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Conn's Current Therapy

LATEST APPROVED METHODS OF TREATMENT
FOR THE PRACTICING PHYSICIAN

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nent visual loss is the most dreaded complication and occurs in 6% to 10% of patients.

The laboratory hallmark of PMR and GCA is an elevated sedimentation rate. It is usually in excess of 50 mm/h, and may exceed 100 mm/h. Patients may also have a mild anemia. The diagnosis of PMR is primarily clinical, but it is aided by the elevated sedimentation rate and a dramatic response to therapy. The diagnosis of GCA can be confirmed by a temporal artery biopsy. The biopsy specimen must be as long as possible (2–4 cm) and sectioned in multiple areas because the process can be segmental.

TREATMENT

The symptoms of PMR usually respond dramatically to 10 to 20 mg/d of prednisone. The response usually occurs within 24 to 48 hours and always within 1 week. Occasionally, there is only a partial response to a single daily dose, but a complete response can be obtained by splitting the dose, such as 10 mg in the morning followed by 5 mg in the evening. After 1 month at the response dose, a slow taper of no more than 1 mg every 2 weeks should be begun. If symptoms recur, the prednisone dose can be increased by 1 to 2 mg/d. The sedimentation rate should improve with therapy, but symptoms should guide the taper primarily. Once the dose has reached 7 mg/d, the taper should be 1 mg/month. The dose should be reduced to the lowest level required to suppress symptoms. Sixty percent to 70% of patients are able to stop therapy after 2 years. PMR patients should be warned about the symptoms of GCA.

GCA treatment requires 40 to 60 mg/d of prednisone in divided doses. Response is usually rapid; and once corticosteroids are started, blindness is rare. If GCA is suspected, start therapy and arrange for a biopsy within 1 week. Do not withhold therapy if clinical suspicion is strong enough to warrant a biopsy. If visual loss occurs in a patient with GCA, the prognosis for restoring sight is poor. However, in such cases, a course of high-dose corticosteroids should precede prednisone therapy (e.g., 1 g of methylprednisolone [Solumedrol] administered intravenously daily for 3 days). After 1 month of prednisone therapy, the dose should be decreased by 5 mg every week until a dose of 20 mg/d is reached. Thereafter, a conventional PMR taper can be used. Because the sedimentation rate should drop with therapy, a patient's symptoms should guide the taper. Patients should be warned to expect treatment for approximately 2 years.

PMR and GCA patients who are unable to taper their prednisone dosage because of recurring symptoms can be treated with corticosteroid-sparing medications such as hydroxychloroquine (Plaquenil Sulfate),* methotrexate,* or azathioprine (Imuran).* The efficacy of corticosteroid-sparing agents in PMR and GCA is equivocal. Careful monitoring and preventive therapy for corticosteroid-induced osteoporosis is essential for patients on prolonged prednisone therapy.

*Not FDA approved for this indication.

OSTEOMYELITIS

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Osteomyelitis is characterized by bacterial or fungal infection of the bone, commonly the cortical and/or medullary portions. The terms *osteo* and *myelo* refer to bone and to the marrow cavity, respectively, both of which are involved in the disease. This infectious disease is progressive and results in inflammatory destruction of bone, bone necrosis, and new bone formation.

CONTIGUOUS FOCUS OSTEOMYELITIS WITH *NO* GENERALIZED VASCULAR INSUFFICIENCY

In contiguous focus osteomyelitis, the organism may extend from adjacent soft tissue infections or be directly inoculated into the bone by trauma or pre/intraoperative procedures. Common predisposing conditions include open fractures, surgical reduction and internal fixation of fractures, chronic soft tissue infections, and radiation therapy. In contrast to hematogenous osteomyelitis, multiple bacterial organisms are usually isolated from the infected bone. The bacteriology is diverse, but *Staphylococcus aureus* remains the most commonly isolated pathogen. In addition, aerobic gram-negative bacilli and anaerobic organisms are frequently isolated. Bone necrosis, soft tissue damage, and loss of bone stability occur, often making this form of osteomyelitis difficult to manage. The long bones are most frequently involved.

