Spinal Infections

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Abstract

Spinal infections can occur in a variety of clinical situations. Their presentation ranges from the infant with diskitis who is unwilling to crawl or walk to the adult who develops an infection after a spinal procedure. The most common types of spinal infections are hematogenous bacterial or fungal infections, pediatric diskitis, epidural abscess, and postoperative infections. Prompt and accurate diagnosis of spinal infections, the cornerstone of treatment, requires a high index of suspicion in at-risk patients and the appropriate evaluation to identify the organism and determine the extent of infection. Neurologic function and spinal stability also should be carefully evaluated. The goals of therapy should include eradicating the infection, relieving pain, preserving or restoring neurologic function, improving nutrition, and maintaining spinal stability.

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Before the introduction of modern antibiotic therapy, mortality in patients with vertebral osteomyelitis was as high as 25%.[1] Antibiotic therapy combined with surgical débridement and stabilization has decreased mortality to less than 5% to 15%.[2-4] Early diagnosis also has improved outcomes by facilitating rapid initiation of antibiotic treatment and preventing abscess formation, structural instability, and neurologic deterioration.

Spinal infections are evaluated according to their location, the pathogen or pathogens involved, route of the infection, age of the patient, and immune status of the host. The location of the infection may involve the osseous vertebra, the intervertebral disk, the epidural space, or the surrounding soft tissues. The pathogens are usually either bacterial or fungal; however, the widespread use of broad-spectrum antibiotics and the increasing number of immunocompromised patients have led to infections with unusual organisms.

A systematic approach must be taken in the diagnosis and treatment of each type of spinal infection. The presentation and efficacy of the various elements of the initial evaluation differ markedly for acute hematogenous infection, granulomatous spinal infection, pediatric hematogenous diskitis, epidural abscess, and postoperative spinal infection.

Pathophysiology of Spinal Infection

Pyogenic vertebral osteomyelitis is a bacterial infection that can arise from a number of sources—direct inoculation, contiguous spread from an adjacent infection, or hematogenous seeding. Direct inoculation can result from penetrating injuries or from percutaneous or open spinal procedures (eg, chemonucleolysis, diskography, diskectomy) done on the intervertebral disk. Local spread of bacteria or fungi can occur following intra-abdominal and retroperitoneal abscesses. Although local spread from direct inoculation of bacteria into the spinal canal is likely to become more prevalent as the number of spinal procedures increases, hematogenous seeding of infection is still by far the most common mechanism of spinal infection. Potential sources of pathogenic organisms include skin and soft-tissue infections, infected vascular access sites, and the urinary tract.

The two major theories for hematogenous dissemination are the venous theory and the arteriolar theory. Batson[5] developed the venous theory using both live animal and human cadaveric models. He demonstrated retrograde flow from the pelvic venous plexus to the perivertebral venous plexus via valveless meningorrhachidian veins. In the arteriolar theory, Wiley and Trueta[6] proposed that bacteria can become lodged in the end-arteriolar

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network near the vertebral end plate. Both mechanisms are likely significant in the establishment of an infectious focus in the spinal column. In the cervical spine, an extensive prevertebral pharyngeal venous plexus also may act as a conduit for the spread of bacteria.[7]

Local spread of infection can occur in a number of ways. Once the infection is established adjacent to the end plate of one vertebral body, it can rupture through that structure into the adjoining disk and infect the next vertebral body. The disk material is relatively avascular and is rapidly destroyed by the bacterial enzymes (Fig. 1). In the cervical spine, if the infection penetrates the prevertebral fascia, it can extend into the mediastinum or into the supraclavicular fossa, markedly increasing the extent and severity of the process. From the lumbar spine, abscess formation may track along the psoas muscle and into the buttock (piriformis fossa), the perianal region, the groin, or even the popliteal fossa. The extension of infection from the vertebral body or disk into the spinal canal may result in an epidural abscess or even bacterial meningitis. Destruction of the vertebral body and intervertebral disk can potentially lead to instability and collapse. In addition, with collapse of the vertebral body, infected bone or granulation tissue may be retropulsed into the spinal canal, causing neural compression or vascular occlusion. With pyogenic osteomyelitis, the lumbar spine is more commonly affected than the thoracic or cervical spine.[8]

The pathogenesis of spinal infection differs markedly between children and adults because of anatomic differences in the vascular anatomy of the vertebrae. In children, vascular channels cross the cartilaginous growth plate and end within the nucleus pulposus. These channels provide pathways for direct inoculation of organisms into the avascular nucleus pulposus. Since these vascular channels are not present in adults, the direct seeding of the disk does not occur, but rather spreading occurs by direct extension with rupture of the infective focus through the end plate into the disk.

