Infection of Bones and Joints

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The route of a joint or bone infection has a major impact on the onset and type of clinical signs that are seen. Therefore, the determination of the route of infection is important in initiating the proper course of therapy. Author’s address: Rood and Riddle Equine Hospital, 2150 Georgetown Rd., P.O. Box 12070, Lexington, KY 40580. © 1998 AAEP.

1. Introduction

Infection of a bone or joint implies that one or more species of bacteria have colonized the structure involved and are proliferating, causing a pathologic condition because of their presence. In the past, these conditions were termed osteomyelitis or infectious arthritis, and the treatment was not significantly different from case to case.1 The successful management of orthopedic infection necessitates distinguishing between the different anatomic locations and structures involved and adapting the most appropriate treatment methods. Therefore, categorization of orthopedic infections with regard to their mode of infection is helpful in determining prognosis and treatment.

Orthopedic infections may originate from an endogenous or exogenous route. Endogenous infections are usually hematogenous, although occasionally septic lymphangitis can result in an infection of bones or joints.2 In exogenous infections, bacteria gain access to the affected site by external inoculation such as by direct trauma, injection, or surgical intervention.

How infection is introduced can also influence the local blood supply, which, therefore, has a significant impact on the approach to treatment. With time, all orthopedic infections result in tissue destruction and loss of blood supply in the more central portions of the affected area, with hypervascularity occurring around the periphery. When possible, treatment is aimed at interrupting the process prior to local tissue destruction or abscess formation, and a careful assessment of the route of infection at the initial examination is helpful in approaching treatment.

Hematogenous orthopedic infections are most common in the neonate in which the immune system is not fully developed. The most commonly involved sites are those at which bone growth is most active, such as the metaphyseal side of the physis. The artery enters and perfuses this area, and the venous return exits at 180° to retrace the same path as the arterial circulation.3 This hairpin-loop capillary bed facilitates bacterial attachment, multiplication, and thrombosis of the vessels.

Septicemia is common in neonates and may result from gastrointestinal absorption or infection of the umbilicus. Circulating bacteria have access to all tissues, but bone and joint tissues are particularly vulnerable because of their vascular anatomy. Hematogenous orthopedic infections are rare in adults because the vascular anatomy changes with skeletal maturity. The site of hematogenous localization is often on the metaphyseal side of the physis, which has abundant capillary loops to support bone growth (Fig. 1). A similar vascular organization exists in the subchondral bone of the articular cartilage and the underlying growth cartilage of the epiphysis. An epiphyseal infection can often lead to...
2. Clinical Signs of Hematogenous Infections

The prodromal signs of a hematogenous orthopedic infection are often minimal. Affected foals rarely show the severe lameness associated with acute infectious arthritis that is seen in adult horses. There is often a vague history of lameness and an inability of the foal to get comfortable, as indicated by the continual movement of a limb or limbs during periods of recumbency. The initial localizing signs are often minimal or absent and often manifest as mild edema or a local increase in heat.

Initially there are often no abnormal radiographic changes, but changes may appear within 4–5 days and, in some instances, as rapidly as 1–2 days after clinical signs appear, because the infection may have been present for days prior to the appearance of clinical signs. Therefore, a radiographic assessment of any swelling associated with a bone or joint is important to help establish a baseline and ultimately determine the progression of changes. Hematogenous infections in the diaphyses of bones are rare because of the differences in vascularity in that part of the bone.

The source of the infection should always be investigated in foals. Septicemia may result from pneumonia or another systemic disease. Another common source is the umbilicus, because the urachal stump and umbilical vessels often become infected. The physical examination of a lame foal must include these areas; in addition, the limbs should be palpated for areas of increased heat, which may be an early sign of infection.

Synovial effusion is an important clinical feature if the infection enters a joint. Therefore, synovial fluid cytology and culture are important. An elevation of synovial fluid protein may precede an elevation in white blood cell count, because initially the bacteria are located in the bone or joint capsule and have not entered the synovial cavity. Occasionally, diagnostic local anesthesia may help localize the site of the infection. However, intra-articular anesthesia in the face of severe inflammation may not be effective, presumably as a result of the low pH.

