Osteomyelitis in Elderly Patients

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In elderly persons, osteomyelitis is second only to soft-tissue infection as the most important musculoskeletal infection. Acute osteomyelitis is usually acquired hematogenously, and the most common pathogen is Staphylococcus aureus. Acute osteomyelitis can usually be cured with antimicrobial therapy alone. In contrast, chronic osteomyelitis may be caused by S. aureus but is often due to gram-negative organisms. The causative organism of chronic osteomyelitis is identified by culture of aseptically obtained bone biopsy specimens. Because of the presence of infected bone fragments without a blood supply (sequestra), cure of chronic osteomyelitis with antibiotic therapy alone is rarely, if ever, possible. Adequate surgical debridement is the cornerstone of therapy for chronic osteomyelitis, and cure is not possible without the removal of all infected bone.

Osteomyelitis is a common infectious disease among elderly patients. Older adults are predisposed to osteomyelitis either because of an increased incidence of associated disorders that predispose to osteomyelitis (e.g., peripheral vascular disease, diabetes mellitus, and poor dentition) or because of surgical procedures that are frequently performed in the elderly population (e.g., dental extractions, open-heart surgery, and prosthetic joint replacement). As with osteomyelitis in other age groups, osteomyelitis in the elderly population may also be considered in terms of acuteness of the infectious process (acute osteomyelitis, subacute osteomyelitis, or chronic osteomyelitis). Osteomyelitis may be caused by a variety of microorganisms, but osteomyelitis in the elderly population is most often caused by pyogenic organisms [1-6] (table 1).

ACUTE OSTEOMYELITIS

General concepts. Acute osteomyelitis is an infection of the bone that involves the periosteum, cortex, and/or medullary cavity. Elderly persons frequently fall, which may result in closed or open bone trauma. Acute osteomyelitis secondary to closed trauma is usually due to Staphylococcus aureus. Acute osteomyelitis may be acquired hematogenously after closed trauma. In acute hematogenous osteomyelitis, the bacteria reach the metaphyseal blood vessels of bone to initiate the infectious process. Pathogenic bacteria in the smaller arterioles of the metaphyses multiply, which leads to microabscess formation. The abscess formation in acute osteomyelitis within the medullary cavity of bone, metaphyseal space, or subperiosteal space leads to further bone necrosis because of increased pressure. Eventually, bone fragments are formed (sequestra) that are, in effect, floating fragments of infected dead bone without a blood supply. If the process becomes chronic, extensive bone destruction occurs, which may be accompanied by fistula formation [1, 6].

Clinical presentation. Patients with acute osteomyelitis present with pain in the affected bone. There may be point tenderness over the bone if there is subperiosteal involvement. In addition to local tenderness, patients may have systemic symptoms, such as fever or chills. Acute osteomyelitis may be defined as a first episode of osteomyelitis occurring in a patient that is cured by medical means in <6 weeks.

The presumptive diagnosis of acute osteomyelitis is clinical and is confirmed by bone scan. In acute osteomyelitis, soft-tissue or periosteal elevations are the first changes, followed in 10–12 days by periosteal proliferation and by irregular bone reabsorption in 3 weeks. Bone sclerosis occurs months later. The erythrocyte sedimentation rate (ESR) is often elevated (=100 mm/h) in patients with acute osteomyelitis, particularly in those with vertebral osteomyelitis. If acute osteomyelitis is hematogenously acquired, blood cultures may yield positive results early in its course. Plain films are unhelpful diagnostically in the early phases of acute hematogenous osteomyelitis. Bone scans yield positive results within the first 2 or 3 days in...
<table>
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<th>Type of osteomyelitis, related disorder</th>
<th>Usual pathogen(s)</th>
<th>Means of diagnosis</th>
</tr>
</thead>
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<td><strong>Acute</strong></td>
<td></td>
<td></td>
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<tr>
<td>Open trauma</td>
<td><em>Staphylococcus aureus</em>, aerobic gram-negative bacilli</td>
<td>Clinical finding of exposed infected bone</td>
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<td>Infected joint replacement-associated osteomyelitis</td>
<td><em>S. aureus, Staphylococcus epidermidis, coagulase-negative staphylococci</em></td>
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<td>Foot osteomyelitis associated with peripheral vascular disease</td>
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<td>Foot osteomyelitis associated with diabetes mellitus</td>
<td><em>S. aureus</em>, group B streptococci, aerobic gram-negative bacilli, <em>B. fragilis</em></td>
<td>Bone scan, CT, MRI, measurement of ESR</td>
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</table>

**NOTE.** ESR, erythrocyte sedimentation rate.
acute osteomyelitis and are the preferred diagnostic test. Gallium scans also yield positive results and are useful, but indium scans often yield false-negative results [2–5].

