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Musculoskeletal Infections in the Emergency Department

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INTRODUCTION

Bone and joint infections are a relatively uncommon cause of musculoskeletal complaints among patients seeking care in the emergency department (ED). Atypical and non-specific presentations can be misleading and definitive diagnosis of infection challenging, often requiring invasive and time-consuming procedures. This review will outline the clinical signs and symptoms that should lead emergency physicians to consider a musculoskeletal infection, the diagnostic workup, and key therapeutic interventions when the clinical suspicion for infection is high. The approach to osteomyelitis, spondylodiscitis, spinal epidural abscess, antibiotic prophylaxis for an open fracture, septic arthritis, and periprosthetic joint infection in the ED will serve as the primary focus.

OSTEOMYELITIS

Osteomyelitis is an inflammatory reaction of the bone due to infection, most often bacterial in nature. Infection can involve the bone marrow, cortex, periosteum, or surrounding soft tissues, leading to destruction of any or all of these anatomic structures.[1] In a U.S.

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population-based study conducted in Olmstead County, Minnesota, the overall incidence of osteomyelitis increased from 11.4 cases per 100,000 person-years in the period from 1969 to 1979 to 24.4 per 100,000 person-years in the period from 2000 to 2009.[2] While rates remained stable among children and adults under fifty years of age, incidence nearly tripled among those aged sixty years or greater, fueled by a significant rise in diabetes-related osteomyelitis over the past four decades.

Osteomyelitis develops from one of three mechanisms of pathogenesis: bacteremia leading to hematogenous seeding of bone, contiguous spread of infection from adjacent soft tissue to bone, or direct inoculation of microorganisms into bone. Hematogenous osteomyelitis results either from the introduction of microorganisms into the bloodstream (*e.g.*, via injection drug use or an infected central venous catheter) or an infection elsewhere that has now been complicated by bloodstream involvement (*e.g.*, endocarditis, urinary tract infection). Osteomyelitis due to contiguous spread occurs most frequently in the setting of skin breakdown (*e.g.*, diabetic foot ulcer, vascular ulcer, or pressure-related decubitus ulcer) and soft tissue infection extending to underlying bone. Infected joints, both native (septic arthritis) or prosthetic, and other infected orthopedic devices can likewise involve adjacent bone. Osteomyelitis from direct inoculation classically arises in the setting of an open fracture or surgery. Patients with osteomyelitis will often have one or more pathologic risk factors associated with these mechanisms. (Table 1) *Acute* osteomyelitis progresses over days to weeks and is characterized by inflammation of viable bone. In contrast, *chronic* osteomyelitis evolves over weeks, months, or even years and is distinguished by progression to osteonecrosis with the formation of sequestrum, often in the setting of recurrent or refractory infection.

Several classification systems exist to categorize osteomyelitis. The Waldvogel classification system differentiates osteomyelitis by mechanism of pathogenesis, focusing on hematogenous seeding, contiguous spread, and vascular insufficiency.[3] The Cierney-Mader classification system organizes osteomyelitis by the extent of host anatomic involvement, physiologic status, and comorbid factors that may influence clearance of infection.[4]

The causative pathogen associated with osteomyelitis is determined by the risk profile of the patient (*e.g.*, age, comorbid diseases, immune status, exposure or travel history) and the suspected mechanism of pathogenesis.[5] *Staphylococcus aureus* remains the most common microorganism isolated across all forms of osteomyelitis.[6] Coagulase-negative staphylococci are often associated with osteomyelitis due to prosthetic joint and other orthopedic hardware infections.[5] *Streptococcus* species, Gram-negative bacteria, and anaerobes are also frequently encountered, particularly in polymicrobial infections associated with a diabetic foot ulcer, vascular ulcer, or pressure-related decubitus ulcer.[1, 6] Infections due to Gram-negative bacteria, including *Pseudomonas aeruginosa* and *Escherichia coli*, are often seen in the elderly.[7] *P. aeruginosa* is frequently associated with healthcare-associated osteomyelitis.[1] *Salmonella* is a well-recognized cause of osteomyelitis in patients with sickle cell anemia.[5] Immunocompromised patients are at risk for infection due to a wide range of unusual microorganisms including *Bartonella henselae* [8], *Mycobacterium avium* complex [9], and fungi (*Aspergillus*, *Candida*).[10]