Therapy

Adequate drainage, thorough débridement, obliteration of dead space, wound protection, and specific antimicrobial coverage are the mainstays of therapy. Loss of bone stability, bone necrosis, and soft tissue damage frequently occur, making this form of osteomyelitis difficult to treat. Surgical débridement of infected bone and soft tissue provides specimens for culture and hastens eradication of the infection. Other steps in the surgical management of contiguous focus osteomyelitis should be tailored to the specific anatomy of the bone infection. Antimicrobial therapy should begin with a broad-spectrum antibiotic regimen. Table 1 outlines initial antibiotic therapy choices. Once the specific organism is identified, the antibacterial activity of different antibiotic classes can be determined by appropriate sensitivity methods. The disk diffusion method is often a sufficient guideline for antibiotic therapy. However, quantitative antibiotic sensitivity testing by the macro-

TABLE 1. **Initial Choice of Antibiotics for Therapy for Infectious Arthritis and Osteomyelitis (Adult Doses)**

Organism	Antibiotics of First Choice	Alternative Antibiotics
Methicillin-sensitive *Staphylococcus aureus*	Nafcillin (Unipen), 2 g q4h, or clindamycin, 900 mg q8h*	Cefazolin (Ancef)
Methicillin-resistant *Staphylococcus aureus*	Vancomycin (Vancocin), 1 g q12h*	Trimethoprim-sulfamethoxazole (Bactrim), or minocycline (Minocin) + rifampin (Rifadin) Quinupristin/dalfopristin (Synercid), linezolid (Zyvox)
S. epidermidis	Vancomycin, 1g q12h,* or nafcillin, 2 g q4h	Cefazolin, clindamycin
Group A *Streptococcus*	Clindamycin (Cleocin), 900 mg q8h	Benzylpenicillin, cefazolin, ampicillin
Group B *Streptococcus*	Clindamycin, 900 mg q8h	Benzylpenicillin, cefazolin, ampicillin
Enterococcus species	Ampicillin, 2 g q6h ± gentamicin, 5 mg/kg/d q8h or qd*	Vancomycin, linezolid (VRE) Quinupristin/dalfopristin (VRE)
Escherichia coli	Cefotaxime (Claforan), 2 g q6h	Cefazolin, levofloxacin (Levaquin), ampicillin
Proteus mirabilis	Ampicillin, 2 g q6h	Cefazolin, levofloxacin, trimethoprim-sulfamethoxazole
P. vulgaris, P. rettgeri, Morganella morganii	Cefotaxime, 2 g q6h, ± gentamicin, 5 mg/kg/d q8h* or qd	Ticarcillin-clavulanic acid (Timentin), levofloxacin
Serratia marcescens	Cefotaxime, 2 g q6h, ± gentamicin, 5 mg/kg/d q8h* or qd	Ticarcillin-clavulanic acid, levofloxacin
Pseudomonas aeruginosa	Ceftazidime (Fortaz), 2 g q8h or cefepime (Maxipime), 2 g q12h	Amikacin (Amikin), ticarcillin-clavulanic acid, ciprofloxacin (Cipro), imipenem (Primaxin)
Bacteroides fragilis group	Clindamycin, 900 mg q8h	Metronidazole (Flagyl), ampicillin-sulbactam (Unasyn)
Peptostreptococcus species	Clindamycin, 900 mg q8h	Metronidazole, ampicillin-sulbactam
Candida species	Amphotericin B (Amphocin, Fungizone), 0.5–0.75 mg/kg/d Fluconazole (Diflucan), 400 mg/d†	
Mycobacterium	Same 12-month antibiotic course as used for pulmonary tuberculosis	
Actinomyces species	Clindamycin, 300 mg qid × 6 months	
Brucella species	Doxycycline + rifampin for 6 to 24 weeks	

*Dose should be individualized with serum level monitoring.
†Increasing reports of fluconazole-resistant *Candida* species, especially *C. krusei* and *C. glabrata*.

dilution or microdilution techniques on all aerobic bone isolates is a prerequisite to determine the minimal concentration of the antibiotic to inhibit (minimal inhibitory concentration [MIC]) and kill (minimal bactericidal concentration [MBC]) the pathogenic organism(s). It is best to choose an antibiotic or antibiotic combination that has a low MIC/MBC relative to its expected serum concentration. The initial antibiotic regimen may be continued or changed on the basis of sensitivity results. The patient is treated for 4 to 6 weeks with appropriate antimicrobial therapy dated from the initiation of therapy or after the last major débridement surgery.

