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Osteomyelitis

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(بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ)

(يَرْفَعُ اللَّهُ الَّذِينَ آمَنُوا مِنْكُمْ وَالَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ وَاللَّهُ بِمَا تَعْمَلُونَ خَبِيرٌ)

صَدَقَ اللَّهُ الْعَظِيمُ



إلى من سالت بسببها دماء زكية من أجل رب الأرض والسموات
ومن أجلها قضى رجالاً في زهرة شبابهم لتحيا بالتضحيات
إلى أرض بلادي العراق الحبيب

إلى من علمني العطاء دون إنتظار ، إلى من أحمل إسمه بكل إفتخار .. وأسأل الله
أن يمد في عمره ليرى ثماراً قد حان قطفها بعد طول إنتظار ، والذي العزيز
إلى أجمل معاني الحياة ومعنى الحب والحنان إلى بسمه حياتي وسر الوجود الذي
كان بدعاؤها سر نجاحي وحنانها بلسم جراحي ، أمي الحبيبة
إلى من شاركوني حزن أمي و أبي وبهم أستمد عزوتي وإصراري أخوتي الأعزاء إلى
من زادني في العلم بهجة وسخر ومد لي بوقته كي أنال بالعلم طريق القمم إلى
الأستاذ المشرف حفظه الله و أساتذتي الأعزاء جميعهم

Abstract

Patients with bone and joint infections endure excruciating pain, and both they and their physicians find it aggravating. Because of the physiological and anatomical properties of bone, bone and joint infections have not yet seen the high success rates of antimicrobial treatment seen in the majority of infectious disorders. Early diagnosis, which includes bone biopsy for microbiological and pathological analysis to enable tailored and long-lasting antimicrobial therapy, is essential for effective care. The different forms of osteomyelitis call for different approaches to medical and surgical treatment. These forms include osteomyelitis due to a contiguous center of infection (after trauma, surgery, or installation of a joint prosthesis); osteomyelitis secondary to vascular insufficiency (in infections of the diabetic foot); and osteomyelitis of haematogenous origin, listed in decreasing order of incidence. Avascular bone necrosis and the development of sequestrum, or dead bone, are linked to chronic osteomyelitis, and surgical debridement is required in addition to antibiotic treatment to treat the condition. On the other hand, antibiotics by themselves can treat acute osteomyelitis. Success often requires a multidisciplinary approach incorporating knowledge of infectious diseases, plastic surgery, orthopaedic surgery, and vascular surgery, especially in complicated cases requiring soft-tissue damage.

Introduction

A bacterium that causes osteomyelitis is an inflammatory condition that also destroys bone. The infection may affect several areas of the bone, including the periosteum, cortex, marrow, and surrounding soft tissue, or it may be restricted to a single section. Differentiating between the three forms of osteomyelitis is helpful from a practical standpoint. (1)

Osteomyelitis following trauma, bone surgery, or joint replacement resulting from localized transmission from a contiguous contaminated source of infection. It suggests a primary infection that penetrates the bone.

Any bone may be affected, and it can happen at any age. The identification of patients with foreign-body implants is crucial in this group due to therapeutic issues as well as their increased vulnerability to infection. (2)

The majority of instances of osteomyelitis related to vascular insufficiency are associated with diabetes, and virtually all of them begin with a soft-tissue infection in the foot that progresses to the bone. The metabolic aftereffects of diabetes; bone and soft-tissue ischaemia; and peripheral motor, sensory, and autonomic neuropathy are some of the major contributing elements to this disease entity. (3)

Haematogenous osteomyelitis is primarily observed in prepubescent children and elderly people. It is typified by the nidation of bacteria within occasionally very slightly wounded bone, which is apparently seeded by blood-borne germs that are not readily visible.(4)

Unlike chronic osteomyelitis, which is loosely defined as a long-lasting infection that develops over months or even years and is characterized by the persistence of microorganisms, low-grade inflammation, the presence of dead bone (sequestrum), and fistulous tracts, acute osteomyelitis progresses over a few days or weeks (figure 1).Relapses in the same location and in conjunction with a fever are unmistakably indicative of persistent osteomyelitis. Chronic osteomyelitis and the emergence of necrotic bone are linked to clinical symptoms that last longer than ten days.(5,6)

For orthopaedic surgeons caring for patients with chronic osteomyelitis, Cierny and Mader created a comprehensive categorization system that works well for long, massive bones and is less helpful for fingers, tiny bones, or the skull.It integrates the host's physiology and both phases of anatomical illness.(7)

Aims of the study

The aim of the study was to identify osteomyelitis definition, mechanism of disease, diagnostic procedure, management.

