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Surgical management of chronic osteomyelitis

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Abstract

Chronic osteomyelitis is a surgical disease that can require significant dedication from both patients and surgeons to eradicate. Osteomyelitis can result from a variety of etiologies but most often is a consequence of trauma to a long bone, frequently the tibia. It is important to understand the etiology of the infection, as well as the pathophysiology of its chronicity. Additionally, the surgeon must individualize treatment for each patient, because host morbidities often play an important role in propagation of infection. Treatment requires isolation of the pathogens, significant debridement for removal of all infective and necrotic material, and then bony and soft tissue reconstruction. We review the literature of surgical treatment of chronic osteomyelitis and discuss the numerous techniques available to the treatment team, including debridement, dead space management, Ilizarov techniques, and vascularized reconstruction. These patients often require a multimodality approach that incorporates a team approach involving orthopedic and plastic surgery, as well as infectious disease and general medicine.

Osteomyelitis is a severe infection of bone that can arise from a variety of mechanisms. Although empirically understood, there is no generally accepted definition of chronic osteomyelitis. Numerous functional definitions have included a variety of criteria: clinical or radiographic evidence of infection >6 weeks, relapse or persistence of infection after appropriate antibiotic therapy, and infections associated with foreign bodies or vascular abnormalities.

The term *osteomyelitis* was first used by French surgeon Edouard Chassaignac in 1852 [1]. Injury (often an open fracture) is the most common etiology in the adult patient. Other etiologies include postoperative infection either from an elective procedure or from open reduction, internal fixation of closed fractures. Rarely, adult patients develop osteomyelitis after hematogenous seeding of long bones during an episode of bacteremia. Often osteomyelitis results in the formation of *sequestra* (termed by John Hunter [2] in 1764, describing pockets of dead cortical bone with abscess) and *involucrum*, or new bone formed in response to the sequestra.

Largely because of the avascular nature of sequestra, osteomyelitis is difficult to treat and can be associated with a high morbidity and possible mortality for the patient. Treatment is aimed at resolution of infection and maximization of patient function. Historically, surgical management has been aimed at extensive debridement of infected bone and lengthy antibiotic regimens. Celsus, in the first century AD, recommended scraping dead bone away until it bleeds during debridement [3]. Before the elucidation of the germ theory, no one understood the significance or principles of infection, and most patients with open fractures subsequently died. As a result, early treatment consisted of amputation of the injured extremity.

Carel and Dakin [4] used continuous irrigation to treat open fractures in soldiers during World War I. Later, Orr and Trueta [5,6] recognized the importance of the surgical management of osteomyelitis and emphasized debridement of sequestrum, stabilization of the bone, and maintaining the open wound. These principles led to increased success with the management of open fractures and osteomyelitis, but still, 30% of cases had persistent local infection or systemic sepsis when only surgical debridement and antibiotic therapy were used [7–12].

It was not until the last 25 years that treatment of chronic osteomyelitis significantly advanced with the use of muscle flaps and vascularized bone transfers to manage large open defects after debridement [12–20]. Further advancements have included antibiotic beads to manage dead space in staged reconstructions [21–23] and the use of external fixators in the Ilizarov technique of skeletal reconstruction [24–30]. These advances have led to increased success in the management of chronic osteomyelitis with success rates >90% in the literature [14,16,18,21,29,31–33].

Chronic osteomyelitis is a surgical disease that can often

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Table 1
May classification of tibial osteomyelitis

Type	Description	Rehabilitation Time
Type 1	Intact tibia and fibula. No osseous reconstruction necessary.	6–12 wk
Type 2	Intact tibia but requiring bone graft for structural support.	3–6 mo
Type 3	Tibial defect ≤6 cm. Intact fibula. Reconstruction required.	6–12 mo
Type 4	Tibial defect >6 cm. Intact fibula. Reconstruction required.	12–18 mo
Type 5	Tibial defect >6 cm without usable fibula. Recommended amputation.	≥18 mo

Adapted from J Bone Joint Surg Am [42].

pose a difficult problem for the treating surgeon. It is important to understand the treatment principles involved, beginning with patient evaluation, debridement techniques, antibiotic therapy, and management of resected dead space with vascularized reconstruction. Additionally, understanding the etiology and pathophysiology of these infections will guide the surgeon in a thorough treatment protocol.

Etiology and pathophysiology

Open fractures are the most common etiology of adult osteomyelitis, often of the tibia. Patzakis et al [34] found that open tibia fractures have a 24% chance of infection without antibiotic therapy, as compared with a 4.5% infection rate when treated with antibiotics. It has been shown that severity of soft tissue injury (by the Gustillo-Anderson classification of open fractures) is correlated with risk of infection, with grade 3B open fractures having a 15% to 40% risk of subsequent infection [35]. Other literature has shown that 60% to 70% of open fractures are contaminated with bacteria [36], most commonly *Staphylococcus aureus* and gram-negative bacilli. Chronic osteomyelitis is often polymicrobial [37,38]. Damaged bone and soft tissue expose numerous proteins, such as collagen and fibronectin, which bacteria can adhere to. Osteomyelitis associated with fractures can cause delayed union or nonunion and complicate the treatment.