**Therapy.** Acute osteomyelitis is usually treated empirically on the basis of the pathogens most likely to cause osteomyelitis, or therapy is based on the pathogens identified from culture material obtained from bone (in open-fracture cases) or blood (in cases due to closed extremity trauma). The selected antibiotic therapy should be active against *S. aureus* and methicillin-susceptible *S. aureus*—for example, antistaphylococcal penicillin, antistaphylococcal cephalosporins, clindamycin, or vancomycin. If the pathogen is methicillin-resistant *S. aureus*, then vancomycin or linezolid may be used. The antibiotic selected should also penetrate the bone in adequate concentration to eradicate the organism. Most antistaphylococcal antibiotics given in the usual recommended dose penetrate bone adequately. Oral antibiotics with good bioavailability and the same or equivalent spectrum of parenteral agents may also be used to treat acute osteomyelitis or to complete a course initiated with parenteral antibiotics [1, 6, 7].

**SUBACUTE OSTEOMYELITIS**

**Vertebral Osteomyelitis**

**General concepts.** Subacute osteomyelitis in the elderly population is most commonly due to vertebral osteomyelitis or osteomyelitis associated with prosthetic joint replacement. Vertebral osteomyelitis may occur through hematogenous dissemination from a distant infected source. Less commonly, in men with urinary tract infections, aerobic gram-negative bacilli may ascend via Batson’s plexus and reach the lumbar spine. The vertebral bodies are not readily accessible to physical examination, and the patient’s symptoms or complications (e.g., neurological or supportive) may suggest the diagnosis. Rarely, vertebral osteomyelitis may occur iatrogenically as a complication of disk-space injections or spinal surgery.

An initial clinical clue to vertebral osteomyelitis may be muscle spasm, which is nonspecific and may occur with a variety of other disorders. Physical or neurological findings are associated with complications, such as perispondylitic or epidural abscess. Pyogenic vertebral osteomyelitis must be differentiated from tuberculous spinal osteomyelitis, which is also common among elderly patients. In general, both pyogenic and tuberculous vertebral osteomyelitis destroy adjacent vertebral bodies and involve the disk space. Factors that favor a pyogenic etiology of vertebral osteomyelitis include a rapid rate of disk-space destruction, a lower incidence of abscess formation, less osteoporosis or sclerosis, and it is usually limited to a single disk space or adjoining vertebra. In contrast, tuberculous osteomyelitis has a slow and indolent process with a higher incidence of abscess formation. The abscess may extend to the psoas muscle and dissect to the groin and present as a groin mass. Typically, but not always, tuberculous osteomyelitis involves >2 contiguous vertebral bodies or disk spaces [8–11].

**Clinical presentation.** In cases of vertebral osteomyelitis secondary to hematogenous dissemination, *S. aureus* is the most common pathogen. Aerobic gram-negative uropathogens are associated with vertebral osteomyelitis in elderly men with urinary tract infections that have reached the vertebral axis via Batson’s plexus. Spinal tuberculosis is a reactivation of initial hematogenous dissemination of *Mycobacterium tuberculosis* during initial infection. *M. tuberculosis* localizes in areas of high oxygen tension, such as the vertebral bodies. Normal host defense mechanisms contain the organisms until old age, when osteoporosis, trauma, or nonmycobacterial infections cause reactivation of latent infection in bone.

The diagnosis of vertebral osteomyelitis is suspected clinically, and the presumptive diagnosis is made on the basis of radiographic findings. In pyogenic vertebral osteomyelitis, culture of blood samples frequently yields negative results. Plain films of the spine usually lack sufficient definition to differentiate tuberculous from pyogenic vertebral osteomyelitis or from other noninfectious processes involving the spine, such as sarcoidosis, hemangiomas, or tumors. Bone scans, and, to a lesser extent, gallium scans, are useful to localize the process, but they are not always helpful in differentiating tumor from tuberculosis or from a pyogenic process. The diagnosis of tuberculous osteomyelitis is favored by finding evidence of tuberculosis elsewhere (e.g., pulmonary tuberculosis). Although anergy is common with increasing age, 95% of patients with vertebral osteomyelitis have a positive result of tuberculin purified protein derivative skin tests. A pulmonary focus of tuberculosis is present in ~90% of patients with tuberculous vertebral osteomyelitis. The ESR is elevated, but this does not help the examiner differentiate between tumor, tuberculosis, or pyogenic osteomyelitis. Although the process in the vertebral bodies can be localized by noninvasive radiological procedures, a definitive etiologic diagnosis can usually only be made by CT-guided biopsy or open biopsy of the infected bone or disk space [12–15].