Mechanism of osteomyelitis

When the acute osteomyelitis region is examined under a microscope, it displays an acute suppurative inflammation that contains embedded bacteria or other microorganisms. The breakdown of bone trabeculae and bone matrix, as well as tissue necrosis, are caused by a variety of inflammatory agents and leucocytes. The inflammatory process compresses and obliterates vascular pathways, and the ensuing ischaemia further exacerbates bone necrosis.(8)

Bone segments that lack blood flow may split apart to generate sequestra and may retain germs even after antibiotic therapy. Because inflammatory cells and antibiotics cannot penetrate this avascular region, osteomyelitis cannot be effectively treated medically. Reactive hyperemia, linked to elevated osteoclastic activity, is seen at the infarction edge.(9)

Osteoporosis and bone loss are the results of this exercise. As this is going on, bone apposition—which can lead to periodontal apposition and the creation of new bone—occurs, sometimes wildly.

Numerous studies have demonstrated the regulation of osteoblast and osteoclast proliferation and activity by growth factors, cytokines, hormones, and medications. In addition to influencing the growth and death

of normal osteoclasts and osteoblasts, growth factors and cytokines also exhibit changed local concentrations during infection.(10,11)

Diagnostic procedure

Laboratory studies

Even in cases of illness, the white blood cell count may remain normal, indicating that it is not a trustworthy predictor. Most of the time, the erythrocyte sedimentation rate is high, but its kinetics make it difficult to monitor in instances of osteomyelitis. For monitoring the response to therapy, the concentration of C-reactive protein, which the liver produces in reaction to any infection, seems to be more dependable.(12)

In most cases, the concentration returns to normal one week after receiving appropriate therapy, having increased within hours of the infection. But for reasons other than osteomyelitis, both the C-reactive protein level and the erythrocyte sedimentation rate may be greater than usual.(13)

Calcium, phosphate, and alkaline phosphatase concentrations are typical in osteomyelitis, but not in metastatic or some metabolic bone disorders.(14)

Imaging procedure

A range of imaging techniques are used to diagnose bone infections, however both presentation and follow-up need conventional radiography. Plain films exhibit periosteal response, bone degradation, joint space narrowing or broadening, and soft-tissue edema. On plain films, however, bone damage is not seen until 10–21 days after infection.(15)

When diagnosing acute osteomyelitis early on or identifying a purulent accumulation in soft tissue, ultrasonography might be helpful.(16)

With their superior resolution power, both CT and MRI can show soft-tissue involvement, articular injury, cortical destruction, periodontal response, and medulla destruction even in cases when conventional radiographs show normal.(17)

Although bone or metal artifacts might cause picture deterioration, CT is still helpful for guiding needle biopsy procedures. MRI is most helpful for early infection identification since it also shows early bone oedema.(18)

For bone scintigraphy, several radiopharmaceuticals are now in use. When it comes to identifying acute osteomyelitis early on, methylene

diphosphonate has a high degree of sensitivity since it binds to regions of enhanced bone metabolic activity.(19)

Although it has been shown to have high sensitivity and particularly specificity for infection imaging, leucocyte scanning—using radiolabelled blood cells (leucocytes or granulocytes labelled with indium-111 or technetium-99m) or specific antibodies—is a technique that is less frequently employed.(20)

When fluorine18-fluoro-D-deoxyglucose (FDG) is used in positron emission tomography (PET), inflammatory cells (leucocytes and macrophages) take up the agent. When paired with a CT scan, FDG PET seems especially promising for identifying lesions and any concurrent infectious or inflammatory processes.(21)