Once bacteria bind to bone, acute inflammation occurs. Local edema and oxidative enzymes released by immune cells can cause 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, and resulting in sequestrum. After cortical and periosteal disruption, infection causes a soft tissue abscess. Chronic abcesses can violate the skin by means of sinus tracks, which, when chronic, are associated with a risk of squamous cell carcinoma [39,40].

Patients with chronic osteomyelitis often have periods of quiescence followed by flare-ups that can continue throughout life [41]. Persistence of infection is the result of a variety of etiologies, including damaged skin coverage, abundant scar tissue, and metal implants that are a nidus for bacteria and impaired vascularity. Decreased perfusion impairs healing, as well as the delivery and function of antibiotics, leading to polymicrobial resistance. Local ischemia also reduces oxygen tension, impairing bacteriocidal polymorphonuclear leukocytes. Patient factors, such as hypotension, malnutrition, alcoholism, and smoking, and systemic diseases, such as diabetes or peripheral vascular disease, also contribute to the persistence of osteomyelitis. These host factors impair the immune system's ability to fight infection. It is the combination of these local and systemic host factors that result in persistent infections.

Classification

Although there are many classification systems that address different clinical aspects of osteomyelitis, no 1 system is universally accepted. Weiland et al [17] classified patients by the amount of bone involved. Type 1 had open, exposed bone, without soft tissue infection. Type 2 was characterized by circumferential, cortical, and endosteal infection. Type 3 was associated with segmental bone defect. These types correlated with patient ambulatory status. May et al [42] developed a classification system based on tibial osteomyelitis. It stratifies the status of the tibia and fibula after debridement, and is useful for determining the length of rehabilitation the patient will need after surgical stabilization (Table 1).

The most widely used system for classification of osteomyelitis is the Cierny-Mader classification system for adult osteomyelitis [31] (Fig. 1). This system is based on anatomic osseous involvement and on the physiologic status of the patient. The 4 anatomic types are prognostic for dead space and bone management (Table 2). Most patients have type 3 or 4 osteomyelitis. The patient is staged based on the combination of their anatomic type and host classification. Staging is correlated with treatment and prognosis.

Preoperative evaluation and diagnosis

Patients with chronic osteomyelitis can present with a variety of symptoms, including localized bone and joint pain, erythema, swelling, fevers, night sweats, and a draining sinus. It is extremely important to take a thorough