Clinical presentation and physical examination

Osteomyelitis can present clinically in a myriad of ways. Reasons prompting ED evaluation may be as obvious as a gaping wound with exposed bone or as cryptic as a fever of unknown origin.[11] Infections can be as insidious and misleading as a solitary draining sinus tract.[1] Non-healing wounds or chronic, recurrent soft tissue infections may portend a deeper bone infection. The presenting complaint will often be related to localized pain at the site of the infection. This may be accompanied by warmth, swelling, or erythema.[12] Patients with diabetes mellitus may lack these signs or symptoms due to underlying vascular disease and/or pre-existing neuropathy.[13] Oftentimes, the patient may lack systemic symptoms such as fever, chills, fatigue, irritability, or malaise.

The most common anatomic locations of osteomyelitis correlate with their mechanism of pathogenesis. Hematogenous osteomyelitis usually occurs in the metaphyseal region of long bones (*e.g.*, femur, tibia) in children and the spine in adults.[7] Contiguous spread of an adjacent infection to bone is most often seen in the lower extremities, namely the feet where diabetic and vascular ulcers are likely to develop.[14] Osteomyelitis from direct inoculation occurs in traumatized long bones as well as adjacent to large joints (*e.g.*, hip, knee) associated with recently implanted prosthetic hardware.

The utility of the physical examination in diagnosing osteomyelitis is variable, ranging from equivocal to all but confirmatory. Acute infection classically includes any combination of the four cardinal signs of inflammation: rubor (redness), calor (heat), dolor (tenderness), or tumor (swelling). [12] In other cases, a draining sinus tract may be the only outward indication of a deeper bone infection. Physical findings associated with a chronic wound suggesting an underlying osteomyelitis can include non-purulent drainage, friable or discolored granulation tissue, undermining of the wound edges, and/or foul odor.[12] Hematogenous osteomyelitis, especially of the spine, may present solely with tenderness to palpation of the affected bone.

In diabetic and other patients with chronic wounds, certain physical examination findings specific to and highly suggestive of osteomyelitis are easily identified at the bedside. Visible bone exposed in the wound or ulcer bed has a positive likelihood ratio (LR) of 9.2 [95% confidence interval (CI), 0.6 – 146.0] for predicting osteomyelitis.[13] Exploration of the wound or ulcer bed with a sterile surgical probe contacting bone (a positive probe-to-bone test) has a positive LR of 6.4 (95% CI, 3.6 – 11.0).[13] An ulcer area larger than 2 cm² has a positive LR of 7.2 (95% CI, 1.1 – 49.0).[13]

Diagnostic evaluation

Clinical suspicion for osteomyelitis based on history and physical examination should lead to formal diagnosis with comprehensive laboratory testing, dedicated imaging, and tissue sampling. Due to the non-specific constellation of signs and symptoms often associated with osteomyelitis, emergency physicians must have a high index of suspicion for this disease.

Initial laboratory testing in the ED should include a complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) at a minimum. If the patient has systemic symptoms to suggest active bacteremia, blood cultures should be

obtained. Patients with osteomyelitis may or may not have a leukocytosis.[6] Inflammatory markers are often elevated acutely and can be helpful in trending response to treatment later on.[1] An ESR 70 mm/hour is suggestive of osteomyelitis with a positive LR of 11.0 (95% CI, 1.6 – 79.0).[13] Superficial wound cultures obtained in the ED have little diagnostic benefit as they correlate poorly with pathogens isolated from the bone.[13] In diabetic foot ulcers, wound cultures correlate with microbiologic findings in bone culture in only 19-36% of cases.[15-17]

Initial imaging should consist of plain radiography of the region of interest. Although plain films may not demonstrate bony changes of acute osteomyelitis until after 2-3 weeks of infection, they help rule out other potential pathology (e.g., fracture) [5] and provide a baseline for future comparison. One meta-analysis of diabetic foot osteomyelitis found a sensitivity of 54% and specificity of 68% when plain radiographs were used to establish the diagnosis.[18] Radiographs may show cortical bone deformity and/or destruction, at times with adjacent soft tissue swelling, joint space widening or narrowing, or periosteal reaction. [13, 19] They may also reveal indirect signs of infection such as subcutaneous gas or radio-opaque foreign bodies that could serve as a nidus of infection. Computed tomography (CT) can demonstrate periosteal reaction, cortical/medullary bone destruction, and/or adjacent soft-tissue infection in greater detail. Magnetic resonance imaging (MRI) is the most sensitive (90%) and specific (83%) imaging modality available for diagnosing osteomyelitis, often as early as 3 to 5 days after initial onset of infection.[20, 21] It can also identify early bone edema, cortical destruction, soft-tissue infection, and/or sinus tract development.

