OSTEOMYELITIS IN REPTILES

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Abstract: Osteomyelitis is a frequently recognized syndrome in reptiles. There are many contributing factors, and treatment in most cases will vary based on the inciting cause, areas affected, organisms involved, and patient and owner compliance. Rapid diagnosis and aggressive treatment are essential to affect a cure.

INTRODUCTION

Osteomyelitis is defined as a localized or generalized inflammation of bone due to pyogenic infection. Sequela of osteomyelitis include bone destruction, stiffening of joints, pathologic fractures, septicemia, and death. In humans, most cases of osteomyelitis are treated with intravenous antimicrobials via an indwelling catheter for a minimum of 4 wk, in addition to aggressive surgery, and still there is a high incidence of therapeutic failure. In reptiles, osteomyelitis is a commonly diagnosed disease, yet is often treated empirically and inadequately, leading to antimicrobial resistance and dissemination of disease which may be misinterpreted as therapeutic failure.

ROUTES OF INFECTION

Primary infectious processes in reptiles are uncommon. Introduction of pathogenic organisms into bone may be exogenous or endogenous. Most cases of microbial infection, whether involving bone or other areas, result from immunocompromise. Environmental factors such as temperature, cage, substrate, crowding, humidity, diet and nutrition, and stress all may contribute to immunosuppression and subsequent colonization. Trauma is a common cause of exogenous immunocompromise, which leads to introduction of pathogens to tissues and vascular dissemination. Injury from cage mates, improper substrate, parasitic infestation, improper shedding, or cage screens or glass are all potential sources of traumatic injury. In turtles, shell trauma provides a direct introduction of pathogenic organisms into bone; however, osteomyelitis in this region is surprisingly rare.

Digit and foot abscesses are common in reptiles, and concurrent osteomyelitis is often present in more than one digit. Obesity may play a role in some species, such as monitor lizards. In others, abrasions caused by rough substrate, nail avulsions, or even constricting lesions may be the inciting agent. If recurrent disease occurs in this area after appropriate therapy and environmental changes, then further investigation is warranted.

Bacterial disease may be present elsewhere in the body, serving as a nidus for recurrence and dissemination. Coelomic granulomas may be present and asymptomatic, but may lead to recurrent infection in areas of increased capillary density, such as digits and joints. Salpingitis or enteritis have been associated with pathogenic dissemination to other areas, particularly to the caudal vertebrae.
COMMON PATHOGENS

Aerobic Bacteria

Most microbial organisms involved in infectious processes in reptiles are normal flora which become opportunistic pathogens. Many gram-negative organisms are normal flora in reptiles. One study of normal bacterial populations in reptiles found *Salmonella* spp., *Salmonella arizona*, *Proteus*, *Pseudomonas*, *Citrobacter*, *E. coli*, and *Staphylococcus* spp. to be the most common isolates. Other isolates included *Klebsiella*, *Serratia*, *Edwardsiella*, *Aeromonas*, and *Chromobacterium*. Organisms found in diseased reptiles evaluated concurrently in most cases were also present in healthy animals in the same collection.

*Salmonella* spp. have been identified in over 90% of reptile populations. Many of these organisms represent normal flora in the animal, creating difficulty in interpretation of culture results. Isolation of *Salmonella* spp. from a site of infection is likely to represent pathogenicity, and appropriate treatment should be initiated; however, isolation of *Salmonella* from a remote site or fecal culture may not correlate with disease and must be interpreted with caution.

*Pseudomonas* spp. are considered normal flora for most reptiles, but certainly behave as opportunistic pathogens. Again, results must be interpreted based on clinical presentation and site of culture. *E. coli* has been implicated in osteomyelitis in reptiles, particularly snakes. Ascending infection from the female reproductive tract may be the initial source.

Isolation of any bacteria from a sterile biopsy of bone should be considered pathogenic, and appropriate therapy is warranted. However, failure to isolate organisms on culture does not necessarily equate to absence of microbes. Histology of bone is essential in identifying bacterial or fungal involvement, and may be the most conclusive diagnostic test.

Anaerobic Bacteria

Anaerobic bacteria are frequently overlooked as a source of infection and osteomyelitis. Many culture media require enrichment or special collection to promote the growth of anaerobic organisms. Isolation of any anaerobic organism should be considered pathogenic, but failure to grow anaerobes on culture does not eliminate the possibility that anaerobes are playing an active role in infection. One study has suggested that anaerobic organisms may be present in 50% of cultured samples.

Mycotic Agents

Fungal pathogens are uncommon infectious agents in reptiles. Infection usually is a sequela of immunosuppression or trauma, and usually results in formation of granulomas rather than systemic disease. However, in resistant cases of osteomyelitis, fungal cultures may provide critical information.