Neurologic deterioration can be a devastating consequence of spinal infection. A number of different factors can cause neural deficit. Direct spread of infected material into the spinal canal can produce an epidural abscess that may compress the neural elements or cause thrombosis or infarction of the regional vascular supply to the spinal cord. Direct hematogenous spread rarely results in epidural abscess without the presence of associated diskitis or osteomyelitis. Pathologic fracture can occur, with associated extrusion of either infected material or bony elements into the spinal canal. Kyphosis and/or spinal instability resulting from destruction of the disk, vertebral bone, and posterior stabilizing structures can cause neural impingement. Eismont et al[4] reported several additional risk factors that predispose to neurologic deterioration: diabetes, rheumatoid arthritis, steroid use, advanced age, a more cephalad level of infection (ie, high thoracic or cervical), and infection with *Staphylococcus* species.

The pathophysiology of granulomatous spinal infection differs from that of pyogenic infections. The most common form of granulomatous disease of the spine is caused by *Mycobacterium tuberculosis* (Pott's disease). Although endemic in many developing countries, tuberculosis (TB) was nearly eradicated in

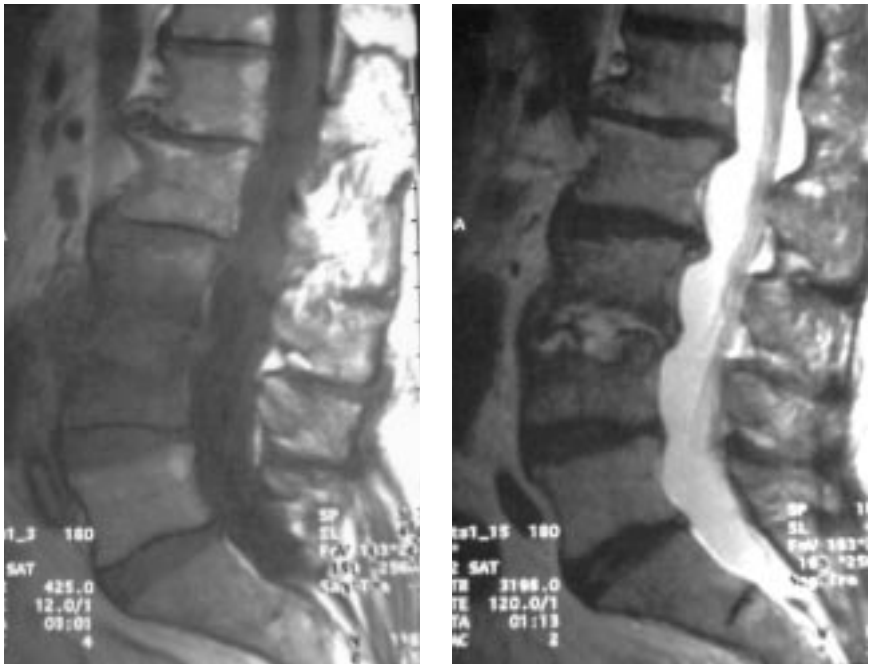

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Figure 1 A 56-year-old man presented with severe back pain following a urologic procedure. He had an elevated ESR but no leukocytosis. **A,** T1-weighted sagittal MR image of the lumbar spine shows severe edema of the L3-4 disk and adjacent soft tissues. **B,** T2-weighted sagittal MR image shows high signal intensity in the L3-4 disk and adjacent vertebral bodies, consistent with pyogenic diskitis and osteomyelitis. Cultures obtained from a CT-guided biopsy of the disk space grew *Staphylococcus aureus*.

the United States; however, there has been a recent resurgence of TB with resistant strains and in patients with human immunodeficiency virus (HIV). Although less than 10% of patients with TB have skeletal involvement, 50% of the skeletal involvement occurs in the spine. Depending on the series, between 10% and 61% of patients present with or develop a neurologic deficit.[9]

With TB, the primary route of infection to the spine is hematogenous from a pulmonary or genitourinary source, although direct spread from adjacent structures can occur. Three major patterns of spinal vertebral body involvement have been documented: peridiscal, central, and anterior.[10] The most common form, peridiscal, occurs adjacent to the vertebral end plate and spreads around a single intervertebral disk. Extension to the adjacent vertebra occurs as the granulomatous abscess material tracks beneath the anterior longitudinal ligament. Unlike the situation in pyogenic infections, the intervertebral disk is usually spared. Central involvement occurs in the middle of the vertebral body and can be mistaken for a tumor. Destruction of the vertebral body will then lead to spinal deformity. Anterior involvement begins beneath the anterior longitudinal ligament, causing scalloping of the vertebral body (Fig. 2). In contrast with peridiscal involvement, which affects a single motion segment, anterior involvement can produce a spinal abscess that extends over multiple levels. Primary involvement of the posterior structures is uncommon. Regionally, the thoracic spine is most often involved, followed by the lumbar spine and cervical spine. Paraspinal extension with abscess formation is common and can occur at any level.

Spinal infections can be classified as acute, subacute, or chronic depending on the duration of symptoms. Symptoms that have persisted for <3 weeks are acute; those lasting from 3 weeks to 3 months are subacute. Chronic infections last >3 months and either are caused by indolent organisms, are granulomatous in nature, or are incompletely treated (eg, infections with resistant organisms, or the presence of foreign material in the area of infection).