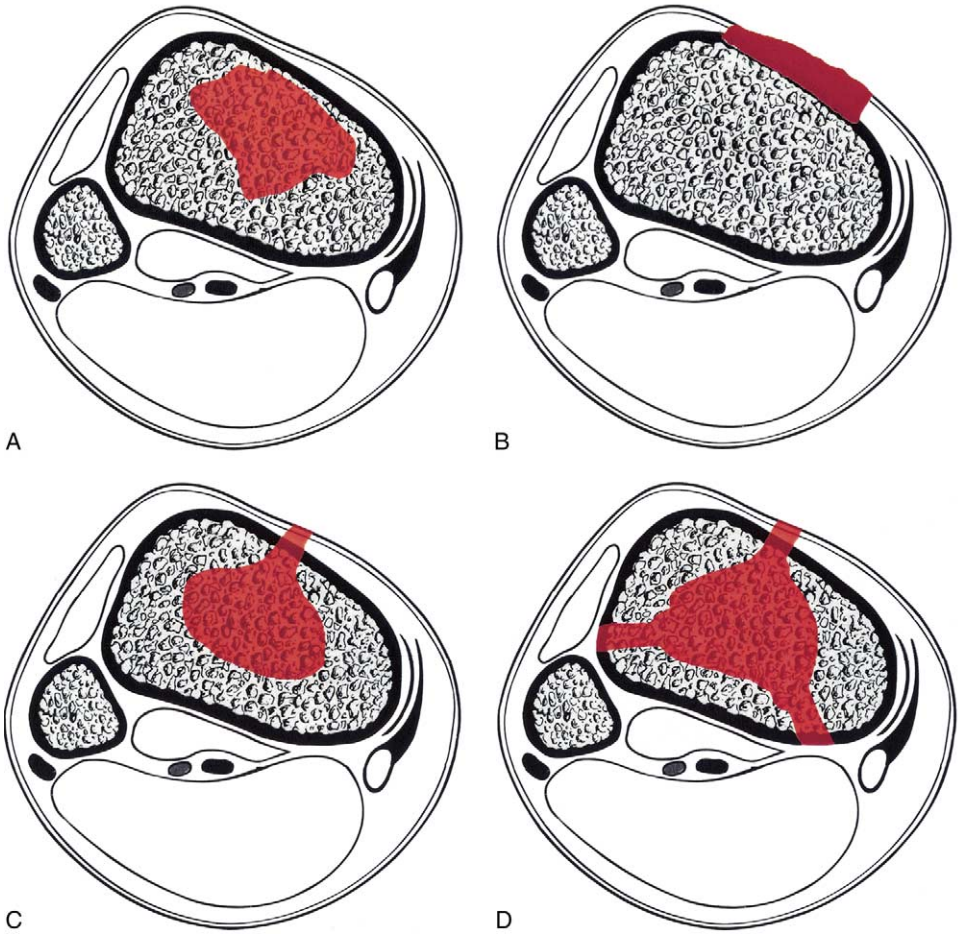

Fig. 1. The Cierny-Mader anatomic types of adult osteomyelitis. (*A*) Type 1 is intramedullary osteomyelitis, where the nidus is endosteal. (*B*) Type 2 indicates superficial osteomyelitis, which is limited to the surface of the bone. (*C*) Type 3 is termed localized osteomyelitis, in which the full thickness of the cortex of the bone is involved. This type of osteomyelitis often requires complex dead space management and osseous stabilization after debridement. (*D*) Type 4 is diffuse osteomyelitis involving the entire circumference of the bone. These lesions are mechanically unstable and require complex reconstruction.

Table 2
Cierny-Mader classification of adult osteomyelitis

Anatomic type	
Type 1	Medullary osteomyelitis (nidus is endosteal). No dead space management. Etiology often hematogenous, post-intramedullary rod.
Type 2	Superficial osteomyelitis. Limited to surface of bone. No dead space management but needs soft tissue coverage.
Type 3	Localized osteomyelitis. Full thickness of cortex. Complex dead space management, simple osseous stabilization
Type 4	Diffuse osteomyelitis. Circumference of cortex. Biomechanically unstable. Complex dead space and osseous management.
Physiologic host	
A Host	Normal host. Normal immune system. Normal vascularity.
B Host	Bs: systemic compromise Bl: local compromise Bsl: systemic and local compromise
C Host	Treatment morbidity worse than present condition with low prognosis for cure.

Adapted from Contemp Ortho [31].

history and physical examination, including date of injury, previous management techniques and procedures, as well as comorbid medical conditions. Patient goals for posttreatment function and outcome should be understood. Physical examination includes not only the site of infection but adjacent joints, muscles, and possible donor flap sites. Lab-

oratory tests, such as leukocyte count, estimated sedimentation rate, and C-reactive protein can be helpful in diagnosis. C-reactive protein is also useful for gauging response to therapy.

Radiographs are helpful in the diagnosis and staging of the patient. Changes can be subtle and can include osteopenia, scalloping of the cortex, and loss of trabecular architecture of cancellous bone. These changes often do not appear for 10 to 14 days. 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. Chronic changes can be significant and are not infrequently confused with cancerous lesions (Fig. 2).

Radionucleotide scans can also be useful in the diagnosis. However, these scans are nonspecific and do not aid in preoperative planning of resection. Technetium-99m scans show accumulation of isotope in areas of increased blood flow and reactive new bone. Gallium scans are more specific for infection and can delineate areas of inflammation.

Computed tomography is integral to identifying sequestra and preoperative resection planning (Fig. 3). Similarly, magnetic resonance imaging is useful for surgical planning because it delineates intraosseous and extraosseous involvement.

Treatment

Appropriate therapy requires a multimodality approach. Principles are aimed at eradication of infection by thorough debridement and appropriate antibiotic coverage. Neovascularization is crucial for keeping the healing debridement site aseptic. Neovascularization techniques must incorporate dead space management and reconstruction to achieve bony stability and cover the wound. Those patients with osteomyelitis associated with failure of fixation require both eradication of infection and osseous union. As such, chronic osteomyelitis is a surgical disease that requires understanding and expertise of debridement and reconstruction modalities. The multimodality approach often includes orthopedics, plastics, microvascular surgery, as well as infectious disease.

Additionally, host morbidities need to be optimized, including optimizing blood sugar levels in patients with diabetes, smoking cessation, and improving liver or renal function. The treatment options, ranging from antibiotic suppression to staged surgical debridement and reconstruction, should be discussed with the patient. Patients need to understand that treatment potentially involves a long process requiring multiple operations and significant patient cooperation. The surgeon faced with challenging patients needs to be able to diagnose, treat, and refer these patients when appropriate.