Surgical Management

The principles of treating any infection are equally applicable to the treatment of infection in bone. These include adequate drainage, extensive débridement of all necrotic tissue, obliteration of dead spaces, stabilization, adequate soft tissue coverage, and restoration of an effective blood supply. The number of performed surgical procedures used to achieve these goals increases with the severity of the infection and procedures can be divided into three categories.

Category 1 is removal of necrotic tissue by extensive débridement. Débridement surgery is the foundation of osteomyelitis treatment. It is the most commonly performed procedure, and patients may require multiple débridements. The goal of débridement is to leave healthy, viable tissue. However, even when all necrotic tissue has been adequately débrided, the remaining bed of tissue must be considered contaminated with the responsible organism. Débridement should be direct, atraumatic, and executed with reconstruction in mind. All dead or ischemic hard and soft tissue are excised unless a noncurative procedure has been chosen. Surgical excision of bone must be carried down to uniform haversian or cancellous bleeding termed *the paprika sign* to ensure a positive outcome.

In category 2, there is dead space obliteration with flaps, antibiotic beads, and bone grafts. Adequate débridement may leave a large bony defect termed *dead space*. Appropriate management of dead space created by débridement surgery is mandatory to arrest the disease and to maintain the integrity of the skeletal part. The goal of dead space management is to replace dead bone and scar tissue with durable vascularized tissue. Local tissue flaps or free flaps may be used to fill dead space. An alternative technique is to place cancellous bone grafts beneath local or transferred tissues where structural augmentation is necessary. Careful preoperative planning is critical to conservation of the patient's limited cancellous bone reserves. Open cancellous grafts without soft tissue coverage are useful when a free tissue transfer is not a treatment option and local tissue flaps are inadequate. Complete wound closure should be attained whenever possible. Suction irrigation systems are not recommended because of the high incidence of associated nosocomial infections and the unreliability of these setups. Secondary intention healing is also discouraged because the scar tissue that fills the defect may later become avascular. Antibiotic-impregnated acrylic beads can be used to sterilize and temporarily maintain dead space. The beads are usually removed within 2 to 4 weeks and replaced with a cancellous bone graft. The most commonly used antibiotics in beads are vancomycin (Vancocin), tobramycin (Nebcin), and gentamicin (Septopal). Local delivery of antibiotics (amikacin [Amikin], clindamycin [Cleocin]) into dead space can also be achieved with an implantable pump. Adequate soft tissue coverage of the bone is necessary to arrest osteomyelitis. Most soft tissue defects are closed by primary closure. Small soft tissue defects may be covered with a split-thickness skin graft. In the presence of a large soft tissue defect or an inadequate soft tissue envelope, local muscle flaps and free vascularized muscle flaps may be placed in a one- or two-stage procedure.

Category 3 includes stabilization by external or open reduction and internal fixation. If movement is present at the site of infection, measures must be taken to achieve permanent stability of the skeletal unit. Stability may be achieved with plates, screws, rods, and/or an external fixator. One type of external fixation allows reconstruction of segmental bone defects and difficult infected nonunions. The Ilizarov external fixation method utilizes the theory of distraction histogenesis whereby bone is fractured in the metaphyseal region and slowly lengthened. The growth of new bone in the metaphyseal region pushes a segment of healthy bone into the defect left by surgery. The Ilizarov technique is used for difficult cases of osteomyelitis when stabilization and bone lengthening are necessary. It can also be used to compress nonunions and correct malunions and in a small group of patients for reconstruction of difficult deformities that results from osteomyelitis. However, this technique is labor intensive and requires an extended period of treatment averaging 9 months in the device. The Ilizarov pins usually become infected and the device is painful. Infected pseudarthrosis with segmental osseous defects can be treated by débridement and microvascular bone transfers. Vascularized bone transfer is also useful for the treatment of infected segmental osseous defects of long bones that are more than 3 cm in length. Vascularized bone transfers can be placed after 1 month of inactive sepsis.