Clinical Evaluation

Pyogenic Vertebral Osteomyelitis

Pyogenic vertebral osteomyelitis is more common in males than in females and also more common in elderly populations.[2,11] However, the incidence of infection is increasing in younger age groups in populations with intravenous drug abuse or immunocompromise after organ transplantation or chemotherapy. Accordingly, spinal infection should be considered in the differential diagnosis of acute-onset spinal pain in patients older than 50 years or with diabetes, rheumatoid arthritis, immunocompromise (from medical illness or pharmacologic immunosuppression), or a history of intravenous drug abuse.

The clinical presentation of vertebral osteomyelitis depends on the location of the infection, the virulence of the organism, and the immune status of the host. Back or neck pain is the most consistent symptom of pyogenic infection. Observed in >90% of patients, the pain is often quite severe and is associated with notable paraspinal muscle spasm. The pain may occur

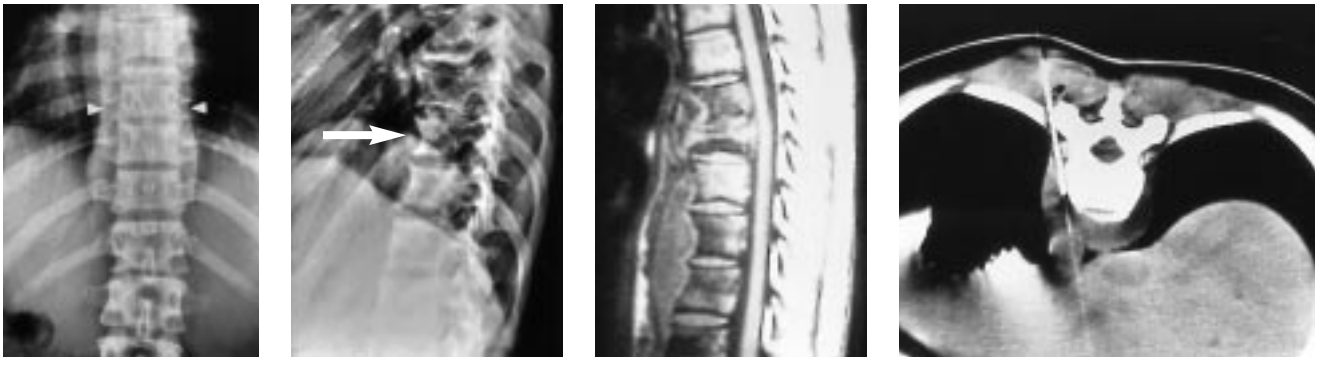

Figure 2 A 33-year-old woman presented with back pain of several months' duration. **A,** Anteroposterior radiograph shows collapse of the vertebral body and paraspinal soft-tissue shadow (arrowheads). **B,** Lateral radiograph also shows collapse and interior scalloping (arrow). **C,** Sagittal T1-weighted MR image shows a large anterior abscess, extensive vertebral body involvement, and relative sparing of disk spaces. **D,** The patient underwent CT-guided biopsy and aspiration with placement of a pigtail catheter for 1 week to drain this abscess. She underwent anti-TB treatment for 1 year, with resolution of pain and no development of deformity.

at night and is usually present regardless of activity level. Radicular leg or arm pain is less common but may be present with neurologic involvement, which occurs in less then 10% of patients. Fevers are documented in approximately 50% of the affected population.[12] Weight loss is common but may not be easily recognized by patients because it may occur slowly over a period of weeks to months before the infection is diagnosed and treated.

The presence of other signs or symptoms depends on the extent of the infectious process. A patient with a psoas abscess may have pain with hip extension. Cervical abscess formation may lead to torticollis or dysphagia. Radiculopathy, myelopathy, or even complete paralysis can occur with neural compression as a result of abscess, instability, or spinal deformity. Direct spread of the infection into the epidural space can cause meningitis.

Gram-positive organisms are responsible for the majority of vertebral column infections in both adults and children, with *Staphylococcus aureus* accounting for >50%. Infection with gram-negative organisms such as *Escherichia coli*, *Pseudomonas*, and *Proteus* may occur following genitourinary infections or procedures. Intravenous drug abusers are also prone to *Pseudomonas* infections. Anaerobic infections may be encountered in patients with diabetes or following penetrating trauma. Low-virulence organisms such as coagulase-negative staphylococci and *Streptococcus viridans* may cause indolent infections. These organisms may not be detected unless blood cultures are held for more than 10 days and should not be disregarded as contaminants in the presence of clinical infection. *Salmonella*, presumably from an intestinal source, can cause vertebral osteomyelitis in children with sickle cell anemia.