Systemic signs generally include mild fever and leukocytosis because of the presence of bacteria in the circulatory system. The foal is usually lethargic and all signs are proportional to the extent of the infection. The identification and elimination of the infection at its origin are essential to successful treatment.

3. Clinical Signs of Posttraumatic Infections

The extension of an infection into a joint from another site, such as an adjacent cellulitis, may initially manifest as a periarticular infection followed later by signs of infectious arthritis. However, some periarticular infections are difficult to differentiate from intra-articular infections. Joint infections resulting from direct inoculation follow an opposite course. Inoculation initiates an intra-articular infection in which synovial effusion is the first sign; it is present even before periarticular edema begins. Infection is usually the result of a traumatic or surgical wound or an intra-articular injection. A surgical wound infection may result by extension from a suture, but it may also result from intraoperative contamination. The injection of some medications into the joint has been shown to reduce the dose of bacteria necessary to create an intra-articular infection. Therefore, when signs of pain, heat, or swelling result from an intra-articular injection, a joint infection should be considered.

Signs of infectious arthritis advance rapidly once bacteria start to multiply in the joint. Infectious arthritis is one of the few clinical conditions that will cause grade 5/5 lameness in an adult horse. Foals less frequently show severe lameness, at least in the early stages of disease. In adults, a physical examination alone will usually identify the affected joint. Diagnostic local anesthesia is not usually necessary.
and it is sometimes difficult to induce analgesia unless local anesthesia is completed on nerves that are some distance from the affected joint. The systemic white blood cell count will usually be normal in the early stages of posttraumatic infectious arthritis; however, the synovial fluid evaluation is usually diagnostic. The initial rise in synovial fluid protein levels is followed by a rapid increase in the intra-articular white blood cell count, until the white blood cell count exceeds 50,000 cells/µl and often exceeds 100,000 cells/µl. These counts are often present within 24 h of the onset of clinical signs. If the infection is a result of an intra-articular injection, medication may accelerate or retard the clinical signs associated with infectious arthritis. A polysulfated glycosaminoglycan product (Adequan®) has been shown to promote the development of infectious arthritis, and corticosteroids have been shown to delay the development of clinical signs. However, after this delay in the onset of clinical signs with corticosteroids, the progression of the infection is the same for all other joint infections. Bacteria begin to proliferate rapidly once established.

The time frame of the infection after direct articular injection will be influenced by the medication injected, but the infection usually appears before the second day postinjection. With the injection of corticosteroids, however, the onset may be delayed up to 1 week or more. The most common time for clinical signs to appear after surgery is 3–4 days postoperatively if the infection was introduced at the time of surgery. Signs will develop later if the infection progresses into the depths of the wound as a result of a wound or suture infection. Signs of joint infection after a traumatic wound do not occur until the joint reforms a closed cavity. As long as the synovial space is open and draining, fluid distention does not occur and inflammatory mediators do not accumulate. Although the joint may be contaminated, it will not form an abscess until the joint cavity closes. Therefore, clinical signs are often delayed for some period of time after a traumatic joint wound is incurred.

4. Clinical Signs with Traumatic Infections of Bone

Traumatic infections of bone occur as a result of the direct bacterial inoculation of the bone. Bacterial contamination of bone will not result in an infection unless the blood supply is disrupted by trauma or thrombosis (Fig. 2). The blood supply to bone is vulnerable to direct surface trauma or fracture, particularly the bones of the distal aspect of the limb (such as the cannon bones). Direct trauma destroys the periosteum, which exposes the surface of the bone to the environment, which thereby results in thrombosis of the vessels of the affected bone. The circulation of the bone comes from the interior of the bone and exits at the surface. Wounded bone is usually superficial, although with serious trauma (such as with the creation of excessive heat by friction), the loss of blood supply and bacterial invasion may occur to deeper levels.