Surgical procedures for long bone osteomyelitis can be tailored to the specific anatomy of the bone infection. When the nidus of infection is entirely within the medullary canal of the bone, surgical treatment is usually more straightforward than in other types of bone involvement. Adult patients are surgically treated with a thorough intramedullary reaming, and unroofing is usually performed with or without bone grafting. Soft tissues are reapproximated, and the limb is protected by external means (brace or cast) until the structural integrity of the bone is re-established by normal remodeling. When osteomyelitis is characterized by a full-thickness, cortical sequestration, patients can usually be treated with removal of the dead infected bone (bone saucerization). Bone grafting may be necessary to augment structural support. These patients may require external fixation for structural support while the bone graft incorporates. Complex reconstruction of both bone and soft tissue is frequently necessary. In some cases, osteomyelitis progresses to an infection involving a segmental section of the bone. These patients often require an intercalary resection of the bone to arrest the disease process. Because this advanced stage of osteomyelitis involves an entire through-and-through section of bone, there is a loss of bony stability either before or after débridement surgery. As a result, treatment often must be directed toward establishing structural stability and obliterating débridement gaps by means of cancellous bone grafts or the Ilizarov technique (see earlier). Free flaps and vascularized bone grafts are other possible treatment modalities. All of the modalities previously discussed may have a place in the treatment of this type of osteomyelitis.

After surgery, patients are initially given a broad-spectrum antibiotic that is changed to specific antimicrobial therapy based on meticulous bone cultures taken at débridement surgery or from deep bone biopsy specimens. For a list of antimicrobial regimens for specific pathogens usually associated with osteomyelitis, see Table 1. The authors recommend 2 weeks of intravenous antibiotic therapy followed by 4 weeks of oral therapy except in cases of candidiasis, tuberculosis, actinomycosis, and brucellosis of the bone (also see Table 1 for these antimicrobial regimens).

Host Defect Alteration

If the patient is a compromised host (Table 2), an effort is also made to correct or improve the host defect(s). These include improving the nutritional, medical, and vascular status of the patient and treating any underlying diseases. Host factors are primarily involved with containment of the infection once it is introduced adjacent to or into bone. A systemically and/or locally compromised host does not contain the infection as well as a normal host, and the infection may permeate the bone. Host deficiencies that lead to bacteremia favor the development of hematogenous osteomyelitis.

Exceptions for Obtaining Cultures

Although surgical management is usually initiated before antimicrobial therapy, there are instances when antibiotics are given first. Examples of these situations include delaying surgical treatment whe the treatment is worse than the disease or when the patient's condition is serious. Under these conditions,

TABLE 2. **Cierny and Mader Classification System**

Anatomic Type	
Stage 1	Medullary osteomyelitis
Stage 2	Superficial osteomyelitis
Stage 3	Localized osteomyelitis
Stage 4	Diffuse osteomyelitis
Physiologic Class	
A Host	Normal host
B Host	Systemic compromise (Bs) Local compromise (Bl)
C Host	Treatment worse than the disease
Systemic or Local Factors That Affect Immune Surveillance, Metabolism, and Local Vascularity	
Systemic (Bs)	*Local (Bl)*
Malnutrition	Chronic lymphedema
Renal, liver failure	Venous stasis
Diabetes mellitus	Major vessel compromise
Chronic hypoxia	Arteritis
Immune deficiency	Extensive scarring
Malignancy	Radiation fibrosis
Extremes of age	Small vessel disease
Immunosuppression	Complete loss of local sensation
Tobacco abuse	
Intravenous drug use	

patients are treated with antimicrobial therapy until they have stabilized. Antibiotics are then halted for 2 to 3 days, and surgical management is performed.

CONTIGUOUS FOCUS OSTEOMYELITIS WITH GENERALIZED VASCULAR INSUFFICIENCY

The majority of the patients placed into this category of osteomyelitis have diabetes mellitus or peripheral vascular disease from atherosclerosis. The small bones of the feet, talus, calcaneus and distal fibula, and tibia are commonly involved in this category of infection. The infection is usually initiated in soft tissue by minor trauma of the feet, such as infected nail beds, cellulitis, or a trophic skin ulceration. The diminished arterial blood supply has traditionally been considered to be the major predisposing factor. Recent observation suggests that neuropathy is an equally important factor in diabetic patients. Identifiable neuropathy as a complication of diabetes mellitus is present in approximately 80% of patients with foot disease. Multiple organisms are found in patients with osteomyelitis involving the small bones of the feet including *S. aureus*, coagulase-negative *Staphylococcus* species, *Streptococcus* species, *Enterococcus* species, gram-negative bacilli, and anaerobes. Aerobic gram-negative bacilli are usually a part of mixed infection. Cultures obtained by deep bone biopsy or during débridement procedures are indispensable in the diagnosis and choice of directed antimicrobial therapy of contiguous focus osteomyelitis. Culture results not only accurately identify responsible pathogens but also identify those patients with bone lesions that resemble but are not osteomyelitis.