Definitive diagnosis of osteomyelitis is made by bone biopsy with histopathology and microbiologic culture. Isolation of the infecting microorganism from bone followed by antibiotic susceptibility testing are important in directing treatment.

Management

Limited well-designed prospective randomized clinical trials exist to guide the management of patients with osteomyelitis. Treatment recommendations are based on experimental animal models, retrospective studies, and expert opinion.[12] Eradicating a deep-seated infection of the bone can be difficult and successful treatment of osteomyelitis often requires a combination of medical and surgical therapies.

The first determination an emergency physician must make in caring for a patient with osteomyelitis is to decide whether antibiotic therapy needs to be initiated in the ED. If the patient is hemodynamically stable and without signs of systemic illness, consider delaying empiric antibiotic therapy until a bone biopsy can be obtained in order to optimize culture yield and better guide long-term antibiotic therapy tailored to the patient's infection. However, if the patient is unstable or has signs of sepsis, empiric antibiotic therapy in the ED is justified.[12] If hematogenous osteomyelitis or active bacteremia is suspected, blood cultures should be obtained prior to antibiotic administration to aid in the isolation of a causative microorganism.

Empiric antibiotic therapy should include broad coverage of Gram-positive (e.g., *S. aureus*, *Streptococcus* spp.) and Gram-negative bacteria, including antibiotic-resistant organisms

(*e.g.*, methicillin-resistant *S. aureus*) depending on patient risk factors and any prior history of a resistant infection. If a polymicrobial infection is suspected, usually in the setting of a chronic wound, anaerobic coverage should be added. Gram-positive coverage is most commonly achieved with vancomycin; alternatives include daptomycin or linezolid. Gram-negative coverage, including that of *P. aeruginosa*, can be achieved with cefepime or meropenem. If cefepime is used, metronidazole should be added for anaerobic coverage; meropenem has intrinsic activity against anaerobes. Use of vancomycin in combination with piperacillin-tazobactam for empiric therapy should be avoided whenever possible to limit the risk of nephrotoxicity.[22]

The indications for surgical treatment of osteomyelitis will depend on many factors including the anatomic location, duration of symptoms, mechanism of pathogenesis, and patient comorbidities. The main objectives of surgical intervention include source control (*e.g.*, debridement of infected tissues, removal of infected orthopedic prostheses or other hardware), management of wounds and dead space (*e.g.*, flap coverage), and stabilization of any infected and unstable bone that cannot be resected.[7] Chronic osteomyelitis requires surgical debridement as antibiotics have limited ability to penetrate devascularized or necrotic bone (*e.g.*, sequestrum). In patients with osteomyelitis and vascular insufficiency, revascularization procedures may be necessary to ensure adequate delivery of systemic antibiotic therapy to the site of infection.

With regards to disposition, patients with acute osteomyelitis should generally be admitted to facilitate bone biopsy, determine any need for surgical intervention, and initiate long-term intravenous antibiotic therapy (usually lasting six weeks) in consultation with an infectious disease specialist. Patients with chronic osteomyelitis that have already been cultured, are on appropriate antibiotics, and are without signs of systemic infection or instability may be considered for outpatient treatment provided adequate follow-up is available.