Antibiotic management

Antibiotics should be started empirically in patients after cultures have been obtained, usually at the time of debridement. Once cultures and sensitivities are obtained, the antibiotic regimen should be tailored accordingly. Often patients with chronic osteomyelitis have polymicrobial infections that can include both aerobic and anaerobic bacteria. Regimens are patient and infection specific, but some guidelines do exist. Mader et al [43] recommend regimens based on their staging system. Type 1 osteomyelitis is treated with 4 weeks of parenteral antibiotics. Type 2 often resolves after 2 weeks of antibiotics after debridement. Types 3 and 4 each require 4 weeks of parenteral antibiotics from the last debridement. Some centers use 2 weeks of parenteral antibiotics followed by 4 weeks of oral antibiotics. The requirement of 4 to 6 weeks is based on the fact that it takes that long for debrided bone to be protected by revascularized tissue [44]. Most antibiotic regimens will fail without neovascularization, because introducing local blood flow is crucial for a successful outcome [43].

Debridement

The surgical philosophy of debridement has been examined by numerous clinicians [19,32,45,46]. Debridement is aimed at removing all infected or necrotic bone and soft tissue, often fully identified at the time of surgery. Techniques are similar to those used for tumor surgery. Gentle soft tissue handling and knowledge of local vascularity in order to maintain preoperative blood supply and soft tissue coverage is crucial [45,47]. Often this requires multiple debridements in a staged fashion [32]. Debridement should be done with tourniquet control as an option.

Soft tissue debridement involves an expansile rather than an extensile approach. Previous scars should be crossed in a perpendicular fashion to maintain vascularity (Fig. 4). Accessory sinus tracts often do not need to be excised, because they will close with resolution of infection. In general, a scalpel should be used instead of cautery. Excision should obtain margins in normal healthy bleeding tissue. Tetsworth and Cierny [45] recommend a “pants over vest” incision, in which the subdermal dissection is directed tangentially toward fascia with improved blood supply. This allows for 2 separate layers for improved closure. Scar tissue should be removed because it causes tension, decreases the rate of wound healing, and acts as foci for infection. Braided or absorbable sutures should be avoided because they can harbor microorganisms and cause inflammatory responses. Serial debridements should not be done until the wound has pink, bleeding granulation tissue and cultures are negative.

Bone debridement follows specific guidelines. Bone is exposed in an expansile manner that is extraperiosteal. Periosteal stripping should be avoided because it results in avascularity. Involucrum surrounding infection is living