Therapy

Determination of the vascular status of the tissue at the infection site is crucial in the evaluation of these patients. Although several methods can be used to determine the vascular status, measurement of cutaneous oxygen tensions and pulse pressures are the most commonly employed. Cutaneous oxygen tensions are obtained using a modified Clark electrode applied to the skin surface. Cutaneous oxygen tensions provide guidelines for determining the location of adequately perfused tissue. The values are also helpful in predicting the benefit of local débridement surgery and in selecting surgical margins where healing can be expected to occur. Revascularization, if possible, and/or hyperbaric oxygen therapy facilitates healing in areas where borderline oxygen tensions are present.

The patient may be managed with suppressive antibiotic therapy, local débridement surgery, or ablative surgery. The decision regarding treatment options used is based on tissue oxygen perfusion at the infection site, extent of the osteomyelitis and patient preference. The patient can be offered long-term suppressive antibiotic therapy when a definitive surgical procedure would lead to unacceptable patient morbidity or disability, or in cases in which the patient refuses local débridement or ablative surgery. Even with suppressive antibiotic therapy, in time, most of these patients will require an amputation of the involved bone. Local débridement surgery and a 4-week course of antibiotics may be employed in the patient who has osteomyelitis in a bone(s) amenable to débridement. Unless good tissue oxygen tensions are present, the wound will fail to heal and ultimately require an ablative procedure. Digital and ray resections, transmetatarsal amputations, midfoot disarticulations, and Chopart, Lisfranc, and Syme amputations (amputation of the foot with retention of the heel pad) permit the patient to ambulate without a prosthesis. The amputation level is determined by the vascularity and potential viability of the tissues proximal to the site of infection. The patient is given 4 weeks of antibiotics when infected bone is surgically transected. Two weeks of antibiotics are given when the infected bone is completely excised but some residual soft tissue infection remains. When the amputation is performed proximal to the bone and soft tissue infection, the patient is given 1 to 3 days of antibiotic therapy.

HEMATOGENOUS OSTEOMYELITIS

Hematogenous osteomyelitis is caused by microbial seeding of the involved tissue through the host's circulatory system and mainly occurs in infants and children. Because of the abundant vascular supply, the most frequent sites of hematogenous osteomyelitis are the metaphyses of the long bones (tibia, femur). A single pathogenic organism is almost always recovered from the bone in hematogenous osteomyelitis. In the infant, *S. aureus*, group B *Streptococcus*, and *Escherichia coli* are the most frequently recovered bone isolates, whereas in children older than 1 year of age *S. aureus, Streptococcus pyogenes*, and *Haemophilus influenzae* are most commonly isolated. After age 4 the incidence of *H. influenzae* osteomyelitis decreases. However, the overall incidence of *H. influenzae* as a cause of osteomyelitis is decreasing because of the *H. influenzae* vaccine now given to children. In the adult, *S. aureus, S. epidermidis,* and aerobic gram-negative organisms account for the majority of the bone or blood isolates.

Therapy

Appropriate therapy for hematogenous osteomyelitis includes correct antimicrobial therapy and, if necessary, adequate drainage, thorough débridement, obliteration of dead space, and wound protection.