Histopathology

Isolating the causing organisms is the most crucial stage in treating any type of osteomyelitis so that the right antibiotic therapy may be selected. Blood cultures, which are often exclusively used in cases of hemorrhagic osteomyelitis, or direct bone biopsies from the affected area can also be used to achieve isolation.(22)

Although some patients require regional or general anesthesia for bone biopsy procedures, the significance of these procedures cannot be overstated. Sampling material from an open sinus canal may contain non-pathogenic bacteria that are colonizing the location, which might lead to false findings. Samples from bone biopsies should always be prepared for both aerobic and anaerobic cultures.(23)

If the clinical symptoms are suitable and there are no routinely cultivated microorganisms, samples for mycobacterial and fungal cultures should be obtained and processed. For the best diagnostic result in implant-associated infections, deep specimens should be taken during debridement from up to five locations surrounding the device. Important tissue specimens for histopathology are those acquired by frozen sections after surgery or by

biopsy, as a considerable quantity of neutrophils is suggestive of infection.(24)

A high-power field containing five or more neutrophils is indicative of infection; this has a 43–84% sensitivity and a 93–97% specificity. An early diagnosis of mycobacterial infection is made when granulomatous lesions with positive Ziehl-Neelsen staining are visible, rather than waiting for culture findings.(25)

Medical management for osteomyelitis

Treatment of osteomyelitis

It has been effectively utilized to avoid wound infections following surgery for noncomplicated hip fractures by administering antibiotic prophylaxis. as well as in the positioning of complete knee and hip prosthesis. Depending on the type of joint replacement, standard preoperative procedures (such as antimicrobial showers, shaving, and soap-disinfectants), the use of surgical rooms with laminar air flow, and prophylactic antibiotic treatment have reduced the infection rate following prosthesis placement to 0.5-2.0%.(26,27)

Intravenous antibiotics should be given to patients having clean bone surgery starting 30 minutes before to the skin incision and ending no later than 24 hours following the procedure. Antistaphylococcal penicillins, first-generation (cefazolin) or second-generation (cefamandole and cefuroxime) cephalosporins reduce the incidence of postoperative infection in closed fracture surgery.(28)

Open fractures are an exception. Antibiotics or first- or second-generation cephalosporins should be administered for 24 hours to patients who can get them within 6 hours of damage and who receive timely surgical care. If a postoperative infection is found, antimicrobial therapy should be followed by careful monitoring, treatment with the proper antibiotics, and surgery. Longer durations of more comprehensive antibiotic treatment are necessary for complex fractures with significant soft-tissue injury.(29)

In all cases of osteomyelitis, a combined antimicrobial and surgical approach should be taken into consideration. While surgery is typically not necessary for haematogenous osteomyelitis, at the other end of the disease spectrum (such as an infected fracture), minimal antibiotic treatment may be sufficient to cure the condition as long as necrotic bone and foreign material are removed. In any event, potential problems such as the

development of a subcutaneous, subperiosteal, or intramedullary abscess, the creation of sequestra, or the presence of foreign material should be taken into consideration if indications of infection do not go away after a week of antibiotic medication.(30)

Surgery often resolves this issue more completely than switching to a different antibiotic, if sufficient microbiological data have produced the right kind of first antimicrobial treatment.(31)

Selection of antimicrobial therapy and routes of administration

For the majority of instances of acute osteomyelitis, antibiotic therapy is sufficient. Figure 1 provides a standard selection of antibacterial drugs for the most prevalent pathogens. With the exception of infections of prosthetic joints, which are often treated with an antibiotic combination that includes rifampicin, and persistent osteomyelitis, single-agent antimicrobial therapy is typically sufficient for treating osteomyelitis. These antibiotics should generally be used for 4-6 weeks, preferably intravenously. When quinidines are taken, it is advisable to transition to oral medication as soon as possible.(32)