The disruption of the blood supply by exudate seldom occurs from the exterior if the periosteum remains intact. If the periosteum remains intact, external infections such as cellulitis and lymphangitis rarely extend into the bone. Hematogenous bacterial infections that originate within the medullary cavity have greater access to the arterial circulation, and because of associated increases in medullary pressure, they can progress more readily into the vascular supply. When normal medullary cavity circulatory pressures are exceeded by exudate pressure, bacteria progressively invade the vascular canals of the bone.

The most devastating circulation loss occurs with an open fracture in which the vasculature of the fracture ends becomes thrombosed and the bone penetrates the skin, resulting in gross contamination. Some exposed bone sites are more vulnerable to infection than others. These include bone fragments that are totally separated from parent bone and its attendant blood supply, as well as the proximal end of the distal fragment of parent bone. If no bacterial inoculation occurs when the blood supply is interrupted, then normal revascularization can occur once the fragment becomes stable. However, once bacterial invasion is severe, it is difficult for the blood supply to re-establish and eradicate the infection. Stability is the primary aid in allowing the re-establishment of the local blood supply. If a bacterial infection becomes well established, significant inflammation develops around the avascular infected bone. This results in a decrease in local pH and calcium resorption, and the avascular piece of bone begins to separate from the surrounding vascularized bone. This creates the clinical entity referred to as a sequestrum, which is a dead piece of bone surrounded by inflammatory tissue and exudate (Fig. 3).

A granulomatous reaction develops around the sequestrum with a local accumulation of white blood cells and the mucinous glycoalyx that forms on the surfaces of dead bone. Peripherally, mononuclear
cells and fibrous tissue accumulate in an attempt to further isolate the sequestrum. With a sequestrum, exudate continues to accumulate until it periodically ruptures through the fibrous capsule. Since in bone the exterior of this local granuloma is of osseous origin, the granuloma mineralizes, forming an osseous exterior to the granuloma. When pressure builds within this cavity, pain and lameness result. When periodic rupture and drainage occurs, lameness subsides. The draining tract is recognizable radiographically in the chronic stages as the cloaca of the involucrum (Fig. 4). These structures result from repeated episodes of pressure, rupture, and drainage.

Sometimes the proliferation of tissue in the area of a sequestrum will sufficiently stabilize the bone fragment so that revascularization can occur. In other instances, small sequestra are simply decalcified by the severe inflammatory process associated with the reduced pH at the area of the inflammation. However, neither of these processes is dependable or predictable, and surgical removal of the infected fragment is the preferred treatment.

If a bone fracture is unstable, then progressive destruction of the blood supply and progression of the infection more deeply into the bone occur. With the progressive loss of blood supply, bacteria progress into the bone via the normal vascular canals of the cortical bone. Treatment strategies in this situation aim to stabilize the bone so that the normal blood supply can help protect the bone from the infection.

An infected bone implant creates the same avascular situation for bacteria as does a sequestrum, and the reaction to an infected implant is similar to the reaction to a sequestered piece of bone. Treatment strategies are aimed at removing the infected and dead pieces of bone or implants to allow revascularization of the area and any remodeling that is necessary to reconstitute the blood supply.

Clinical signs associated with this type of infection are usually very localized. There is usually localized swelling, with minimal to absent systemic signs. If the infection is postsurgical, the appearance of the soft tissue is a much more sensitive indicator of local infection than the results of a complete blood count or radiographic examination. Acutely, a radiographically detectable bony response has not occurred. However, chronically, radiographs are the best diagnostic tools in determining the presence or absence of an infection and its duration. In cases with sequestra, a culture often results in a mixed growth. Postsurgical cultures will more often be indicative of the singular bacteria that colonized the wound. Culture results from chronic cases often include gram-negative opportunists such as Proteus spp. that may have gained access to the wound by means of the chronically draining tract. Localized signs usually do not include lameness unless there is instability or unless exudate has accumulated to the point at which excessive pressure is created.

References

Fig. 3. Illustration of the osseous granuloma that develops in response to an infected sequestrum.

Fig. 4. Radiograph of a sequestrum involving the third metacarpal bone, illustrating the clinical situation shown in Fig. 3.