**Therapy.** Treatment depends on the causative organism. Vertebral osteomyelitis due to *S. aureus* is treated as staphylococcal osteomyelitis in other anatomic locations, and the duration of treatment is 4–6 weeks. Surgical debridement may or may not be necessary, depending on the degree of bone destruction. Antibiotic selection for aerobic gram-negative bacilli should be based on the susceptibility of the isolate recovered from a septic blood culture. The duration of treatment is ordinarily 4–6 weeks. If the patient has been found to have tuberculous vertebral osteomyelitis, treatment is with double-drug antituberculosis therapy (e.g., isoniazid and rifampin), unless a drug-resistant strain is present. If that is the case, 3
or 4 antituberculous drugs will be necessary and therapy should be for 6–12 months [7, 16, 17].

**Osteomyelitis with Joint Replacement**

**General concepts.** Some elderly persons develop osteomyelitis following total hip replacement or total knee replacement. Patients with rheumatoid arthritis are immunocompromised hosts and are predisposed to a higher incidence of prosthetic joint–related infections than are patients with osteoarthritis [18–20].

**Clinical presentation.** Osteomyelitis associated with infected prostheses in total hip replacement or total knee replacement presents with loosening of the prosthesis and with little or no fever. Prosthetic loosening may be mechanical or may be a sign of associated osteomyelitis. The patient’s ESR is not elevated if loosening of the prosthesis is the cause of joint instability or periprosthetic lucencies are visible on plain film radiographs, and it is elevated if these findings are because of prosthetic-associated osteomyelitis. Radiological imaging techniques, such as bone and gallium scans, are useful in differentiating simple mechanical loosening from prosthetic-related infection [7, 21, 22].

**Therapy.** If the prosthesis and surrounding bone are infected, then cure necessitates removal of the prosthesis and debridement of all involved bone, followed by appropriate antimicrobial therapy. In replacing a prosthetic device, adequate debridement of the surrounding infected bone is essential for cure. There are no good data on the duration of antimicrobial therapy after reimplantation of the new joint prosthesis [7, 23, 24].

**CHRONIC OSTEOMYELITIS**

Chronic osteomyelitis may be defined as osteomyelitis that has a duration of ≥6 weeks or as osteomyelitis that recurs or is not cured after the initial infection. Chronic osteomyelitis is often accompanied by fistula formation to the skin surface. Chronic osteomyelitis is, by definition, an indolent, slow process with few systemic symptoms. Chronic osteomyelitis has been associated with performance of certain surgical procedures (e.g., sternal osteomyelitis after open-heart surgery), has occurred secondary to poor dentition or dental extraction (mandibular osteomyelitis), and, more commonly, has been associated with systemic disorders (e.g., peripheral vascular disease and diabetes mellitus). Far and away the most common problem in the elderly population is chronic osteomyelitis due to peripheral vascular disease or diabetes mellitus.

**Sternal Osteomyelitis**

**General concepts.** Sternal osteomyelitis complicates any open-heart surgical procedure that involves dividing the sternum. Bone wax and stainless steel sutures are used, which may become a nidus for bacteria acquired via the wound (e.g., gram-positive cocci) or from irrigant solutions (e.g., aerobic gram-negative bacilli) during the thoracic surgical procedure. The organisms most commonly associated with sternal osteomyelitis are *S. aureus* and aerobic gram-negative bacilli. *S. aureus* may gain access to the wound via the skin during the surgical procedure. Aerobic gram-negative bacilli may be the cause of infection if the irrigation fluids are colonized by these organisms [3, 4, 7].

**Clinical presentation.** Clinically, sternal osteomyelitis presents weeks or months after open-heart surgery, as either a flail sternum or as a draining sinus tract. The draining sinus tracts in patients with sternal osteomyelitis most commonly occur at the styloid process or at the top of the incision. Cracking sounds on chest palpation or instability of chest wall segments suggests sternal osteomyelitis until proven otherwise. In the diagnosis of sternal osteomyelitis, bone scans and CT or MRI are usually unhelpful.

**Therapy.** Sternal osteomyelitis is diagnosed clinically, and the definitive treatment is surgical with adjunctive antimicrobial therapy. Because the involved segments are devitalized and have no blood supply, thorough surgical debridement is necessary to effect cure in sternal as well as other forms of chronic osteomyelitis. If the debridement is inadequate, osteomyelitis will eventually become clinically manifest again at the sites of inadequate surgical debridement. Antimicrobial therapy is adjunctive but is directed against *S. aureus*, coagulase-negative staphylococci, or aerobic gram-negative bacilli that are aseptically cultured from the debrided, surgically removed bone specimens [7, 25, 26].