SPONDYLODISCITIS (VERTEBRAL OSTEOMYELITIS)

Spondylodiscitis is an infection of the intervertebral disc (discitis) and adjacent vertebrae (osteomyelitis). Commonly referred to as vertebral osteomyelitis, spondylodiscitis has an annual incidence of 2.4 to 4.8 cases per 100,000 persons.[23, 24]

Infection primarily arises as a sequela of bacteremia (*e.g.*, urinary tract infection, endocarditis, infected central venous catheter, injection drug use) with subsequent hematogenous seeding of the spine. Spondylodiscitis typically affects two contiguous vertebrae and the intervertebral disc space between them.[7] The most common site of infection is the lumbar spine (58%), followed by the thoracic (30%) and cervical spine (11%).[25] Diabetes mellitus, immunocompromised state, systemic steroid use, chronic kidney disease requiring hemodialysis, liver disease, malignancy, bacteremia/endocarditis, previous back surgery, and injection drug use are established risk factors for this infection. [26, 27] Spondylodiscitis can also result from contiguous spread of an infection involving adjacent structures (*e.g.*, aorta, esophagus, colon).[28-30] Direct inoculation of the spine with microorganisms leading to spondylodiscitis can occur in the setting of spine surgery

and has been reported rarely after epidural corticosteroid injections as well as penetrating trauma.[31-33]

S. aureus remains the predominant causative microorganism associated with spondylodiscitis, although *Streptococcus* species are also common, particularly among the elderly and those with diabetes mellitus.[25, 34] Infection with *P. aeruginosa* is frequently associated with injection drug use. Other bacteria including *Mycobacterium tuberculosis* and *Brucella* can also cause spondylodiscitis and should be considered based on exposure and travel history.[35, 36]

Clinical presentation and physical examination

Diagnosing spondylodiscitis can be especially challenging as the disease is rare and clinical presentations are non-specific and highly variable. More than 90% of patients will complain of localized back or neck pain that is often worse at night and not alleviated by rest or analgesics.[37-39] Anywhere from 35% to 60% of patients will present with fever.[25, 40] In one study, only a quarter of patients had neurologic symptoms such as a sensory deficit, muscle weakness/paralysis, radiculopathy, or sphincter dysfunction due to spinal cord compression.[41]

When infection due to spondylodiscitis spreads to adjacent structures, more salient findings are also possible. (**Table 2**) Infection of the cervical spine can spread to the retropharyngeal space leading to abscess formation.[42] In addition to paraspinal abscesses, thoracic spine infections can be accompanied by a reactive pleural effusion or empyema.[43] Lumbar spine infections can precipitate abscesses of the psoas muscle.[44]

Diagnosis is frequently delayed by up to two to four months after symptom onset.[45] In one study, more than a third of patients with vertebral osteomyelitis were initially misdiagnosed [46], highlighting the under-recognition of this infection in clinical practice. Atypical presentations and late diagnoses are particularly common among patients over the age of 65 years and a low threshold should exist to further investigate new or worsening back pain in this high-risk population.[47]

Diagnostic evaluation

The diagnostic workup for spondylodiscitis is similar to that of non-axial osteomyelitis. Typical laboratory tests should include a CBC, ESR, and CRP. Up to 40% of patients with vertebral osteomyelitis may have a normal WBC count.[48] A high ESR or CRP in a patient with back pain has a sensitivity of 94% to 100% for the disease.[48, 49] As vertebral osteomyelitis is often hematogenous in origin, multiple sets of blood cultures should be obtained. Blood cultures are positive in 58% of cases and approximately 25% to 59% of positive blood cultures accurately identify the causative microorganism responsible for the spine infection.[25, 50, 51]

As part of an initial evaluation, plain radiographs may demonstrate narrowing of the disk space and destruction of the vertebral endplates or vertebral body as well as alternative etiologies for back pain (e.g., compression fracture, metastatic disease).[52] However, their sensitivity for detecting osteomyelitis or an isolated discitis is poor.[37, 52] MRI remains the

preferred imaging modality for early diagnosis of spondylodiscitis as it is both highly sensitive (93-96%) and specific (92.5-97%).[53, 54] More than half of patients with spondylodiscitis will have characteristic radiographic findings of the disease on MRI within two weeks after onset of infection, followed by another 20% over the following two weeks. [55] CT is helpful in identifying cortical destruction and adjacent soft-tissue infection when MRI is contraindicated.