Antimicrobial Therapy

Appropriate antimicrobial therapy for osteomyelitis depends on the identification of the causative pathogen and sensitivity results because the infection is usually responsive to specific antimicrobial therapy. Mismanagement with inappropriate antibiotic(s) encourages disease extension, sequestra formation, and the development of a refractory infection. Initially, appropriate culture material must be obtained. A bone biopsy is necessary unless the patient has positive blood cultures along with radiographic or bone scan findings consistent with osteomyelitis. While awaiting sensitivity data, a parenteral antimicrobial regimen is begun presumptuously to cover the clinically suspected pathogens. Table 1 outlines initial antibiotic therapy choices. Once the specific organism is identified, the initial antibiotic regimen may be continued or changed on the basis of sensitivity results. The patient is treated for 4 to 6 weeks with appropriate antimicrobial therapy dated from the initiation of therapy or after the last major débridement surgery. The goal of therapy is to prevent a refractory infection. If the initial medical management fails and the patient is clinically compromised by a recurrent infection, medullary and/or soft tissue débridement will be required in conjunction with another 4 to 6 weeks of antibiotics.

Treatment of Childhood Versus Adult Osteomyelitis

Oral antibiotic therapy can be utilized for treatment of childhood osteomyelitis. However, it is recommended that the patient initially receive 2 weeks of parenteral antibiotic therapy before changing to a 4-week oral regimen. In addition, the patient must be compliant and agree to close outpatient supervision. Absorption and activity of the orally administered antibiotic should be monitored by the measurement of the serum bactericidal activity against the causative pathogen. A peak bactericidal dilution of at least 1:8 or greater should be present and maintained. Oral therapy is possible in pediatric hematogenous osteomyelitis because of an increased bone blood flow and the aggressive mesenchymal and immunologic responses found in this age group. However, patients younger than the age of puberty cannot be given oral antimicrobial therapy with the quinolone class of antibiotics. Although acute hematogenous osteomyelitis in children may primarily be a medical disease, in the adult, débridement surgery, intramedullary reaming, unroofing, and incision and drainage of soft tissue abscesses are often required. Surgical intervention is indicated if the patient has not responded to specific antimicrobial therapy within 48 hours, has evidence of a persistent soft tissue abscess, or is diagnosed or suspected of having joint sepsis.

VERTEBRAL OSTEOMYELITIS

Vertebral osteomyelitis in the adult patient population is usually hematogenous in origin but may occur secondarily to trauma. Clinically, the patient usually presents with vague symptoms and signs consisting of dull constant back pain and spasm of the paravertebral muscles. More specific complaints may localize to a soft tissue abscess. The presence of point tenderness over the involved vertebral body is a characteristic finding. Fever may be low grade or absent.

The lumbar vertebral bodies are most often involved, followed in frequency by the thoracic and cervical vertebrae. The infection is usually monomicrobic when hematogenous in origin. In the normal host, *S. aureus* remains the most commonly isolated organism. However, aerobic gram-negative rods are found in 30% of cases. The primary infection focus of a patient with vertebral osteomyelitis is usually unknown. Intravenous drug abuse shows a high incidence of infection by *Pseudomonas aeruginosa* and *Serratia marcescens*. Unusual pathogens such as fungi may also cause hematogenous osteomyelitis.

Therapy

Antimicrobial Therapy

The therapy for vertebral osteomyelitis requires parenteral antibiotics (see Table 2) and may include early surgery and stabilization. On presentation, the patient should be placed on a broad-spectrum antibiotic regimen for the suspected pathogen. The choice of antibiotic(s) should then be refined based on the biopsy or débridement culture and antibiotic sensitivity results. The antibiotic(s) is given for 4 to 6 weeks and is usually dated from the initiation of therapy or from the last major débridement surgery.

Surgical Intervention and Stabilization

Surgical débridement is usually not necessary when the infection is diagnosed early. The failure rate with bed rest alone is not statistically different from that of patients stabilized with a cast, corset, or brace. Open surgical therapy is usually not necessary, except in cases in which the patient develops an extension of the infection, such as paravertebral or epidural abscesses, when medical management fails, or when instability is pending. The decision to advise an orthosis as opposed to internal fixation or bed rest is best individualized. When epidural and paravertebral abscesses develop, surgical drainage (sometimes emergently) must occur. Fusion of adjacent infected vertebral bodies is a major goal of therapy. Surgical stabilization in the form of vertebrae fusion is not recommended in routine cases because spontaneous bony fusion often occurs after 1 to 2 months with appropriate therapy. Other forms of stabilization, such as plaster body casting, are also usually not necessary because neck or body braces or a molded plastic jacket can usually provide adequate stabilization. In cases in which the infection progresses to the point of neurologic defects, emergent surgical intervention and decompression should be performed. Also, the development or progression of bony destruction or failure of patient improvement with antibiotic treatment often warrants the use of surgical débridement and stabilization.