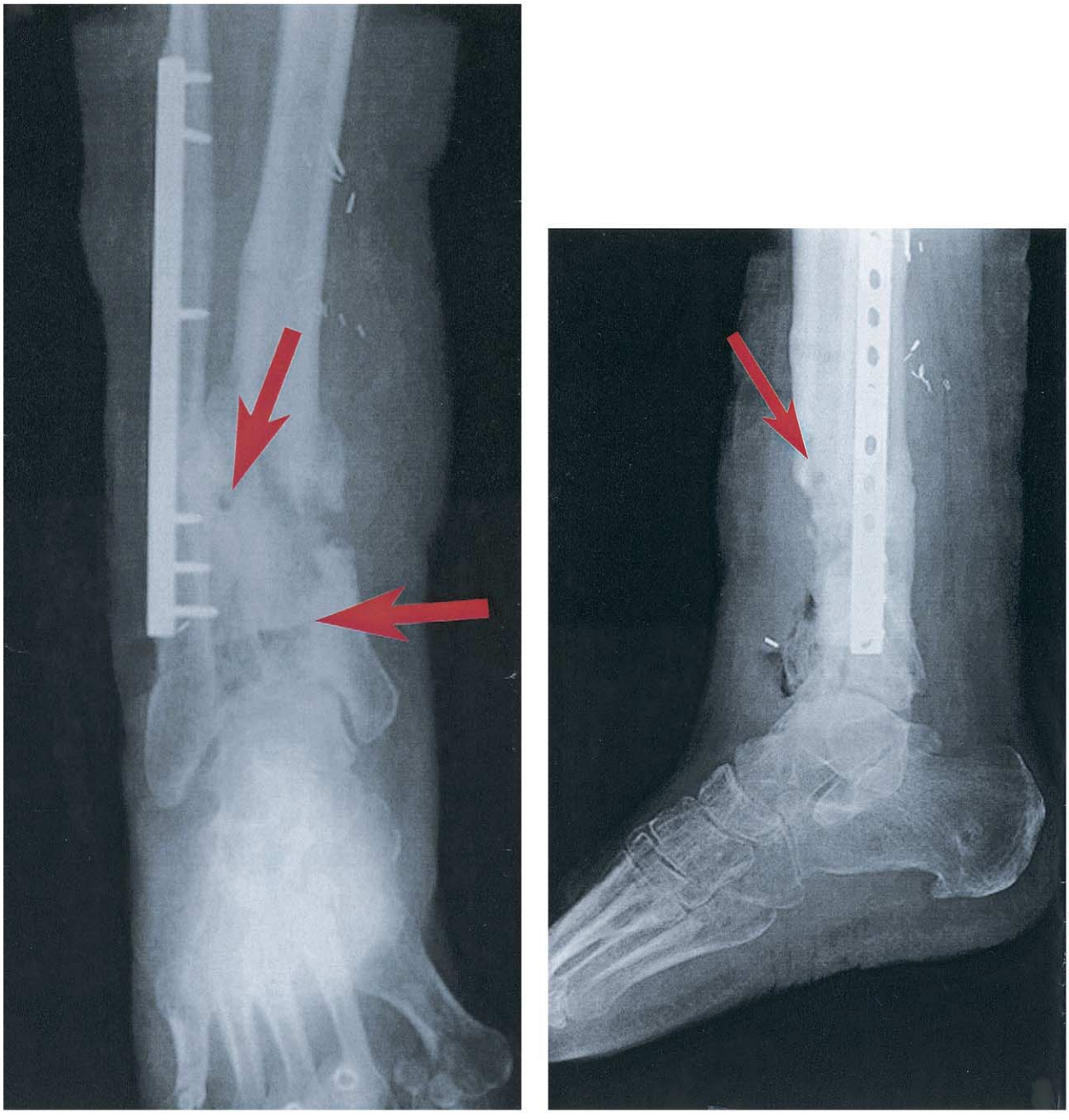

Fig. 2. Anteroposterior (*left*) and lateral (*right*) radiographs of a tibia and fibula with internal fixation showing chronic changes of osteomyelitis of the tibia. Significant cortical destruction is apparent. Areas of radiolucency surrounded by dense sclerotic bone indicate sequestra (*arrows*). As a result of the osseous destruction, this tibia is no longer biomechanically stable and had to be reconstructed.

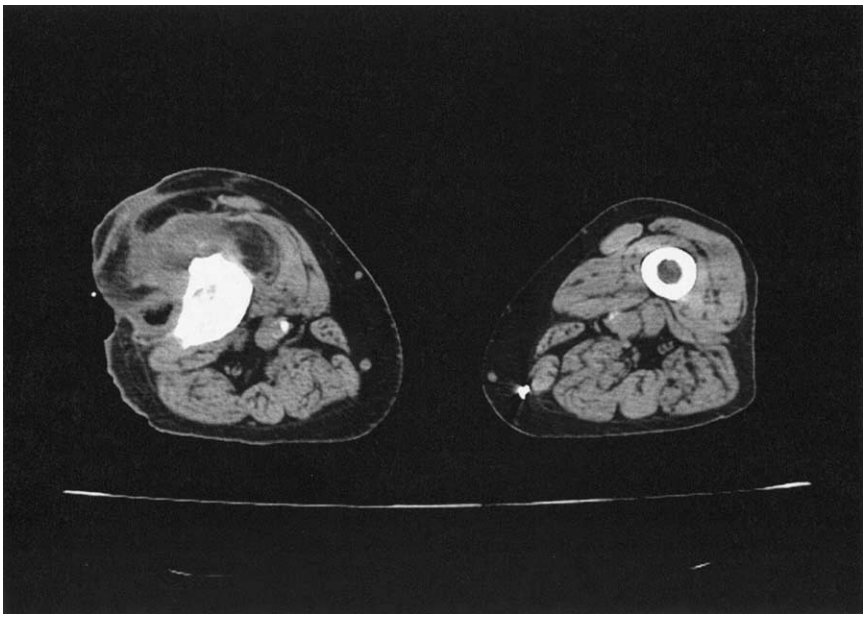

Fig. 3. Computed tomography of a femur with signs of sequestrum. Note thickened cortex and destruction of the normal intramedullary cavity in the right femur. Soft tissue changes anterior and lateral to the right femur are also apparent.