Definitive diagnosis of spondylodiscitis is made by bone biopsy with multiple specimens submitted for microbiologic culture and pathological review.[7] CT-guided bone biopsy has a sensitivity of 50%.[56]

Management

The mainstay of treatment for spondylodiscitis is medical management with long-term antibiotic therapy for at least six weeks.[57] If the patient is hemodynamically stable and has a normal neurologic examination, consider withholding antibiotic therapy until blood cultures and a bone biopsy can be obtained to guide antibiotic selection. If the patient is clinically unstable or has signs of sepsis, empiric antibiotic coverage for *S. aureus* (including MRSA), streptococci, and Gram-negative bacilli can be achieved with a combination of vancomycin and a third or fourth-generation cephalosporin, ideally after blood cultures have been obtained.

Consultation with a spine surgeon and infectious disease specialist is advised along with hospital admission to facilitate further evaluation and treatment. Surgical debridement may be necessary in the setting of epidural or paravertebral abscess formation, evolving neurologic deficits, or spinal instability.[7, 57]

SPINAL EPIDURAL ABSCESS

Spinal epidural abscess (SEA) is a collection of pus located in the spinal canal between the dura mater and overlying vertebral column. While SEA can develop independently of spondylodiscitis, it often occurs as direct complication. In one study, a third of spondylodiscitis cases were accompanied by a secondary SEA.[58] Overall, SEA remains uncommon with an estimated incidence of 1.8 per 100,000 persons per year.[59] As with spondylodiscitis, *S. aureus* is the predominant causative microorganism, although infections involving other Gram-positive and Gram-negative bacteria are also possible.[60]

Clinical presentation and physical examination

Patients with SEA can present to the ED anywhere along a continuum of disease with a wide range of clinical signs and symptoms. (Table 3) When each symptom is considered in isolation, back pain is present in three-quarters of patients, fever in nearly half of patients, neurologic abnormalities in a third of patients, and paralysis in a fifth of patients.[61-64] The classic triad of fever, back pain, and neurologic deficits is exhibited by only a minority of patients with SEA.[60] A high index of clinical suspicion followed by timely diagnosis of SEA is essential to prevent complications of cord compression and irreversible sensory loss or paralysis.

Diagnostic evaluation

The initial evaluation of SEA in the ED should mirror that of spondylodiscitis. The presence of leukocytosis is neither sensitive nor specific for the diagnosis.[60] While an elevated ESR and CRP is sensitive, neither is specific to the disease. Blood cultures are positive in about 60% of patients with SEA.[65, 66]

While plain radiography and non-contrast CT cannot directly visualize SEA, both may provide indirect evidence of its presence, usually in the form of a concomitant spondylodiscitis.[67, 68] MRI and CT myelography are highly sensitive (>90%) in diagnosing SEA.[61, 69] MRI is the imaging modality of choice as it is non-invasive and facilitates assessment of surrounding structures for spread of infection (*e.g.*, paraspinal abscess) and evaluation for spinal cord compression.[60] Lumbar puncture is not recommended as Gram stain and cerebrospinal fluid culture are not sensitive enough to detect the presence of SEA and may inadvertently introduce infection into the central nervous system.[60]

Management

Whereas spondylodiscitis is often managed medically with long-term antibiotic therapy alone, SEA frequently requires a combined medical and surgical approach, particularly when the cervical or thoracic spine is involved and spinal canal narrowing with cord compression can progress rapidly to neurological dysfunction.[38] Consultation with a spine surgeon is paramount to consider surgical drainage of the abscess, debridement of adjacent infected tissue, preservation of the local blood supply to promote tissue healing, and maintenance of spinal stability.[70] Empiric antibiotic therapy with a combination of vancomycin and a third or fourth-generation cephalosporin is reasonable until cultures of the abscess or blood permit tailored coverage for a prolonged course, best determined in concert with an infectious disease specialist.

ANTIBIOTIC PROPHYLAXIS FOR OPEN FRACTURE

Osteomyelitis can complicate up to a quarter of open fractures due to direct inoculation of bacteria into bone, either from the patient's skin flora or the external environment.[6, 71] The risk of post-traumatic osteomyelitis hinges on the severity of the fracture and any associated soft tissue defect, vascular compromise, or gross wound contamination, as well as surgical debridement and post-injury antibiotic prophylaxis.[71-76]