Mycobacteria

Mycobacteriosis in reptiles is rare, and usually exists as cutaneous disease. Osteomyelitis due to mycobacteria is unlikely in many species, but can be readily diagnosed with acid fast staining techniques.
DIAGNOSIS

Osteomyelitis is most often suspected based on radiographic changes; however, it is not appropriate to diagnose solely based on radiograph. Supporting evidence, such as obvious trauma, or histopathologic or bacteriologic confirmation, is required. Several disease processes may have similar radiographic presentations, including neoplasia and Paget's disease. Osteomyelitis may also exist as a sequela to neoplasia.

Radiographic changes associated with osteomyelitis in reptiles are unlike mammalian lesions. In reptiles, osteomyelitis causes lytic lesions, which may persist for a prolonged period after the organisms have been cleared. The periosteal reaction typically present in mammals is absent or less recognizable in reptiles. Osteolytic areas without evidence of new bone formation is characteristic of osteomyelitis in reptiles, but resembles the appearance of neoplasia in mammalian bone and may be misinterpreted by practitioners unfamiliar with this unique feature.

Bony proliferation along the spine of snakes, often with fusion of vertebrae and skeletal deformity, is seen periodically. Lesions are similar to Paget's disease in humans. In some cases, an infectious agent has been identified, but it remains unclear whether it was a primary or secondary pathogen. Other etiologies, including trauma, parasitic migration, and immune-mediated etiologies have been proposed.

Histology of osteomyelitis will demonstrate the presence of organisms. Special stains may be required to demonstrate fungal or mycobacterial involvement. The typical inflammatory response in reptiles is either heterophilic (granulomas forming around central masses of necrotic heterophils) or histiocytic (induced by intracellular bacteria, leading to granulomatous accumulation surrounding macrophage aggregates). Affected bone is typically osteolytic or osteoporotic.

Hematologic parameters may support osteomyelitis. Heterophils are responsible for phagocytosis of organisms, and a systemic heterophilia indicates an active inflammatory response, particularly microbial infection. Toxic heterophils may also be identified, and are most commonly associated with bacterial disease. Monocytosis, or specifically azurophilia, may also indicate infectious disease.

SAMPLE COLLECTION

Samples for culture and sensitivity should be collected aseptically from affected areas of bone. Premoistened culturettes are rarely acceptable for isolation of pathogens involved in osteomyelitis. Surgical debridement and curettage is often required to obtain a representative sample of a bony lesion, and may have therapeutic benefits. Bone biopsy may also be obtained with the use of a Jamshidi biopsy instrument, or, in severely affected areas, a core bone biopsy may be obtained with a 16 ga or 18 ga needle. Samples should be collected aseptically and placed in appropriate media for culture. Sterile saline is appropriate media for aerobic cultures which will be plated within 24 hr; if longer shipment is anticipated, transport media is required to sustain microbial growth. Thioglycolate may be added to support anaerobic growth. It may be necessary to request specific transport media from a laboratory prior to sample collection to enhance diagnostic value.
TREATMENT

Effective treatment of osteomyelitis is dependent on accurate diagnosis, aggressive debridement, appropriate antimicrobial selection, and effective delivery of therapy to the affected site. Because the inflammatory process in reptiles is granulomatous, in many cases, the antimicrobial may be effectively prevented from reaching the organism without surgical intervention. Aggressive debridement is required. If an encapsulated lesion is present, an attempt should be made to remove the lesion in toto, with the capsule intact. This may require removal of large sections of bone. In severely affected digits, amputation may be required to prevent further infection. In other bony lesions, curettage and lavage of all affected bone is important in eliminating foci of infection.

For mild cases of osteomyelitis, aggressive antimicrobial therapy may be attempted. Therapy should be initiated upon collection of samples, while pending culture results. Gram stains are valuable to identify gram negative or positive organisms, and aid in choosing interim therapy. Antimicrobials with good penetration of bone, such as cephalosporins, extended-spectrum penicillins, flouroquinolones, or aminoglycosides, should be chosen until sensitivities are available. Intravenous or intraosseous administration of the first doses may aid in establishing therapeutic concentrations earlier in the course of treatment, and then intramuscular administration should be continued for a minimum of four weeks. Ideally, a second sample should be obtained for repeat culture after 4 wk, and at 2-week intervals subsequently, with continuation of therapy for 1-2 wk after elimination of bacteria, although this is not always practical in a clinical setting. Oral therapy is discouraged when treating osteomyelitis.

LITERATURE CITED