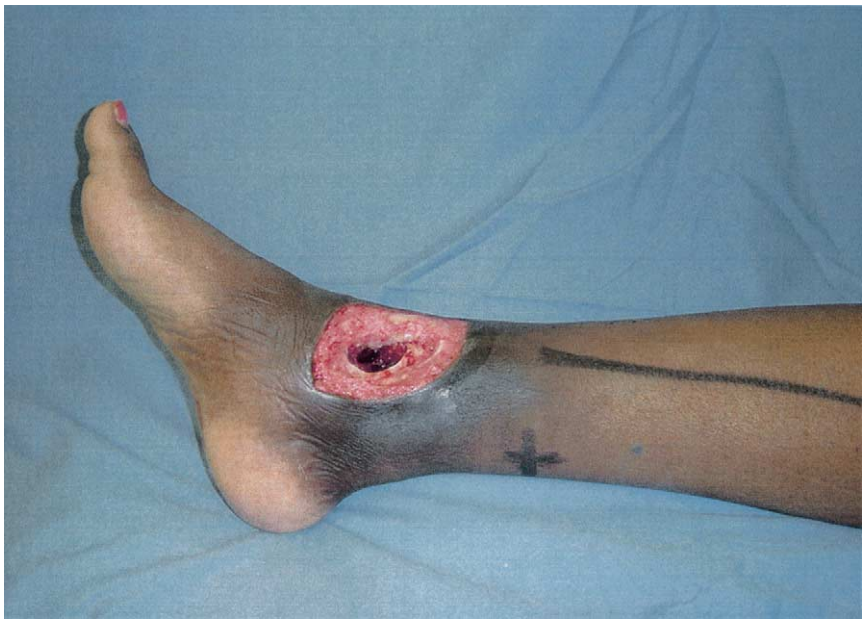

Fig. 4. Techniques of improved soft tissue dissection include using an expansile approach to the infected area. This patient developed osteomyelitis after an open ankle injury and had a persistent draining sinus. The involved area was debrided with wide margins in an effort to eradicate necrotic and infected tissue.

bone and does not need to be debrided. Precise bone debridement is best done with high-speed burrs and is performed until the paprika sign [31] (defined as scattered pinpoint bleeding indicating good vascularity) is obtained (Fig. 5). Thermal effects from the burr are minimized with irrigation.

Endosteal infection (type 1 by Cierny-Mader classification) can sometimes be debrided with intramedullary reaming and canal irrigation [48], especially in patients with infected intramedullary rods. However, if infection extends into the metaphysis or preoperative radiographs reveal endosteal scalloping, intramedullary reaming is not adequate. In such situations debridement of the intramedullary canal needs to be done by means of a trough in the bone. Biomechanical studies have shown that an elongated oval trough oriented parallel to the long axis of the bone is structurally the best [49] (Fig. 6). The trough should be no larger than 7 to 10 mm in diameter and 3 to 9 cm in length, depending on the size of the bone. Once the trough is made, it is important to understand the risk of iatrogenic fracture. In general,

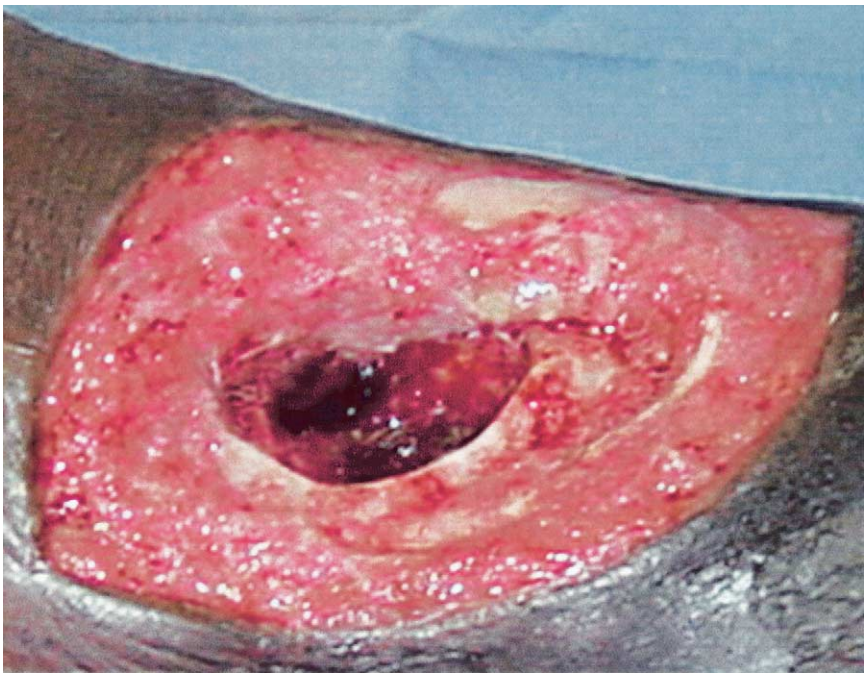

Fig. 5. The goal of osseous debridement is to remove dead and infected material until healthy bleeding bone is obtained. Resection should be continued until healthy bleeding bone is obtained (termed the "paprika sign").

when ≥70% of the original cortex remains intact, stabilization is not necessary.

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. Osteotomies are done with an oscillating saw and irrigation to prevent thermal damage of the bone. Osteotomy sites are inspected to ensure the cut is through vascular bone. It is important to take surrounding thickened, possibly infected, periosteum with the resected segment.

Tissue obtained at the time of debridement is sent for culture and pathology, including frozen section. Wounds with questionable margins should be serially debrided, and repeat cultures should be taken. In general, erring on the side of increased debridement is recommended (more is better). Once the wound is composed of healthy granulation tissue with negative cultures, debridement can be stopped [32]. Often such aggressive resection leaves a significant dead space of bone and soft tissue that must be addressed.