Laboratory Studies

Laboratory studies may be useful but are usually nonspecific. The white blood cell count will be elevated in approximately half the cases of acute pyogenic osteomyelitis but typically is normal in the presence of subacute or chronic infection. The erythrocyte sedimentation rate (ESR) is a more sensitive test and is elevated in >90% of patients. The C-reactive protein (CRP) level, an acute-phase reactant with a much quicker normalization time, may be more helpful in following the course of treatment than the ESR. A rapid decrease in the CRP level indicates an adequate response to treatment and can help determine when to switch from intravenous to oral antibiotics. Blood cultures may be negative in up to 75% of patients, particularly if the infection involves a low-virulence organism. It is extremely important to delay antibiotic therapy until appropriate cultures have been obtained unless the patient is septic and critically ill. Even then, blood and urine cultures should be obtained before the administration of antibiotics.

Evaluation of laboratory measurements for malnutrition is as important as the diagnostic tests that detect the presence of infection. Weight loss >30% of ideal body weight during the course of the infection indicates severe malnutrition. Other laboratory measurements that are associated with severe malnutrition include a serum albumin level of <3 g/dL, serum transferrin measurement of <150 µg/dL, and an absolute lymphocyte count of <800/mL. Although it is a measurement less commonly used in orthopaedics, a 24-hour urinary creatinine excretion of <10.5 mg in men or <5.8 mg in women indicates a negative nitrogen balance associated with malnutrition.

Biopsy

The definitive diagnosis of spinal pyogenic osteomyelitis requires identification of the organism through a positive blood culture or from a biopsy and culture of the infected site. Blood cultures may be diagnostic in as few as 25% to 33% of cases.[2] Cultures taken during fever spikes may provide better diagnostic results. Biopsy of the infected area is often necessary to initiate the appropriate antibiotic regimen. Other sources of obvious infection, such as the urine, must also be cultured. Spinal biopsies may be done percutaneously, using computed tomography (CT) or fluoroscopy to localize the focus of infection. The accuracy of closed biopsy techniques varies and has been reported to be about 70%.[13] Key factors may be insufficient tissue retrieval or administration of antibiotics prior to biopsy. A core sample obtained from a Craig biopsy needle for bone or a TruCut (Baxter Travenol, Deerfield, IL) or similar needle for soft tissue is preferable to fine-needle aspiration except when an abscess cavity is present. Antibiotics must not be started until the biopsy is done and sufficient tissue is obtained for culture, gram stain, and histology. If a diagnosis is not confirmed on the first attempt, a second closed biopsy should be considered before open biopsy is done.

An open biopsy is indicated when needle biopsy fails to identify an organism, when the infection is inaccessible by standard closed techniques, or when there is marked structural damage with neurologic compromise. Open biopsies are diagnostic in >80% of cases.[14] Minimally invasive techniques, such as a laparoscopic or thoracoscopic approach, may be considered when that approach is appropriate to decrease the morbidity of the procedure.

Biopsies should be sent for gram stain, acid-fast stain, and aerobic, anaerobic, fungal, and TB cultures. Bacterial cultures should be maintained for 10 days to detect low-virulence organisms. Histologic stud-

ies also should be done, if possible, to detect metabolic or neoplastic processes. If tissue is available, pathologic examination should be conducted to differentiate between acute and chronic infection and to help detect the presence of acid-fast bacilli and fungal elements. The development of polymerase chain reaction as a diagnostic tool has facilitated rapid detection of the infecting agent, especially when indolent and low-virulence organisms are involved.[15,16] However, technical problems with cross-contamination can lead to false-positive results.

Tuberculosis

The clinical presentation of a patient with a tuberculous spinal infection is highly variable. As with pyogenic infections, back pain is the most common symptom; however, it is usually less severe than in a pyogenic infection. Patients with chronic infection also may experience weight loss, malaise, fevers, and night sweats. Kyphotic deformities, neurologic deficits, or cutaneous sinuses may occur after prolonged or very severe infections. Neurologic deficit can occur from epidural extension of the tuberculous infection, from destruction of bone with retropulsion of infected material into the spinal canal, or from progressive kyphotic deformity. Elderly patients appear to be at higher risk for developing a neurologic deficit. The differential diagnosis of spinal infection includes primary and metastatic tumors; infections with atypical bacteria such as *Actinomyces*, *Nocardia*, and *Brucella*; infections with atypical mycobacteria; and fungal infections such as coccidioidomycosis, blastomycosis, cryptomycosis, candidiasis, and aspergillosis. Immunocompromised patients are at risk for developing infections with atypical mycobacteria. Fungal infections also have become more common with the increasing use of broad-spectrum antibiotics, especially in combination with central venous catheters for parenteral nutrition (Fig. 3).

Suspicion of a mycobacterial infection is the basis for establishing the diagnosis. Patients from Southeast Asia or South America, prison populations, and frequenters of homeless shelters are at high risk for contracting TB. A patient with a family member or household contact with TB also should be considered as at high risk. Laboratory tests are usually nonspecific. A leukocytosis may or may not be present. The ESR may be normal in up to 25% of cases. Although the purified protein derivative skin test can help detect active infection or past exposure to TB, the test is not fully reliable because of false-negative results that can occur in the malnourished and the immunocompromised. Polymerase chain reaction for detection of tuberculous infection holds great promise for a faster diagnosis.

