Posttraumatic osteomyelitis

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By definition osteomyelitis is a bacterial infection of bone and may lead to bone destruction (1). It may be acute or chronic and it is defined as hematogenous when it originates from a bacteremia and as contiguous focus when it originates from an infection in nearby tissue.

In adults osteomyelitis is most often related to open fractures or to any surgical procedure affecting bone or its adjacent tissues. Traumatic contaminated wounds may also progress to deep infection and osteomyelitis. Especially diabetic patients with poor circulation are prone to that mechanism.

Osteomyelitis associated with fractures can cause delayed union or non-union and complicate the treatment. According to the classification of Gustilo and Andersen the risk of a clinical infection in open fractures depends on the severity of the injury and ranges from 0% to 2% for type-I open fractures, 2% to 10% for type-II, and 10% to 50% for type-III. (2).

Etiology and pathogenesis

Multiple organisms are usually isolated from infected bone as a result of an open fracture. Yet Staphylococcus aureus remains the most commonly isolated pathogen. Gram-negative bacilli and anaerobic organisms are also frequently isolated.

Damaged bone and soft tissue expose numerous proteins, such as collagen and fibronectin, which bacteria can adhere to. In acute osteomyelitis inflammation is followed by local edema, bone infarction and resorption. Infection can track along Haversian and Volkmann canals out of the intramedullary canal to the cortex, causing disruption of cortical blood supply. Some of the dead cortical bone is detached gradually from the living bone to form a sequestrum. After cortical and periosteal disruption, infection may cause a soft tissue abscess.

Host related factors, like malnutrition, alcoholism, smoking, and systemic diseases, such as diabetes or peripheral vascular disease, also contribute to the persistence of osteomyelitis.

Pathologic features of chronic osteomyelitis are the presence of necrotic bone, the formation of new bone, and the exudation of polymorphonuclear leukocytes joined by large numbers of lymphocytes, histiocytes, and occasionally plasma cells. New bone forms from the surviving fragments of periosteum and endosteum in the region of the infection. It forms an encasing sheath of live bone, known as an involucrum, surrounding the dead bone under the periosteum. The involucrum is irregular and is often perforated by openings through which pus may track into the surrounding soft tissues and eventually drain to the skin surfaces, forming a chronic sinus (1).

Classification

There are several systems for classification of osteomyelitis. One commonly used classification was described by Cierny et al (3). It is based on anatomic osseous involvement and on the physiologic status of the patient.

Cierny-Mader classification, includes four anatomic stages:

Stage-1, medullary osteomyelitis, confined to the medullary cavity of the bone.

Stage-2, superficial osteomyelitis, involves only the cortical bone.
Stage-3, localized osteomyelitis, involves both cortical and medullary bone.
Stage-4, diffuse osteomyelitis, involves the entire thickness of the bone, with loss of stability.

With this system, a patient with osteomyelitis is classified as an A, B, or C host.
A-host, no systemic or local compromising factors.
B-host, affected by one or more compromising factors
C-host, severely compromised.

Assessment and diagnostics

Patients with posttraumatic osteomyelitis may have pain, edema, swelling and erythema in the site of previous surgery or trauma. In chronic cases there are sometimes draining sinuses in the skin. Probing these sinus tracts may reveal connection to infective focus in the bone. Signs of bacteremia such as fever and chills may be present in the acute phase of osteomyelitis but are uncommon in the chronic phase.

The leukocyte count may be elevated in cases of acute osteomyelitis, but it is often normal in chronic cases. The erythrocyte sedimentation rate is usually elevated in both acute and chronic osteomyelitis, and it decreases after successful treatment. The erythrocyte sedimentation rate usually rises immediately after operative débridement. An erythrocyte sedimentation rate that returns to normal during the course of therapy is a favorable prognostic sign. The C-reactive protein levels rise in acute and chronic osteomyelitis and decrease faster than the erythrocyte sedimentation rate in successfully treated patients. The leukocyte count, erythrocyte sedimentation rate, and C-reactive protein level should be monitored at the time of admission and during treatment and follow-up in all patients with osteomyelitis (1).

The diagnosis and determination of the etiology of osteomyelitis in the long bones depend on the isolation of the pathogen or pathogens in cultures of specimens from the bone lesion, blood, or joint fluid. If possible, culture specimens should be obtained before antibiotics are initiated.

It may take at least two weeks from the beginning of the infectious process until first radiographic changes appear. The earliest changes are swelling of the soft tissue, periosteal thickening and focal osteopenia. Then scalloping of the cortex, and loss of trabecular architecture of cancellous bone. Sequestrum appears as a dense bone surrounding a lucent area of bone destruction. Periosteal new bone, or involucrum, can be seen adjacent to radiolucent areas, often widening the diameter of the bone.

Computed tomography is integral to identifying sequestra and preoperative resection planning.

Magnetic resonance imaging is useful for differentiating between bone and soft-tissue infection.

In clinical practice plain radiographs should be made whenever acute or chronic osteomyelitis is suspected because they are simple, economical, and usually effective. Magnetic resonance imaging should be requested if the diagnosis is doubtful. If magnetic resonance imaging is not feasible because of the presence of hardware, bone scintigraphy (ideally, leukocyte scans for acute osteomyelitis and technetium scans for chronic osteomyelitis) should be performed. Computed tomography scans can be used to help establish a surgical plan both for acute and for chronic osteomyelitis (1). Metallic hardware may disturb the diagnostic value both of magnetic resonance imaging and computed tomography.