**Mandibular Osteomyelitis**

Mandibular osteomyelitis occurs in elderly patients with poor dentition or periodontal disease. Periapical abscesses are common in the elderly population and may present with local symptoms or those of intracranial mass lesions. Hematogenous spread to the brain may be clinically expressed as CNS mass lesions resembling a neoplasm. It is difficult to differentiate CNS tumors from abscesses, even with MRI or CT imaging studies, and often only craniotomy and examination of biopsy specimens can differentiate these clinical entities. Local extension of a periapical abscess may result in mandibular osteomyelitis. The organisms involved in mandibular osteomyelitis are those of the oropharyngeal anaerobic flora. Such organisms as *Actinomyces, Eikenella*, and *Peptostreptococcus* species are commonly isolated from dental abscesses associated with mandibular osteomyelitis. Patients present with a swelling or tenderness of the jaw, regional adenopathy, and low-grade fever. Blood culture results are usually negative. Plain film or panorex radiographs of the jaw are usually diagnostic. Nuclear scanning...
or CT imaging is usually unnecessary. Treatment of mandibular osteomyelitis involves removal of the diseased tooth and root along with debridement of the involved mandibular bone [3, 4, 27, 28].

Chronic Osteomyelitis Associated with Decubitus Ulcers
Elderly patients who are unable to turn themselves over in bed frequently develop pressure sores or decubitus ulcers. Pressure (usually over a bony prominence) may result in a decubitus ulcer. The severity and depth of a decubitus ulcer depends on the duration of pressure on the skin over a bony prominence. Decubitus ulcers are staged by severity and depth. Superficial decubitus ulcers (i.e., stage 1 or 2) may be treated locally and, being superficial, are not associated with osteomyelitis. Decubitus ulcers of the deepest variety (i.e., stage 3 or 4) are often complicated by osteomyelitis. Chronic osteomyelitis is usually present when bone is visible in a long-standing deep decubitus ulcer. The organism responsible for osteomyelitis associated with decubitus ulcers depends on the location of the ulcer. Decubitus ulcers on the extremities, above and below the waist, are usually due to *S. aureus*. Decubitus ulcers involving the sacrum in the perianal area are usually due to *S. aureus* or organisms of the fecal flora, such as *Bacteroides fragilis*.

Although superficial decubitus ulcers may be managed with local care and do not require systemic antibiotics, stage-3 decubitus ulcers often require administration of systemic antibiotics for control of local extension of infection. However, stage-4 decubitus ulcers associated with osteomyelitis require appropriate antimicrobial therapy as well as adequate surgical debridement for cure. Culture of blood samples usually yields negative results in cases of sacral osteomyelitis associated with decubitus ulcers. The patient’s ESR is frequently elevated. The definitive diagnosis is achieved by means of bone scan or imaging studies with CT or MRI. It is often not possible to obtain an aseptic bone sample for culture during the surgical debridement procedure because of fecal contamination with sacral osteomyelitis. For this reason, patients with sacral osteomyelitis from a decubitus ulcer are treated empirically for *S. aureus*, aerobic gram-negative bacilli, and *B. fragilis*. Debridement is often superficial or inadequate, resulting in persistence of chronic osteomyelitis. After bone debridement, plastic surgical flap procedures are useful in covering large soft-tissue defects [29–31].

Chronic Osteomyelitis Associated with Peripheral Vascular Disease
Elderly persons with peripheral vascular disease are at risk for developing peripheral gangrene as well as osteomyelitis. Osteomyelitis due to peripheral vascular disease usually involves the digits of the foot. The extent and distribution of the osteomyelitis is related to the distribution and extent of underlying macular vascular compromise. Osteomyelitis due to peripheral vascular disease is related to an inadequate macrovascular blood supply. These patients clinically present with cool or cold extremities, often accompanied by gangrenous changes in 1 or more toes. Diagnosis of osteomyelitis is confirmed as it is with other forms of osteomyelitis.

Treatment of peripheral vascular disease associated with osteomyelitis depends on adequate debridement of the involved bone or gangrenous soft tissue, as well as restoration of the blood supply to the involved extremity. Vascular bypass procedures are important to prevent extension or recurrence in the involved extremity and are particularly useful for patients without diabetes mellitus. Antimicrobial therapy is directed against skin pathogens is adjunctive and surgical therapy is primary [32, 33].