Pediatric Diskitis

The highly variable clinical presentation of a child with diskitis may lead to delays in recognition and diagnosis. Active children may often associate the onset of pain with some activity or minor trauma. In the absence of systemic symptoms of infection, further workup is necessary if the pain does not resolve in 1 to 2 weeks. In general, however, vertebral infection should be suspected when the child has a low-grade fever and pain, refuses to bear weight, or assumes a flexed position of the spine. The patient also may complain of abdominal pain. These nonspecific findings are more common in children over the age of 5 years.[17-19] In contrast, infants are more likely to be systemically ill. Older children are more likely to be able to identify the spine as the source of pain. Although uncommon, these same symptoms can be observed with spinal tumors in children, such as Ewing's sarcoma.

The white blood cell count may or may not be elevated, but the ESR is usually mildly elevated and the CRP level, markedly elevated. Infants typically will demonstrate a leukocytosis and elevated ESR.[20] Blood cultures can be positive in up to 50% of cases.[19]

Acute infections are more likely to yield positive blood cultures.[19] Certainly the child who appears ill and febrile should have all possible sources of infection cultured. If a biopsy is needed, it can be done under CT guidance; a 60% to 70% yield rate for infectious lesions can be expected.[21] If a trial of antibiotics was initiated prior to biopsy without response, antibiotics should be suspended for 3 to 4 days before the procedure to ensure greater accuracy from the cultures.

A B

Figure 3 A 40-year-old woman with rheumatoid arthritis and chronic steroid use developed severe back pain and paraplegia after treatment with broad-spectrum antibiotics for necrotizing fasciitis. **A,** Lateral radiograph of the lumbar spine shows bony destruction of the end plates of L2 and L3. **B,** T2-weighted sagittal MR image of the lumbar spine demonstrates diskitis and vertebral osteomyelitis at L2-3, with severe canal stenosis from an epidural collection (arrowhead). Cultures taken at the time of anterior débridement were consistent with a *Candida* infection. The patient obtained pain relief and improvement in motor function after aggressive anterior débridement and reconstruction with an autogenous tricortical iliac graft and 6 weeks' administration of intravenous liposomal amphotericin B.

Epidural Abscess

The presence of a spinal epidural abscess is usually associated with the occurrence of diskitis or vertebral osteomyelitis. Rarely does an epidural abscess occur hematogenously without spinal involvement. This condition is caused by direct seeding of bacteria into the epidural venous plexus, in contrast with the more common route of local extension from adjacent disk or bone. In the absence of diskitis or vertebral osteomyelitis, an epidural abscess can be difficult to diagnose and can progress rapidly, with devastating consequences; prompt diagnosis and early treatment are critical in these rare cases. Risk factors for the development of epidural abscess include history of intravenous drug use, diabetes, trauma, obesity, percutaneous or open procedures (eg, spinal surgery, nerve or epidural block, or diskography), HIV, and renal failure.[22-26] Patients may present with back pain, progressive neurologic deficit, or fever. Although leukocytosis may not be present, the ESR is almost always elevated.

Radiographic Evaluation

Imaging studies are crucial to localize the infection, assess the extent of involvement, and determine the response to treatment. Radiographs may demonstrate progressive osteolysis and end plate destruction, often best seen on the anteroposterior view (Fig. 2, A). As the disease progresses, the disk space narrows and eventually collapses (Fig. 3). Plain radiographs, however, may not demonstrate abnormal findings for up to several weeks after the process has begun. Soft-tissue extension must be suspected in the presence of an abnormal psoas shadow, widening of the mediastinum (Fig. 2, A), or enlargement of the retropharyngeal soft-tissue shadow. The presence of gas in the soft tissues suggests an infection with an anaerobic organism.

In contrast with pyogenic infections, skeletal radiographs in a tuberculous infection often demonstrate vertebral destruction with relative preservation of the disk spaces. As the infection progresses, the disk is also destroyed and a kyphotic deformity may be present, especially in the thoracic spine. A chest radiograph always should be obtained to assess for active pulmonary disease.

In pediatric diskitis, radiographs of the spine should be assessed for disk space narrowing, end plate erosions, bony destruction, and paravertebral soft-tissue swelling. These changes may not occur for several days or weeks after onset of symptoms. They usually persist, eventually leading to disk space narrowing or autofusion.[18,27] Although late kyphosis is rarely seen in pediatric spinal infections, a notable exception is infantile osteomyelitis, which generally is associated with more initial bony destruction and resembles congenital kyphosis in late stages.[20]

Radionuclide studies can be much more sensitive than radiographs in detecting early infections. Technetium 99m bone scintigraphy is sensitive (~90%) but nonspecific, especially in adults with degenerative joint disease.[28] Because the study is dependent on local blood flow, false-negative results have occurred in areas of relative ischemia in very young and elderly patients. In pediatric vertebral osteomyelitis, the technetium 99m bone scan is positive in 74% to 100% of cases,[17,19] facilitating earlier diagnosis of diskitis in children. Wenger et al[19] showed that use of bone scans allowed diskitis to be diagnosed an average of 8.3 days earlier than without.