Principles of treatment

Chronic posttraumatic osteomyelitis is a surgical disease. Treatment protocol consists of debridement, systemic and local antibiotic treatment, skeletal stabilization, soft-tissue coverage, and bone-grafting and/or reconstructive procedures for treatment of ununited fractures and existing bone defects. These principles can be incorporated in a staged protocol, often implemented by a multidisciplinary team consisting of an orthopaedic surgeon, an infectious disease specialist, and a microvascular surgeon (4).

In order to develop a detailed management plan, imaging studies should be reviewed to assess the status of bone healing, the location and extent of cortical and medullary bone involvement, and the status and integrity of existing implants. The quality of the soft-tissue envelope, and the need for flap coverage, should be evaluated.

Debridement

Radical debridement of all nonviable tissue, including skin, soft tissue, and bone, is necessary. Debridement proceeds until bleeding, viable tissue is seen at the re-

section margins, to ensure that all foci of infection are removed. Viable bone is characterized by punctuate bleeding, known as the "paprika sign", indicating good vascularity (5). After surgical debridement the wound should be irrigated with a copious amount of saline solution, preferably by a pulsatile lavage system.

In patients with extensive or circumferential involvement of cortical bone, extensive resection of the involved area may be required. In these situations, preresection stabilization with external fixation should be obtained.

In patients with infected intramedullary rods, removal of the rod and reaming of the medullary canal are essential components of treatment. Reaming removes intramedullary debris and infected bone surrounding the removed intramedullary device. The Reamer-Irrigator-Aspirator (RIA) has distinct features that appear to be beneficial for management of intramedullary infections. It allows reaming under simultaneous irrigation and aspiration, which minimizes the residual amount of infected fluid and tissue in the medullary canal and the propagation of infected material (6).

Specimens of purulent fluid, soft tissue, and bone from the affected area should be sent for aerobic and anaerobic cultures; it is especially important to perform cultures for mycobacteria and fungi when the patient is immunocompromised or has a chronic infection (7).

Hardware policy

The decision to retain or remove implants from the site of an infected fracture must be individualized and depends on the time since the fracture fixation, bone-healing status, stability provided by the hardware, and fracture location. If the fracture has healed, the internal fixation device should be removed. When the fracture has not healed, the internal fixation device should be left in place as long as it is stabilizing the fracture. Loose hardware that is not providing stability should be removed. If the fracture has not healed and the hardware is removed, the fracture should be stabilized with another device, preferably by external fixator for diaphyseal non-unions of the tibia (4).

Bone defect

Successful treatment requires adequate management of dead space created by debridement. Osseous defect reconstruction has involved a variety of techniques, including healing by secondary intention, closed irrigation and suction systems, temporary antibiotic-impregnated beads, autologous bone graft, free vascularized bone grafts and muscle flaps.

In staged protocol, the cavity in the bone, created by debridement, can be filled temporarily with polymethyl methacrylate (PMMA) antibiotic-impregnated beads. The beads are usually removed within two to four weeks and replaced with a cancellous bone graft. The antibiotics that are most commonly used in beads are vancomycin, tobramycin, and gentamicin. Local antibiotic therapy is a safe technique resulting in high local concentration of antibiotics with minimal systemic levels (8).

The Papineau technique has been used to manage small defects that are less than 30% of bone volume. After debridement the wound is left open or filled with antibiotic beads and observed for a period of 10 to 14 days. This allows granulation tissue to line the defect, and once sufficient granulation tissue is present, the defect is filled with bone graft (9).

In larger osseous and soft tissue defects reconstruction by local or free microvascular muscle or osteomuscular flaps may be necessary in order to get stability for bone and vascularity for healing. Muscle flap reconstruction in conjunction with autologous bone grafting is a reliable method, in one stage or two stage procedures. Vascular bone is option in large bone defects when it is important to get better primary stability after reconstruction. Common donor sites for free bone grafts are iliac crest, fibula and scapula. They all can include muscle and skin, depending on the needs of the clinical situation (10–12).

An alternative to vascularized bone grafts involves bone transport mechanisms based on Ilizarov technology. The procedure involves an external fixation frame that is oriented to allow both distraction of an osteotomy site and compression of a docking site. The Ilizarov frame has been used successfully in treatment of large segmental defects resulting from debridement of osteomyelitis (13,14).

Bioactive glass is a relatively new product for filling osteomyelitis related bone defects. Bioactive glasses (BAGs) are synthetic biocompatible osteoconductive bone substitutes, with bone bonding capacity and documented antibacterial and angiogenesis-promoting properties. According to the primary clinical experience from osteomyelitis related bone defects, bioactive glass can be used in a one stage procedure:

after debridement immediate filling of the cavity and wound closure. With this protocol there is no need for second procedure, like it is in the traditional protocol: removal of antibiotic beads and autologous bone grafting (15,16).

Antibiotic treatment

Antibiotic treatment should be based on sensitivity studies of cultures of bone taken at the time of débridement or deep bone biopsies. The traditional duration of treatment in most stages of osteomyelitis is four to six weeks. The rationale for this duration is based on the results of animal studies and the observation that revascularization of bone after debridement takes about four weeks. There is no clear clinical or scientific evidence supporting longer treatment periods. Treatment failures occur, whatever the type of the antibiotic or duration of the treatment, if surgical debridement has been inadequate (17).

Summary

Posttraumatic osteomyelitis is challenging and expensive to treat, despite advances in antibiotics and new operative techniques.

Plain radiographs still provide the best screening tool for acute and chronic osteomyelitis. Other imaging techniques may be used to determine diagnosis and aid in treatment decisions.

Operative treatment includes debridement, obliteration of dead space, restoration of blood supply, adequate soft-tissue coverage and stabilization of the bone.

Antibiotic treatment is an integral part of the treatment of osteomyelitis. The duration of antibiotic treatment is long, in most stages of osteomyelitis four to six weeks.

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