Monitoring Treatment Success

Erythrocyte sedimentation rates should be monitored to gauge the success of treatment. Using scanning techniques may also provide an indication of patient response to therapy. Although radiographic tests can be useful, favorable therapy response often progresses for a number of weeks before it is detected on plain films. Magnetic resonance imaging is clearly superior in the early detection and evaluation of vertebral osteomyelitis. However, computed tomography is very useful in detecting sequestra and guiding bone biopsy for cultures and histology. Radionuclide scans demonstrate excellent sensitivity usually detecting the infectious process within a few days of infection. However, these scans are not specific and often give false-positive results for noninfectious reactive bone formation such as occurs after trauma or surgery. For patients with vertebral osteomyelitis, magnetic resonance imaging is the most beneficial scan, followed by computed tomography and the gallium-67 citrate scan.

CHRONIC OSTEOMYELITIS

Although both contiguous focus and hematogenous osteomyelitis can progress to a chronic bone infection, no precise criteria distinguish acute from chronic osteomyelitis. Clinically, newly recognized bone infections are considered acute, whereas a relapse of a treated infection represents a chronic process. The hallmark of chronic osteomyelitis is the simultaneous presence of organisms, necrotic bone, and a compromised soft tissue envelope. In addition, the pathologic features of chronic osteomyelitis are the presence of necrotic bone and the exudation of polymorphonuclear leukocytes joined by large numbers of lymphocytes, histiocytes, and occasionally plasma cells. Even after years of apparent eradication of an osteomyelitis infection, success is not ensured. A quiescent focus of bacteria can be reactivated years later and cause a recurrence of infection.

New bone formation is another characteristic of chronic osteomyelitis. New bone forms from the surviving fragments of periosteum, endosteum, and cortex in the region of the infection and is produced by a vascular reaction to the infection. The formation may reach from the periosteum and along the intact periosteal and endosteal surfaces, thereby surrounding the dead bone, forming the involucrum. The involucrum is irregularly shaped and contains openings through which pus may permeate into the surrounding soft tissues. This forms a sinus tract that allows the pus to travel from the involucrum to the skin surfaces. The involucrum may gradually increase in density to form part or all of a new shaft. Depending on the size of the bone and the duration of the infection, the amount and density of the new bone may progressively increase for weeks or months. The formation of new endosteal bone may proliferate and obstruct the medullary canal. Once the sequestrum has been surgically removed, the remaining cavity may be filled with new bone, especially in children. However, in adults the cavity may persist, or the space may be filled with fibrous tissue that connects with the skin surface through a sinus tract.

Multiple species of bacteria are usually isolated from biopsy specimens of infected granulations from deep within the wound. Chronic hematogenous osteomyelitis is the exception to this statement because a single organism is often recovered from these patients even after years of intermittent drainage. The possibility of attenuating the infection is reduced when the integrity of the soft tissue surrounding the infection is poor or the bone itself is unstable secondary to an infected nonunion or a septic joint.

Therapy

The infection will not resolve until the nidus for the persistent contamination is removed. Persistent drainage and/or sinus tract(s) are common, and antibiotic therapy alone is usually unsuccessful in the treatment of chronic osteomyelitis. The components of chronic osteomyelitis treatment include patient evaluation, staging assessment, identification and sensitivity of microorganisms, aggressive débridement surgery, prolonged administration of antibiotics, dead space management, and, if necessary, stabilization (see earlier for descriptions). Reconstruction is considered at the first surgery. In chronic osteomyelitis, there are usually large areas of devitalized cortical and cancellous bone within the wound. The dead areas must be completely débrided, including devitalized scar tissue, marrow, and cortex. The soft tissue covering the area of bone trauma must heal. If healing does not occur, the existing infection will persist or a new infection could form. Compromise of local soft tissue is a major reason for continued drainage.