Microorganisms isolated	Treatment of choice	Alternatives
Penicillin-sensitive <i>Staph aureus</i>	Benzylpenicillin (12–20 million units daily)	Cefazolin (1 g every 6 h) Clindamycin (600 mg every 6 h) Vancomycin (1 g every 12 h)
Penicillin-resistant <i>Staph aureus</i>	Nafcillin* (1.0 or 1.5 g every 4–6 h) or cefazolin (2 g every 8 h)	Second-generation cephalosporin (eg, cefuroxime, cefamandole) Clindamycin (600 mg every 6 h) Vancomycin (1 g every 12 h) Ciprofloxacin (750 mg orally every 12 h) or levofloxacin in combination with rifampicin (600 mg per day) is also frequently used
Meticillin-resistant <i>Staph aureus</i> †	Vancomycin (1 g every 12 h)	Teicoplanin (400 g every 24 h, first day every 12 h)
Various streptococci (group A or B β-haemolytic; <i>Strep pneumoniae</i>)	Benzylpenicillin (12–20 million units daily)	Clindamycin (600 mg every 6 h) Erythromycin (500 mg every 6 h) Vancomycin (1 g every 12 h)
Enteric gram-negative bacilli	Quinolone (eg, ciprofloxacin, 400–750 mg every 12 h, with early switch to oral)	Third-generation cephalosporin (eg, ceftriaxone 2 g every 24 h; cefepime)
<i>Serratia</i> sp; <i>Pseudomonas aeruginosa</i>	Piperacillin‡ (2–4 g every 4 h) and aminoglycosides	Cefepime 2 g every 12 h‡ or a quinolone and aminoglycosides (according to sensitivity, one daily dose)
Anaerobes	Clindamycin (600 mg every 6 h)	Ampicillin-sulbactam (2 g every 8 h) Metronidazole for gram-negative anaerobes (500 mg every 8 h)
Mixed infection (aerobic and anaerobic microorganisms)	Ampicillin-sulbactam (2–3 g every 6–8 h)§	Imipenem (500 mg every 6 h)¶

All doses are given for normal renal/hepatic function and should be adjusted in renal or hepatic failure. Treatment is intravenous unless stated otherwise. Teicoplanin is presently available only in Europe. *Flucloxacillin, oxacillin, or cloxacillin in Europe. †Most coagulase-negative staphylococci are meticillin-resistant and treated with vancomycin or teicoplanin. ‡Depends on sensitivities; a fourth-generation cephalosporin (cefepime), piperacillin/tazobactam, meropenem, and imipenem are useful alternatives. §Amoxicillin-clavulanate in Europe (1.2–2.2 g every 6–8 h). ¶In cases of aerobic gram-negative microorganisms resistant to amoxicillin-clavulanate.

Figure 1: Antibiotic treatment of osteomyelitis.

Clindamycin is advised for long-term oral therapy in infections with susceptible organisms, either alone or in combination, because to its exceptional bone penetration and oral bioavailability. The fluoroquinolones have been more well-known in recent years due to their superior oral absorption; they are effective against some adult infections as well as infections in experiments. Controlled trials have not yet demonstrated a benefit over conventional therapy for infections with *Pseudomonas aeruginosa*, *Serratia* spp., or *Staph aureus*, despite its undeniable effectiveness against Enterobacteriaceae.(33)

For Staph aureus-related osteomyelitis, a prolonged course of parenterally given semisynthetic penicillin or vancomycin is now advised as a therapy. However, there is a chance that this treatment will result in long-term hospital difficulties, adverse events from using intravenous catheters, and more financial and personal expenses from the prolonged hospital stay. Numerous studies have demonstrated the effectiveness of oral rifampicin therapy for bone staphylococcal infections in the presence of implants or prosthetic joints when combined with ciprofloxacin, ofloxacin, or fusidic acid. We stress that only organisms that are sensitive to both of these drugs should be treated with rifampicin in addition to quinolones.(34,35)

The combination of rifampicin and quinolone has the primary benefit of having great bioavailability, allowing for oral delivery, and achieving serum concentrations comparable to those reached with intravenous treatment. Furthermore, both medications exhibit strong intracellular penetration and action against biofilm-associated and intracellular staphylococci.(36)

Surgical management of osteomyelitis

Chronic osteomyelitis

Surgery is usually necessary to completely remove chronic osteomyelitis.

A sustainable vascularized environment and the removal of dead bone, which functions as foreign material, are the two main objectives of surgery.

