An Overview of Osteomyelitis: Part I

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This is the first in a two-part series of articles dealing with orthopedic infections, and will examine osteomyelitis as a clinical condition from its initial development through to its clinical resolution.

Osteomyelitis is a term describing an inflammatory condition which involves the periosteum, cortex, and/or medullary cavity. While fungal, viral and parasitic invasion of bone are known, it is bacterial contamination and overgrowth that most commonly results in osteomyelitis.[1,3] Regardless of the particular pathogen involved, bone typically becomes infected by one of two primary routes: hematogenous or exogenous.[1,3]

Hematogenous Sources of Osseous Infection

This clinical situation occurs only rarely in veterinary medicine, but is a fairly common entity in human medicine. When seen in veterinary cases, it typically involves younger animals and reflects the juvenile orthopedic nutritive vascular pattern. Hematogenous osteomyelitis in the immature animal forms almost exclusively in the metaphyseal area. This correlates well with the sharp turns the nutrient vessels undergo as they approach this bony compartment. Bacteria coursing in these vessels often become lodged near the metaphysis and induce an inflammatory response with subsequent formation of a focus of microthrombi. An area of avascular necrosis is thus initiated and a fine media for microorganism growth and multiplication may thus come to fruition.

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Exogenous Sources of Osseous Infection

Osteomyelitis may occur as a sequellae to contamination of open fractures, open surgical repair of closed fractures, as well as spread from associated soft tissue infections. The majority of infections to bone tissue in veterinary medicine occurs via exogenous routes. Open reduction and fixation of closed fractures has been reported to account for over half of all encountered cases of canine osteomyelitis.[2]

Although the pathogenesis of osteomyelitis is well described,[1,3] the simple presence of a contaminating or invading pathogen does not always dictate the subsequent development of clinical osteomyelitis. This reflects a well-recognized array of host defense systems at the cellular and subcellular level. The inflammatory reaction induced by the presence of bacteria results in the invasion of the area by polymorphonuclear cells, monocytes, antibodies, and complement. The above response by bony tissue is not unlike that seen in inflammatory conditions of other tissue systems. Both defensive cell and bacterial death ensues with release of proteolytic enzymes. Associated tissue necrosis and accumulation of exudate occurs. One of several possible clinical outcomes may occur: the body may completely clear the pathogen, the body may wall off the focus of infection early in its development with a resulting chronic sterile bone abscess, or the natural defense mechanisms will be overwhelmed by the pathogens and the area of infection will grow as exudate and debris further accumulate. If the last of the possible clinical outcomes develops, then invasion of nearby cortical bone will occur via the Haversian and Volkman's canal systems.

Sequestrae and involucrae typically form in the young patient whereas bone destruction, periostealy new bone formation and development of draining sinus tracts are seen in the mature

patient.[3] The above dichotomy is thought to reflect the loose periosteal attachment and significant subperiosteal potential space for invasion by the spreading pathogens in the young patient, in contrast to the tightly adhered periosteum seen in the adult.[3] The above series of events may result in impeded or full-blown loss of blood supply to the affected bone. This creates an environment which is very condusive to further bacterial proliferation and is likewise difficult for antibiotics, as well as the patient's natural defense systems, to penetrate.

This ischemic condition may further accentuate blood loss which may have been caused by the initial fracture-inducing trauma or that associated with the corrective surgical approach and application of any fixation appliances. Further consideration of stress-induced immunosuppression or that caused by administration of corticosteroids to treat shock may further compromise the orthopedic patient's ability to fight off infection. With these facts in mind, it may be somewhat surprising that more patients do not develop osteomyelitis as a result of fracture-induced trauma.

In conclusion, regardless of the means of their introduction to bony tissue, when the balance between invading microorganisms and the body's defense mechanism swings in favor of the pathogen, clinical osteomyelitis will occur. In the next segment in this series, the biological character, clinical signs, diagnosis, and treatment of this condition will be examined.

References

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