When used in conjunction with technetium 99m scans, gallium 67 citrate scans have high sensitivity and specificity in detecting foci of infection. The tracer, an analog of ferritin, is secreted by leukocytes at sites of infection. Gallium scans also normalize during the recovery phase and may be used to follow treatment response. This test, however, may not be effective in leukopenic patients and may not detect low-virulence organisms. Indium 111-labeled scans have a poor sensitivity in vertebral osteomyelitis (17%) and are not recommended.[29]

CT is useful in delineating the extent of bony destruction and soft-tissue extension and is helpful in preoperative planning. However, the status of the neural elements cannot be accurately assessed without the use of myelographic dye, which is contraindicated in suspected infection because it places the patient at risk for developing meningitis or arachnoiditis. Although the CT scan with intravenous contrast also can demonstrate soft-tissue extension, distinction between abscess and granulation tissue may be difficult.

Magnetic resonance imaging (MRI) is the modality of choice in the diagnosis and evaluation of spinal infections because it provides excellent imaging of the soft tissue, neural elements, and inflammatory changes in the bone (Figs. 2, B and 3, C). MRI has an extremely high sensitivity (96%) and specificity (93%) in detecting infections of the vertebral column.[28] It is noninvasive, allows detection of paravertebral and epidural extension, and clearly visualizes neurologic structures. T1-weighted sequences demonstrate decreased signal intensity in both the vertebral body and disk from edema. T2-weighted images show increased signal intensity in both the vertebral body and disk with loss of the normal intranuclear cleft (Fig. 1).

The administration of gadolinium in combination with MRI improves resolution and allows an infectious process to be distinguished from

degenerative changes of the end plate and intervertebral disk (Fig. 4). The vascular-based enhancement also allows differentiation of an epidural granulation from an epidural abscess. An epidural mass may be isointense or hypointense on T1-weighted images, shows high signal on T2-weighted images, and may show peripheral enhancement visible with gadolinium.[30] Short T1 inversion recovery sequences often can help to differentiate an infection from other pathologic entities. Even with MRI, however, granulomatous infections can be difficult to distinguish from tumors of the spine. Thus, a biopsy is often required to make a definitive diagnosis.

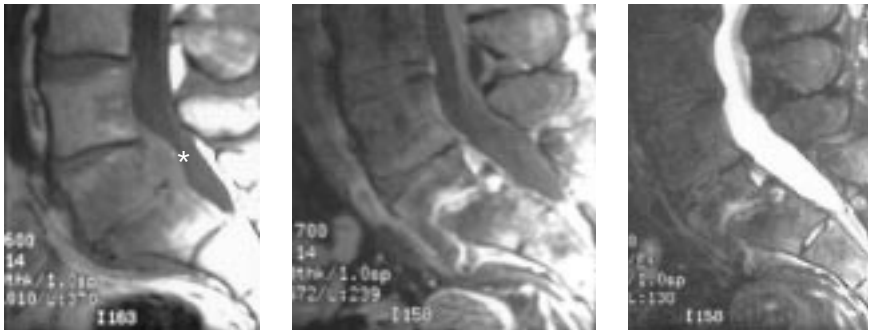

Figure 4 A 38-year-old man with HIV and a CD4 cell count of 20 presented with back pain of several weeks' duration and no radiculopathy. **A,** T1-weighted sagittal MR image shows edema at the L5-S1 disk space and adjacent end plates. The asterisk (*) indicates an epidural collection consistent with an epidural abscess. **B,** T1-weighted gadolinium-enhanced sagittal MR image shows uptake at the L5-S1 disk space and the epidural collection. **C,** T2-weighted sagittal MR image shows no notable canal compromise by the anterior collection. However, there is severe destruction of the adjacent bone of L5 and S1.

Treatment

Pyogenic Infections

The goals for treatment of spinal infections should be to establish a diagnosis and identify the pathogen, eradicate the infection, prevent or minimize neurologic involvement, maintain spinal stability, and provide an adequate nutritional state to combat infection. Establishing a diagnosis and identifying the pathogen is of primary importance. Once the organism has been identified, intravenous antibiotic therapy should be initiated according to the culture results and sensitivities. A course of 2 to 6 weeks of parenteral antibiotics is usually recommended. This is followed by a course of oral antibiotics, depending on the virulence of the organism, susceptibility of the host, and other factors, such as retained hardware. Conversion to oral antibiotics should be made only with clinical improvement, normalization of the ESR and CRP level, or resolution of the infection as demonstrated in imaging studies.

In addition to antibiotic therapy, immobilization, rest, and proper nutrition are recommended. Molded contact braces are effective in the lumbar region, whereas a halo or a rigid cervicothoracic orthosis may be required for cervical osteomyelitis. Immobilization of the affected area aids in pain relief and helps prevent deformity.