In the ED, any gross contaminant should be removed from the wound followed by copious irrigation with normal saline.[77] The wound should be covered with a sterile dressing pending evaluation by an orthopedic surgeon and formal irrigation and surgical debridement in the operating room. Antibiotic prophylaxis should be administered systemically, preferably within six hours of the injury. Current guidelines from the Eastern Association for the Surgery of Trauma (EAST), the Surgical Infection Society (SIS), and U.S. Department of Defense recommend specific antibiotic regimens based on fracture severity as defined by Gustilo-Anderson classification.[78-80] (**Table 4**) While Gram-positive coverage is universally endorsed, EAST guidelines also recommend Gram-negative coverage

(historically with an aminoglycoside) for type III open fractures.[79] In situations where fecal or clostridial contamination (*e.g.*, farmyard injury) is suspected, the same guidelines also suggest the addition of high-dose penicillin.

SEPTIC ARTHRITIS

Septic arthritis is an infection of the joint. Worldwide, the annual incidence of septic arthritis ranges from 5 to 12 per 100,000 persons.[81-83] In the U.S., more than 16,000 ED visits in 2012 involved a primary diagnosis of septic arthritis, comprising 0.01% of all ED visits during that year.[84]

Most cases of septic arthritis stem from hematogenous seeding of a joint in the setting of bacteremia. Less frequently, overlying soft tissue infections can spread contiguously to the joint. Direct inoculation of bacteria into a joint can occur through a traumatic arthrotomy, open fracture or dislocation, arthrocentesis, or intra-articular injection resulting in infection. Common risk factors for developing septic arthritis include older age, diabetes mellitus, rheumatoid arthritis, joint surgery, prosthetic joint, skin infection, and injection drug use. [81-83, 85]

As with other musculoskeletal infections, *S. aureus* and *Streptococcus* species are the most frequent causative microorganisms associated with septic arthritis.[83, 86] Infections due to Gram-negative bacilli are more common among the elderly (*i.e.*, in the setting of an underlying urinary tract infection) as well as those who inject drugs.[86, 87] Disseminated *Neisseria gonorrhoeae* infection (DGI) can present as septic arthritis, usually in young sexually active adults. *Candida* septic arthritis may be encountered in patients with impaired or normal immune systems.[88]

Clinical presentation and physical examination

Joint pain with associated warmth, swelling, and restricted movement is the primary presenting feature of septic arthritis.[89, 90] Large joints such as the knee and hip are most commonly involved, although smaller joints can also become infected. Fever may be present but is not diagnostic.[89, 91] Examination of the affected joint is often notable for an effusion with associated erythema, warmth, and tenderness. Range of motion of the joint may be limited and painful. Many of these signs and symptoms can be muted in the setting of immunosuppression and fail to distinguish septic arthritis from other non-infectious forms of arthritis. Unrecognized, septic arthritis can progress to joint destruction, osteomyelitis, and sepsis with an overall mortality approaching 10%.[91] Timely recognition of septic arthritis by the emergency physician is necessary to improve outcomes.

Diagnostic evaluation

While CBC, ESR, and CRP should be obtained, definitive diagnosis rests with performing an arthrocentesis of the affected joint. Fluoroscopic or ultrasound guidance by interventional radiology may be necessary to sample certain joints (*e.g.*, hip, sacroiliac joint). Synovial fluid should be submitted for cell count with differential, Gram stain, and microbiologic (aerobic and anaerobic) culture. The higher the synovial white blood cell (WBC) count the greater the likelihood of septic arthritis.[89, 90] A synovial WBC count $>50,000$ cells/mm³

has a positive LR of 7.7 (95% CI, 5.7 – 11.0); a synovial WBC count $>100,000$ cells/mm³ has a positive LR of 28.0 (95% CI 12.0 – 66.0).[89] A synovial WBC count with a differential of $>90\%$ polymorphonuclear cells has a LR of 3.4 (95% CI, 2.8 – 4.2) for septic arthritis.[89] At best, Gram stain has a sensitivity of 50% for detecting bacteria in synovial fluid, while that of synovial fluid culture approaches 80%.[89] If only one test can be sent due to a limited volume of synovial fluid, the culture should receive priority. Multiple sets of blood cultures should also be obtained when hematogenous septic arthritis is suspected to improve the chances of identifying a causative microorganism.