Chronic Osteomyelitis Associated with Diabetes Mellitus
General concepts. Elderly patients with osteomyelitis of the feet present with deep ulcers, usually involving the plantar surface of the foot, or, less commonly, with ulcers between the toes (table 2). In contrast, other patients present without an ulcer but with a chronic draining sinus tract. Patients with diabetes who have chronic, deep-penetrating foot ulcers and/or a chronic draining sinus tract of the foot should be considered as having chronic osteomyelitis until proven otherwise. Studies indicate a high positive correlation between these conditions and chronic osteomyelitis on bone scan [31, 32].

Clinical presentation. Patients with diabetes mellitus and chronic osteomyelitis of the feet have few systemic symptoms and slight or no fever. Blood cultures are usually not positive. Because of diabetic neuropathic changes, pain is usually absent. Peripheral pulses in the absence of associated peripheral vascular disease are usually normal. Diabetes mellitus is a microvascular disease, and even in the presence of accelerated atherosclerosis, the peripheral pulses, particularly the dorsalis pedis pulses, are almost always normal. Determination of a highly elevated ESR (≥100 mm/h) is an inexpensive but nonspecific presumptive test for patients who present with a penetrating foot ulcer or chronic draining sinus tract due to chronic osteomyelitis. Definitive diagnosis is achieved by means of plain film radiography of the foot. Because chronic osteomyelitis is, by definition, a long-standing process, these patients always have changes indicative of chronic osteomyelitis on plain films. Bone scans are not needed unless there are other diagnostic possibilities in particular cases. The organisms most commonly associated with chronic foot osteomyelitis in patients with diabetes mellitus include *S. aureus*, group B streptococci, aerobic gram-negative bacilli, and *B. fragilis*. Importantly, *Pseudomonas aeruginosa* is not associated with chronic foot osteomyelitis in diabetes mellitus. *P. aeruginosa* is often cultured from samples of open ulcers or draining sinus tracts, but this is indicative of
Fever, temperature Variable may occur years later in untreated patients. Similarly, amyloidosis at the site of the ulcer or sinus tract osteomyelitis, over time, may be complicated by systemic non-

antimicrobials is an alternative approach. However, chronic refused, long-term suppressive therapy with orally administered surgical debridement or amputation of the extremity. In cases foot infections in persons with diabetes depends on adequate the infection. The definitive treatment of chronic osteomyelitis depends primarily on the adequacy of surgical debridement, and appropriate antimicrobial therapy is adjunctive [7, 34, 37–40].

**Table 2. Foot infections in patients with diabetes mellitus.**

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Cellulitis</th>
<th>Deep, soft-tissue infection/fasciitis</th>
<th>Chronic osteomyelitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usual pathogens</td>
<td><em>Streptococcus</em> (group A, B, C, G), <em>Staphylococcus aureus</em></td>
<td><em>S. aureus</em>, streptococci, coliform bacilli, <em>Bacteroides fragilis</em></td>
<td><em>S. aureus</em>, streptococci, coliform bacilli, <em>B. fragilis</em></td>
</tr>
<tr>
<td>Fever, temperature</td>
<td>Variable</td>
<td>&gt;38.9°C (≥102°F)</td>
<td>&lt;38.9°C (&lt;102°F)</td>
</tr>
<tr>
<td>Wound appearance</td>
<td>Red, tender, warm</td>
<td>Extremely tender, warm</td>
<td>Erythema, swelling, not warm (fever and other symptoms may not be apparent except during flare)</td>
</tr>
<tr>
<td>Drainage</td>
<td>None</td>
<td>Foul</td>
<td>Purulent</td>
</tr>
<tr>
<td>Crepitance</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>WBC count</td>
<td>Elevated or normal</td>
<td>Elevated</td>
<td>Normal</td>
</tr>
<tr>
<td>Findings on plain film</td>
<td>No gas</td>
<td>Gross gas in the soft tissues</td>
<td>Signs of bone destruction</td>
</tr>
<tr>
<td>Treatment</td>
<td>Antibiotics effective against streptococci and staphylococci, such as antistaphylococcal penicillins or cephalosporins</td>
<td>Surgical debridement plus appropriate antibiotics, such as meropenem or piperacillin-tazobactam or a quinolone plus either clindamycin or metronidazole</td>
<td>Surgical debridement plus appropriate antibiotics, such as meropenem or piperacillin-tazobactam or a quinolone plus either clindamycin or metronidazole</td>
</tr>
</tbody>
</table>

**NOTE.** Adapted from [34].
References