Surgery is indicated in five circumstances: to obtain a tissue diagnosis after a failed closed needle biopsy or from a location inaccessible by closed methods; for drainage of an abscess that is causing sepsis or neurologic deficit; to treat neurologic deficit secondary to compression either by the infection (abscess or granulation) or structural destruction; for structural instability or deformity; or for failure of medical management to reduce persistent symptoms or elevated laboratory measurements.

The location of the infection and the intended purpose of the surgery often dictate the surgical approach. Because the majority of these infections involve the vertebral body and the disk, an anterior approach is most commonly used to maximize access to the infected tissue. A posterolateral approach to the thoracic spine may be considered in certain instances, or a costotransversectomy if only culture, biopsy, or abscess drainage is necessary. Because these and endoscopic approaches avoid thoracotomy, they may cause less morbidity in the medically fragile patient.

If an anterior approach is used for débridement and decompression of the spinal canal, reconstruction should be done with an autogenous structural graft, such as tricortical iliac crest or middle third of the fibula. Iliac crest is preferable because of the abundant amount of cancellous bone. Fresh-frozen allografts in combination with autogenous bone may be considered for structural support, but structural autogenous bone grafts are preferred. Vascularized bone grafts have gained popularity during the last decade because of their intrinsic blood supply and faster rate of incorporation. In the thoracolumbar junction, a vascularized rib graft may be used, and in the lumbar spine, vascularized rib or iliac grafts.[31-34] Recently, titanium surgical mesh filled with autogenous bone has been used as an alternative to structural autogenous graft. Depending on the degree of preoperative kyphosis and length of the reconstruction, a posterior fusion

with instrumentation may be required to adequately stabilize the spine. This is usually undertaken 1 to 2 weeks after the initial surgical débridement. The staging of the procedures allows for an interval of intravenous antibiotics and optimization of medical and nutritional parameters before placement of the instrumentation.

Hyperalimentation is an effective way to maximize the patient's nutritional status before and after surgery and between stages. The infection places the patient in a catabolic state because of metabolic losses that have occurred before the diagnosis of infection is made. The goal of nutritional supplementation is to restore the patient to the premorbid nutritional status. Nutrition consultation and monitoring of laboratory measurements are helpful in reaching a positive nitrogen balance. These include achieving a serum albumin level >3 g/dL, an absolute lymphocyte count >800/mL, and a 24-hour urine creatinine excretion >10.5 mg in men and >5.8 mg in women.

Tuberculosis

Once the diagnosis of a tuberculous infection is established, aggressive treatment is necessary to eradicate the infection. A four-drug regimen of isoniazid, rifampin, ethambutol, and pyrazinamide is used as first-line therapy for 6 months. The response to treatment is assessed by routine clinical examinations and radiographs. The emergence of multidrug-resistant mycobacteria will provide further challenges in the treatment of these infections in the future.

Indications for surgery in tubercular infections are the same as for pyogenic infections. The most common surgical technique, the Hong Kong procedure, involves débridement of infected bone, decompression of the spinal canal, and correction of the kyphotic deformity using structural grafting[35] (Fig. 5). Additional posterior fusion with instrumentation also may be required. The second procedure can be either staged or done on the same day, depending on the tolerance of the patient. Autogenous iliac crest or fibula is ideal for structural grafting. Rib graft alone has been shown to be inadequate unless a vascularized rib is used to accelerate the rate of incorporation. The Hong Kong procedure is preferred over anterior débridement alone because the addition of an anterior strut corrects and prevents progressive kyphotic deformity. Laminectomy without adjunctive stabilization is contraindicated because damage to the posterior structures in the presence of weakened anterior structures will lead to progressive kyphosis and neurologic injury.

