTINOMYCETES

Actinomycetes are organisms that are somewhat higher on the evolutionary scale than ordinary bacteria. They grow in a much branched mycelium, which may break up into fragments that cannot be distinguished from ordinary bacteria. Some of the actinomycetes are acidfast and evidently are closely related to the acid-fast bacteria. This general family is divided into two genera: actinomyces and nocardia.

ACTINOMYCOSIS

Actinomycosis boas is present in the mouth and gastrointestinal tract in many normal animals. (10,13,28) It usually represents no problems except when a penetrating wound allows the organism to invade local tissues. The actinomycosis organism usually invades the jaw but has been reported in the scapula and other bones. The lesion is characterized most frequently by bone destruction without new bone formation. As the osteomyelitis progresses, bony reaction may be a prominent sign of infection. Clinically, a thick, mucoid, tenacious, greenish yellow, nonodorous pus is characteristic of the disease. Three to four-millimeter "sulfur granules" are found in these draining lesions and are diagnostic of the condition. If the granules are examined freshly after crushing, a ray like fungus can easily be discerned microscopically. Treatment is usually through chemical means with iodine solution and systemically with antibiotics such as streptomycin.

NOCARDIOSIS

Nocardiosis is not an uncommon disease in dogs and cats.(7,28) Although serologic tests can be run to determine strain identities, the generic name is usually used alone. The organisms are soil saprophytes. Pathogenesis of the organism is assumed to be through lung infection with microabscess formation and hematogenous spread from the lungs to other organ systems. Vertebral osteomyelitis has been reported as well as lesions of the hock. Lesions are usually those of chronic suppurative osteomyelitis. The radiographic picture is similar to that of other forms of chronic suppurative osteomyelitis (Fig. 37-9). Two patterns of disease are noted with nocardia: introduction of the organism from the soil into tissue by local invasion may result in a large tumorlike mass on the extremities characterized by many numerous sinuses. The pulmonary form or gastrointestinal forms are caused by ingestion or inhalation of the organism and are the forms seen most commonly. Diagnosis is made by culture or blood agar plates and direct Gram stain of pus from an abscess or draining sinus. Successful treatment of the disease has been reported with large doses of penicillin and streptomycin. Sulfadiazine is also used in the treatment over a prolonged 6- to 12-week period. Surgery may be indicated to remove large masses associated with the disease. According to the literature, recovery may be anticipated.

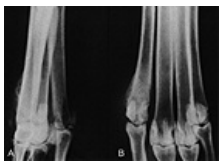

FIG. 37-8 Histoplasma capsulatum osteomyelitis in a metatarsal bone in a dog. Lateral (A) and cranial-caudal (B) radiographic projections demonstrate periosteal new bone formation at the distal aspect of the fifth metatarsal. (Lau RE, Kim SN, Pirozok RP Histoplasma capsulatum infection in a metatarsal of a dog. J Amvet MedAssoc 172:1414, 1978)

FIG. 37-9 A 6-month-old Shetland sheep dog was admitted to the clinic at Auburn University because of self-mutilation of the skin over the carpus bilaterally. Biopsy confirmed the diagnosis Of nocardiosis. (Courtesy of J. E. Hartels)

CRYPTOCOCCOSIS

Cryptococcus neoformans represents another mycosis that affects internal organs of animals.(3) The main entrance of infection is the respiratory tract, resulting in sinusitis and nasopharyngeal and pulmonary granulomas. Extension of the lesion from the respiratory system into the nervous system is not uncommon. Musculoskeletal lesions are also common, and skeletal lesions are seen in the shaft and metaphysis of long bones (Fig. 37-10). Approximately 25% of the cases in the literature indicate some involvement of the musculoskeletal system as the predominant sign.(26) Most commonly the respiratory signs are evident, with the neural and ocular systems presenting in decreasing order of frequency.

FIG. 37-10 This pointer was examined for lameness at Auburn University. The animal had been lame for about 6 weeks. The diagnosis is not particularly evident radiographically, although a biopsy showed cryptococcosis. (Courtesy of J. E Bartels)

ASPERGILLOSIS

Aspergillus fumigatus infection in the dog is not uncommon. Most infections seem to be associated with the nasal passages and result in chronic nasal discharge. Radiographically, nasofungal infections may show loss of turbinate bone detail with large radiolucent spaces as well as areas of increased radiopacity. Differentiation must be made between fungal infections of the nose and intranasal tumors.(9) The prognosis for dogs that have intranasal tumors is very poor. The nonneoplastic nasal diseases are generally more responsive to treatment, which usually consists of surgical removal of the lesion as well as systemic treatment with antifungal agents such as nystatin or amphotericin B.(I2) Radiographic and physical examination can be used to help differentiate between intranasal tumor and fungal disease. Dogs that have nasal tumors show more extensive nasal and sinus radiographic abnormalities than dogs that have benign disease. Erosion of the vomer bone and presence of an external mass are highly suggestive of neoplasia.(9) If both of these radiographic features are absent, the presence of a tumor is unlikely. Areas of increased radiolucency in the nasal cavity are suggestive of nasal fungal infection, particularly if no erosions of vomer bone are present.

OTHER INFECTIONS

Other mycotic infections have been reported in the dog and cat. The general description of these conditions is similar to the above reported mycotic infections affecting internal organs of animal species. Among those described are paecilomycosis, cephalosporium, and adiaspiromycosis.

Public health aspects of any systemic mycosis should be discussed with all clients before instituting treatment. Since the results of treatment for many of the systemic mycoses are not well documented in the veterinary literature, the prognosis for treatment are difficult to substantiate. When advice is necessary, consultation with the infectious disease units of large regional hospitals or the Center for Disease Control in Atlanta, Georgia may be valuable.

NONINFECTIOUS OSTEOMYLEITIS

From the very beginning of metal implantation into the bodies of humans and animals, corrosion of all types has been a problem. Progress in metallurgy has successfully given us high-quality alloys for use as implants. Still, a proportion of patients may react to the implants. These reactions may be in response to an allergy between the patient and the implant or may be a direct response of the metal in the tissues. Certainly the use of dissimilar metals as employed in the Jones splint has been shown to cause frank corrosion and drainage (Fig. 37-11). When drainage occurs in these instances, the wound is initially sterile but may be susceptible to a retrograde infection of the draining tract. In general, drainage associated with the metal implant is a late complication of the internal fixation and may occur months or years after implantation. Removal of all implants usually resolves the problem even when secondary bacterial infection occurs. Antibiotic treatment is usually used following antibiotic sensitivity testing. When using high-quality implants, most corrosion products are formed at articulating surfaces of the device such as the screw-plate interface. A more complete discussion of the material aspects is covered in Chapter 13.

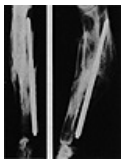

FIG. 37-11 Metalosis was the initiating cause of this secondary bacterial osteomyelitis. Removal of this subcutaneous device allowed healing of the open draining wounds.

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