As far as imaging, plain radiography of the infected joint may reveal joint destruction or an associated osteomyelitis. CT and MRI can be helpful in evaluating for septic arthritis involving difficult-to-assess sites such as the hip or sacroiliac joint.

Management

Empiric antibiotic therapy for septic arthritis in the ED should cover Gram-positive bacteria, including MRSA, with vancomycin being a reasonable initial choice. If the patient has risk factors for a Gram-negative infection (*e.g.*, injection drug use, immunocompromised state, elderly), a third or fourth generation cephalosporin should be added. Ideally, antibiotic therapy should be started after synovial fluid sampling and blood culture collection. An orthopedic surgeon should be consulted to pursue irrigation and surgical drainage in the operating room or serial arthrocentesis. Patients with septic arthritis will generally require hospital admission and several weeks of antibiotic therapy.

PERIPROSTHETIC JOINT INFECTION

Anywhere from 0.5% to 2% of primary total hip or knee arthroplasty procedures are complicated by periprosthetic joint infection (PJI).[92-94] Infections arising within the first few months of joint implantation are more likely to be related to microorganisms acquired at the time of surgery (*e.g.*, *S. aureus*) while late infections may be attributable to chronic infection with indolent microorganisms (*e.g.*, coagulase-negative staphylococci) or hematogenous seeding of bacteria from a distant site (*e.g.*, urinary tract, infected central venous catheter).[95, 96] Risk factors for PJI include male gender, tobacco use, elevated body mass index (> 30 kg/m²), diabetes mellitus, rheumatoid arthritis, corticosteroid use, previous joint surgery (including revision arthroplasty), and depression.[97]

Clinical presentation and physical examination

Similar to patients with septic arthritis, those with PJI may present with warmth, swelling, and tenderness over the affected joint, with or without signs of surrounding soft tissue infection or systemic illness.[95, 96] In late or chronic infections, pain at the site of the prosthetic may be the only complaint endorsed. A non-healing or draining wound overlying or a draining sinus tract communicating deep to the prosthesis is concerning for infection, with the latter considered definitive evidence for PJI.[96]

Diagnostic evaluation

Standard laboratory testing should include CBC, ESR, and CRP, although these tests are nonspecific for PJI.[96] If there is a strong clinical suspicion for PJI, diagnostic arthrocentesis of the affected joint should be pursued in consultation with an orthopedic surgeon and interventional radiologist (particularly in the case of a prosthetic hip). [96, 98, 99] Emergency physicians should recognize that parameters for diagnosing PJI on synovial fluid analysis differ significantly from that of septic arthritis involving a native joint.[95, 96] As part of its definition for PJI, the International Consensus Group on Periprosthetic Joint Infection recommends a minimum synovial WBC count threshold of 10,000 cells/mm³ as one of several criteria for acute PJI (<90 days since surgery) and 3,000 cells/mm³ for chronic PJI (>90 days).[100] Likewise, this definition suggests a minimum synovial fluid WBC differential of 90% polymorphonuclear cells as another criterion for acute PJI and 80% for chronic PJI.[100] Synovial fluid culture is moderately sensitive and highly specific for predicting PJI, with a positive LR of 15.3 (95% CI, 10.6 – 22.1).[101] Superficial cultures should not be obtained from draining wounds or sinus tracts as they correlate poorly with deep cultures obtained from the infected joint.[102]

Imaging as part of the ED evaluation for PJI should include plain radiographs of the affected joint to assess for osteolysis or hardware loosening that might suggest infection; there is no clearly defined role for CT or MRI in making the diagnosis. [95, 103]

While diagnostic work-up in the ED may yield findings supportive of PJI, diagnosis remains clinical in many instances and may ultimately need intraoperative evaluation and tissue sampling by an orthopedic surgeon for confirmation.[96, 98, 103]

Management

In the absence of hemodynamic instability, sepsis, or a severe soft tissue infection, empiric antibiotic therapy in the ED can be withheld until synovial fluid culture is obtained in order to maximize their yield. Patients with PJI will require multidisciplinary care combining surgical debridement with or without removal of the infected prosthesis and appropriate long-term antibiotics.