Dead space management

Successful surgical management requires adequate management of dead space created by debridement. The goal is to replace dead bone and soft tissue with viable vascularized tissue. Osseous defect reconstruction has involved a variety of techniques, including healing by secondary intention, closed irrigation and suction systems, temporary antibiotic-laden polymethylmethacrylate (PMMA) beads, autologous bone graft, and vascularized free fibula or iliac crest bone grafts. Soft tissue defects have been treated with skin graft, local muscle flaps, or vascularized free flaps [12–20].

In general, healing by secondary intention and closed irrigation systems have been shown to be inadequate in resolving infections. Shannon et al [10] had <70% success rate treating patients with primary skin graft of significant dead space. Similarly, closed irrigation systems had poor success in clearing infections in series presented by Kelly et al [8] in 1970 and Clawson et al [50] in 1973. It was thought that these modalities failed because they lacked vascularity and, as a result, granulation and scar tissue encased bacteria in an ischemic pocket [51].

These difficulties led surgeons to use antibiotic cement beads for temporary stabilization and sterilization of osseous defects. Antibiotic-impregnated PMMA beads produce bacterocidal levels of antibiotics for 2 to 4 weeks, depending on the antibiotic, and are usually removed after this time and replaced with cancellous bone graft or vascularized bone graft [23,52–54]. Adams et al [23] found that clindamycin, vancomycin, and tobramycin had the best elution characteristics and highest level in bone and granulation tissue. Cierny [51] cited a 92% overall success rate in staged wound management with beads in 114 patients since 1983. Wounds should be closed over the beads with a large bore drain.

Alternatively, the Papineau [55,56] technique has been used to manage small defects that are <30% of bone volume. Defects >30% need osseous reconstruction, often with vascularized bone transfer or other modalities. Cierny [51] stresses that this technique should be reserved for the uncompromised host with subcutaneous bone involvement.

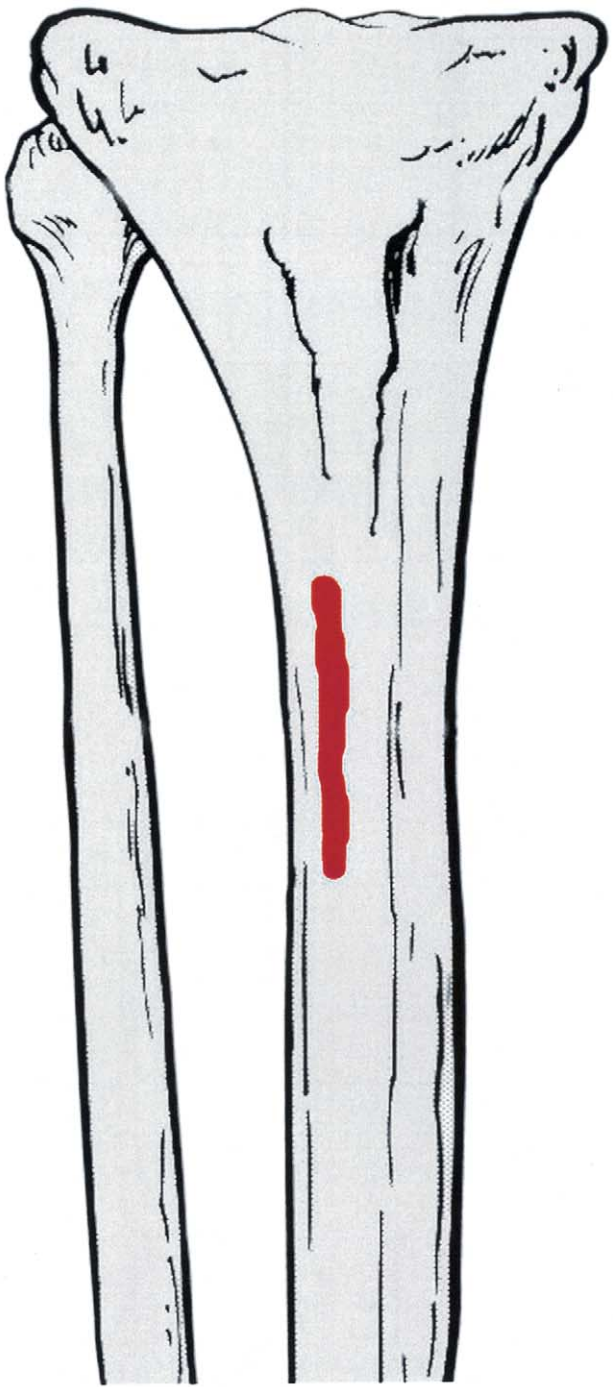

Fig. 6. Optimal intraosseous debridement is performed by making an elongated oval trough oriented parallel to the long axis of the bone to allow access to necrotic and infected material. A narrow trough maintains structural integrity of the bone as much as possible.