In many situations, radical debridement down to live bone is necessary to accomplish this goal. One reason for the high recurrence rates of chronic osteomyelitis is inadequate debridement. Therefore, sequestra removal and soft tissue and bone resection resulting from infection and scarring constitute surgery for persistent osteomyelitis. When a fracture has not yet solidified, a brief course of antibiotics combined with local fixing material—which may be removed once consolidation occurs—might enable complete mechanical healing even in cases of sepsis.(37)

Even with adequate debridement, there may still be a considerable amount of dead space that has to be controlled to avoid recurrence and severe bone loss that might lead to instability. It can be necessary to restore the soft-tissue and bone deficiencies appropriately. A thorough radiological evaluation has to be done. A team with experience in infectious diseases, radiography, orthopaedic surgery, and plastic surgery—particularly in

covering procedures such skin grafts, muscle and myocutaneous flaps, and free flaps—should perform the curative treatment.(38)

Vertebral osteomyelitis

Only the treatment of complications or the failure of therapy can warrant surgical intervention. The primary goals of the procedure are to reduce spinal cord compression, drain epidural or paravertebral abscesses, and enhance spinal stability.(39)

Diabetes foot infection

The degree of local infection, the patient's preferences, and the vascularization of the affected tissue all influence the course of treatment. Prior to considering amputation, surgery to restore vascularization may be helpful. Another alternative is to use long-term oral antibiotic suppression to postpone or prevent amputation. The effectiveness of hyperbaric oxygen in this situation is not shown beyond a reasonable doubt. A patient with good oxygen tension at the site of infection may benefit from debridement and a 4-6 week course of antibiotic treatment, especially if the pathogen's identification has been determined. When there is insufficient oxygen tension, the wound cannot heal and the diseased foot

finally has to be amputated. For this reason, resection of chronic osteomyelitic bone is commonly employed.(40)

Prosthetic-joint infections

The rule is to remove the device. A classic approach for prosthetic infection, well documented for hip infections and by extension for other prosthetic joints, is a twostage exchange arthroplasty. It entails the surgical removal of all foreign material, debridement of the bone and soft tissues, and 4–6 weeks of parenteral antimicrobial therapy before reconstruction.(41)

Immediately following a one-stage exchange arthroplasty, a fresh prosthesis is implanted. The great success rate of the one-stage approach may be attributed to the fact that practitioners always use cement that contains an antibacterial medication. However, one-stage arthroplasty is limited to hip prosthesis infections and has a greater risk of recurring infection than the two-stage operation. For additional prosthetic joints (elbow, shoulder, and knee), two-stage exchange using cement laden with antibiotics is recommended.(42)

Some circumstances involving a stable hip prosthesis infected with very sensitive microorganisms, such as streptococci, or the early diagnosis of device-related infections with staphylococci, which have been reported to be cured by debridement alone followed by several months of treatment with oral quinolones and rifampicin, are exceptions to exchange arthroplasty, which allows cure with antibiotics alone and leaves the prosthetic material locally. Owing to their increased morbidity and mortality, especially morbidity attributable to exchange arthroplasty surgery and associated extended hospital stays, this technique has proven especially beneficial for older patients.(43)

Conclusion

In patients with easy access to contemporary healthcare, the rate of treatment failure in acute osteomyelitis has decreased as a result of increased awareness, improved diagnostic techniques, and improved therapy. The frequency of sequelae has decreased. The incidence of postoperative infection has been further reduced by infection control measures and preventative medicines.

Nevertheless, as no preventative strategy is anticipated to bring the risk of infections below 0.5%, the significant rise in reconstructive orthopedic surgeries using prosthetic materials would raise the total number of infections. In order to more precisely resemble the extracellular matrix and combine superior mechanical and bacterial antiadhesive qualities, new materials for rebuilding are required. Novel surgical strategies that include vascular, plastic, and orthopaedic procedures are required for persistent osteomyelitis. Research is also needed to find novel biological elements that stimulate bone development, to speed up the healing process following surgery, to decrease the time that the body is susceptible to infection, and to enable quicker new bone creation. Osteomyelitis significantly reduces

quality of life and comes with a significant financial cost. In order to effectively treat this condition, a patient-doctor discussion is crucial, founded on a comprehensive medical knowledge of the costs, dangers, and likelihood of success of various treatment choices.

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