CONCLUSION

Clinical presentations of musculoskeletal infection in patients presenting to emergency care can range from chronic wounds and non-specific bone or joint pain to functional debilitation and even neurologic compromise when the spine is involved. Emergency physicians play a critical role in the early recognition, evaluation, and management of these infections. Appropriate collection of microbiologic cultures (*e.g.*, blood, synovial fluid) prior to empiric antibiotic therapy in the ED has significant implications for isolating causative microorganisms and guiding downstream treatment. Antibiotic therapy may even be safely withheld to pursue bone biopsy and culture if the patient is clinically stable. A low threshold should exist to seek orthopedic surgery and infectious disease consultation in patients presenting with bone and joint infections to the ED and many will require hospitalization for definitive care, combining medical and surgical approaches.

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Key points:

- Patients with musculoskeletal infections can have heterogeneous presentations as the signs and symptoms are often occult and non-specific.
- *Staphylococcus aureus* is the most common microorganism associated with bone and joint infections.
- Definitive diagnosis requires sampling of affected tissue for Gram stain and microbiologic culture.
- The mainstay of treatment for bone and joint infections is antibiotic therapy, but surgical consultation for irrigation and/or debridement may be necessary in certain clinical situations.

Synopsis:

Bone and joint infections, including osteomyelitis, spondylodiscitis, spinal epidural abscess, septic arthritis, and periprosthetic joint infection, have the potential to be limb- or even life- threatening diseases. Emergency physicians must consider infection when evaluating musculoskeletal complaints, as misdiagnosis can have significant consequences. Patients with bone and joint infections can have heterogeneous presentations with non-specific signs and symptoms. *Staphylococcus aureus* is the most commonly implicated microorganism. Although diagnosis may be suggested by physical examination, laboratory testing, and imaging, tissue sampling for Gram stain and microbiologic culture is preferable as pathogen identification and susceptibility testing help optimize long-term antibiotic therapy. A combination of medical and surgical interventions is often necessary to effectively manage these challenging infections.

TABLE 1.

Risk Factors for Development of Osteomyelitis

Mechanism of pathogenesis	Risk factor
Hematogenous seeding	Injection drug use Central venous catheter or other long-term vascular device Urinary tract infection Immunosuppression (including chronic corticosteroid use)
Contiguous spread from adjacent tissues	Extremes of age Diabetes mellitus Vascular insufficiency Abscess/cellulitis/infected ulcer
Direct inoculation	Prior orthopedic surgery or indwelling orthopedic hardware Trauma (open fracture) Human/animal bite

TABLE 2.

Sequelae of Vertebral Osteomyelitis

Region	Spread of infection	Symptoms
Cervical	Retropharyngeal abscess	Sore throat, painful swallowing, change in voice
Thoracic	Reactive pleural effusion, empyema, paraspinal abscess	Cough, shortness of breath, chest pain, flank pain
Lumbar	Psoas muscle abscess	Abdominal or flank pain, pain on extension or internal rotation of hip (positive psoas sign)

TABLE 3.

Stages of clinical progression in spinal epidural abscess [104, 105]

Stage	Clinical Findings
1	Back pain at the level of the affected spine, fever, tenderness
2	Radicular pain, nuchal rigidity/neck stiffness, reflex changes
3	Motor weakness, sensory deficit, and bladder and bowel dysfunction
4	Paralysis

TABLE 4.

Antibiotic prophylaxis for open fracture by Gustilo-Anderson classification [79]

Type	Description	Infection rate [71-73]	Antibiotic
I	Open fracture with wound $\leq$ 1 cm and clean	0-2%	Cefazolin 2 g IV ^{a,b}
II	Open fracture with wound $>$ 1 cm without extensive soft tissue injury, flap, or avulsion	2-10%	
III	Open segmental fracture with wound $>$ 10 cm with extensive soft tissue injury or traumatic amputation	10-50%	Cefazolin 2 g IV ^a + gentamicin 5 mg/kg IV (adjusted body weight) ^c

^aIf actual body weight $\leq$ 120 kg, consider 3 g.^bIf penicillin allergy, may substitute with vancomycin 15 mg/kg IV (actual body weight).^cAntibiotic prophylaxis directed against Gram-negative bacteria (including *Pseudomonas aeruginosa*) remains controversial.[78-80] Institution-specific protocols and/or infectious disease consultation can help guide safe and appropriate Gram-negative coverage, particularly in the setting of impaired renal function.