This technique uses thorough debridement with saucerization of the wound to give a brim to hold in bone graft. 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, usually 1/8 inch thick, the defect is filled with bone graft. Larger defects (15% to 30% bone volume) require serial grafting because cancellous graft layers >2 cm thick can result in graft necrosis. Once the graft has revascularized, it can be covered with skin graft or allowed to heal by secondary intention. The success of this technique has been reported in the literature as 70% to 93% [51,55,56]. Approximately 10% to 20% of patients require other surgical management for persistent infection.

Larger defects or those in a compromised host require more aggressive surgical management. Small osseous defects with a large soft tissue component that are structurally stable can be filled with a myoplasty, in which muscle is either rotated to fill the defect or transferred from a distant site using a microvascular free flap. Bone graft can be used in conjunction with the muscle to increase bone regrowth [20,32,33]. This is a more complex and difficult procedure but generally has more success than open bone grafting because it allows for immediate vascularization. Koval et al [19] reviewed a series of 25 patients and found that 80% of the flap-covered wounds healed, compared with 40% to 45% of the suction irrigation or open bone graft–managed wounds. An early series by Weiland et al [17] of 33 patients treated with muscle free flaps reported a 41% failure rate: 21% of grafts failed and 20% had recurrent sepsis. The more recent literature has shown myoplasty and free muscle flaps to have a success rate of 80% to 95% resolution of infection [12–14,16,18,20].

Larger osseous defects require bone for stability and can be managed with a vascularized bone flap. The fibula is used often, although the rib, iliac crest, and scapula have been used as well. These procedures are very complex, and often the bone used is smaller than the defect. The success of these procedures has been less than that of the free flap or myoplasty patients, but this is often because of increased complexity of the procedure and patient wound. Series by Wood et al [57] and Weiland et al [17] show success in 55% to 60% of cases. Similarly, Nahai and Cierny [51] report 80% success in 10 patients treated with vascularized fibula grafts.

An alternative to vascularized bone grafts involves bone transport mechanisms based on Ilizarov technology [24–26]. This method allows for bone reconstruction of segmental defects in both uncompromised and compromised hosts. The success is the result of the fact that new bone formed by means of distraction osteogenesis is highly vascular. The procedure involves an external fixation frame that is oriented to allow both distraction of an osteotomy site or compression of a docking site. Similar to other procedures, a thorough debridement of the infected area is performed. After the frame is secured to the bone, a corticotomy is made away from the area of infection. After 5 to 7 days of preliminary callus formation, the corticotomy is distracted by means of transport wires, thereby closing the skeletal defect. The wires cut through the skin and soft tissue while transporting the intercalary segment, and care must be taken to avoid neurovascular structures when placing the wires and anticipating the path. The intercalary segment is dis-

tracted 0.25 mm 4 times a day. This distraction is continued until the resected defect is filled. At this point compression is applied to the docking site.

The Ilizarov frame has been used successfully in treatment of large defects resulting from debridement of osteomyelitis. Green [27] and Marsh et al [28] reported results of patients treated with Ilizarov techniques versus patients treated with Papineau bone grafting and external fixation. Similar rates of healing and time in fixators were found for both groups. Paley et al [30] reported 100% union and >76% successful resolution of infection in 13 patients with infected nonunions of the tibia. No bone graft was necessary in any patient for filling the defect after debridement. Morandi et al [29] report no recurrence of infection in 13 patients followed for 2 years after Ilizarov reconstruction.

The procedure has been used in conjunction with bone graft, especially at the docking site, as well as soft tissue transfer for large defects, with similar success rates [58–60]. The Ilizarov technique is very labor intensive and requires great compliance by the patients. Pin tract infections and problems with union of the docking site are not uncommon, occurring in 28% to 38% of cases [51]. The Ilizarov method offers surgeons another option when dealing with segmental osteomyelitis.

Conclusion

Chronic osteomyelitis is a condition associated with potentially high morbidity and possibly mortality and has historically been very difficult to cure. Treatment is geared toward resolution of infection while maintaining optimal function in the patient's extremity. Historically, treatment involved amputation, but with the emergence of antibiotics, patients could be managed with suppression. Patients treated in this manner failed treatment >30% of the time. With the advent of free flap technology and Ilizarov techniques, the failure rate has been reduced to approximately 10% to 15%. However, the key to successful eradication of infection remains thorough debridement of all infected and necrotic tissues.

Although a variety of treatment options are available, no set guideline or algorithm is available for treating patients with chronic osteomyelitis. Cierny [51] and others stress that treatment should be individualized to the patient. Management should take into account the anatomic aspects of the patient's infection, comorbid medical conditions, and the patient's perception of the expected outcome. This will allow the optimum outcome in the management of these patients.

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