Hyperbaric oxygen (HBO) therapy should only be used as an adjunct to standard surgical and antibiotic treatment. Although clinical studies evaluating HBO have been somewhat limited, its use in the treatment of chronic refractory osteomyelitis is generally supported, and it can play an integral role in the effective management of chronic osteomyelitis under the direction of a hyperbaric medical specialist. Historically, treatment pressures of both 2.0 and 2.4 atmospheres absolute (ATA) have been used, with treatment times varying from 2 hours at the lower pressure to 90 minutes at 2.4 ATA. Treatment pressure and duration are the same for monoplace and multiplace chambers. Now the higher pressure of 2.4 ATA is almost universally used. Because experimental evidence suggests that twice-per-day HBO treatment interfere with bone healing, we suggest that HBO treatment be provided on a once-per-day basis for the treatment of osteomyelitis.

PREVENTION

Most cases of long bone osteomyelitis are developed after trauma or pre/intraoperatively. A number of studies have strongly correlated the development of *S. aureus* osteomyelitis with patient nasal carriage. Therefore, patient treatment must also include the elimination of nasal carriage through topical antimicrobial agents. Patients with diabetes mellitus can prevent osteomyelitis by minimizing foot trauma and preventing foot ulcers. This includes education on proper foot care, including daily inspection of the feet. Daily foot washing and the use of moisturizing with creams are necessary to avoid the opening of the skin. Furthermore, patients should avoid activities that might cause unnecessary trauma to avascular, neuropathic feet. This includes walking barefoot or wearing improperly fitted shoes. The only way to reduce the number of contiguous focus osteomyelitis is to prevent the development of diabetic foot ulcers or aggressively prevent diabetic foot ulcers from involving bone by treatment of infection, wound care, and off-loading.

COMMON SPORTS INJURIES

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Primary care physicians commonly encounter musculoskeletal injuries. Most of these injuries are due to competitive and/or recreational sports injuries. This article covers the general principles involved in managing common injuries seen among adolescent, younger adult, and adult athletes (Table 1).

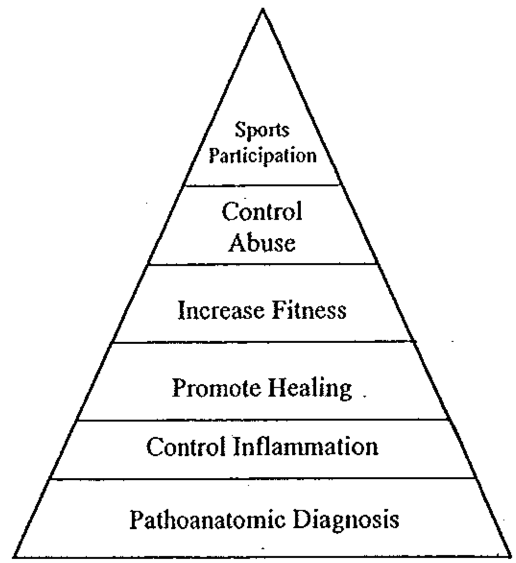

Figure 1. Injury management pyramid.

MANAGEMENT OF ATHLETIC INJURIES

The management of athletic injuries involves a systematic process of diagnosis, rehabilitation, and return to participation. This is accomplished by the application of clinical anatomy with the mechanism of injury. Recognition of all components of an injury allows the clinician to appropriately diagnose and effectively treat an athlete in a stepwise fashion (Figure 1).

The cornerstone of athletic injury management is making an accurate pathoanatomic diagnosis. This requires adept use of a history, physical examination, and selected tests to establish a clear diagnosis. The history attempts to identify the transition that led to the injury. The examination focuses on sorting out victims and culprits. The principle of transition suggests that injury is commonly due to a change in the use of the involved part. The principle of victims and culprits underscores the importance of a biomechanical examination. The presenting injury represents the victim and the subsequent dysfunction represents the culprit (e.g., plantar fasciitis and gastrosoleus inflexibility).

Treatment is initially aimed to control or modify inflammation through protection, rest, ice, compression, elevation, medications, and modalities (PRICEMM). Protection refers to avoiding abusive activity through modifying activity or temporary limb immobilization. Rest alone does not heal, but it allows the body's reparative efforts the opportunity to promote normal healing. Ice is the most effective anti-inflammatory modality used in sports medicine. Ice limits inflammation by causing vasoconstriction and slowing local metabolic activity. An ice cycle, 20