Failure of medical treatment or development of neurologic deficit is

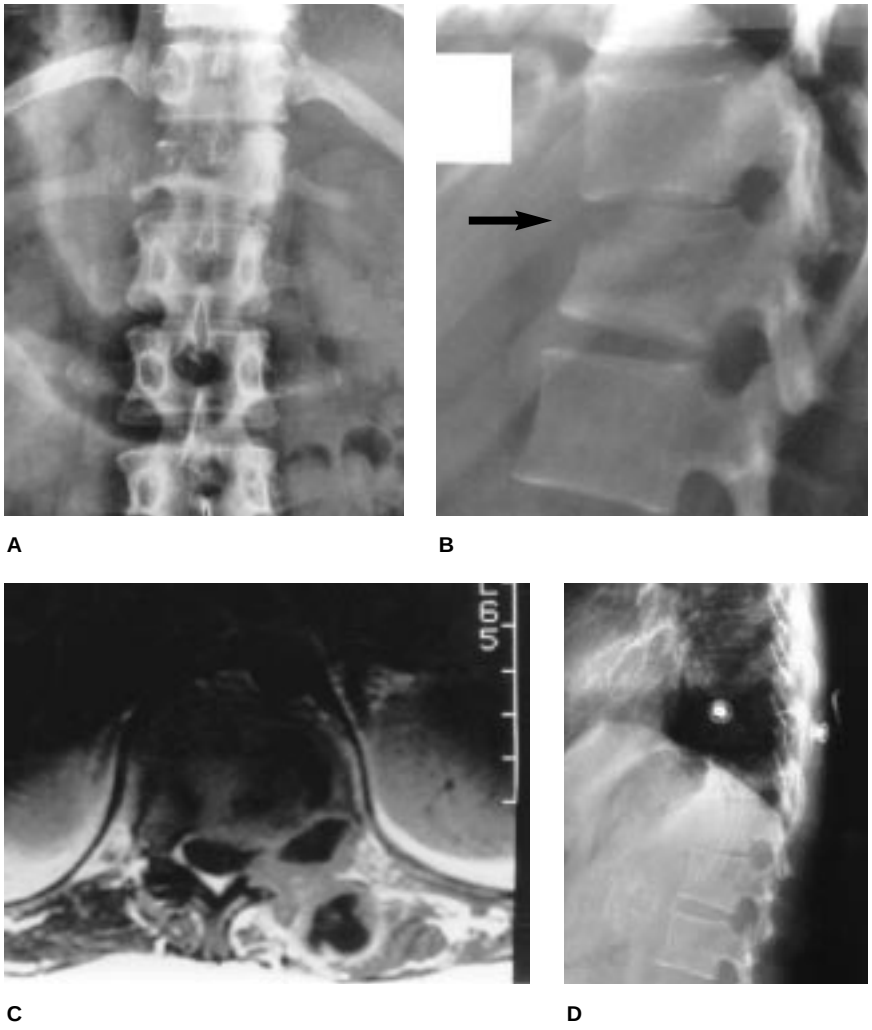

Figure 5 A 22-year-old woman presented with a long history of back pain. Anteroposterior **(A)** and lateral **(B)** radiographs show erosion and partial collapse of the T12 vertebral body (arrow). **C,** T1-weighted MR image demonstrates extensive anterior and posterior column involvement. Because of atypical MR image findings, a posterior biopsy was performed, which revealed TB. **D,** Postoperative lateral radiograph. Because of partial collapse and extensive involvement, the patient underwent anterior reconstruction using autogenous rib graft.

a clear indication for surgical débridement, decompression, and stabilization. Early decompression will maximize the patient's functional recovery. A more chronic neurologic deficit due to cord compression over structural deformity also may be treated with decompression and stabilization. However, the prognosis for neurologic recovery in the face of chronic deficits is not as optimistic.

Pediatric Diskitis

Whether diskitis in children is infectious or inflammatory in origin remains controversial. Although the recommended treatment will vary depending on the suspected origin, immobilization with casting or bracing is uniformly recommended. The use of antibiotics has been controversial, with satisfactory results reported in several studies regardless whether a patient received antibiotics. Scoles and Quinn[18] reported that all patients were asymptomatic at the time of hospital discharge, whether or not antibiotics were administered. In addition, none of these patients had a relapse. In contrast, Ring and Wenger[36] observed that patients treated with intravenous antibiotics for at least 6 days had a more rapid resolution of symptoms and the lowest likelihood of developing recurrent symptoms. Oral antibiotics or no treatment were more likely to lead to prolonged or recurrent symptoms. Based on their experience, they felt that a short course of parenteral antibiotics was more likely to result in rapid relief of symptoms and a lower incidence of recurrent symptoms. Crawford et al[17] reserved antibiotics for patients who failed to respond to immobilization, bed rest, traction, or casting.

Epidural Abscess

Surgical drainage is almost universally recommended for treatment of an epidural abscess (Fig. 4). Conservative management of epidural abscesses, however, may be appropriate if the patient has no neurologic deficit, if the involvement is extensive, if the patient is not expected to survive surgery, or if paralysis has been present for >48 hours so that neurologic improvement would be unlikely.[26,30] For example, patients with lumbar involvement, no neural compromise, and diagnostic cultures can be effectively treated with intravenous antibiotics. As with osteomyelitis, from 2 to 6 weeks of intravenous antibiotics is usually recommended. An extended period of oral antibiotics may be necessary depending on the immunocompetency of the patient and the sensitivity of the organism.

Patients with neurologic deterioration are best managed with surgical decompression and débridement in addition to antibiotic therapy. Anterior abscesses, particularly with vertebral body involvement, should have anterior débridement. This can be done using either an open or endoscopic approach. Posteriorly located infections can be adequately treated by a laminectomy. Patients with extensive involvement can be treated through multilevel laminectomies. However, care should be taken not to remove more bone than is indicated for decompression because of the risk of postlaminectomy deformity. Prompt and aggressive treatment of neurologic compression appears to favorably affect neurologic recovery.[26]

Summary

The most common types of vertebral osteomyelitis are hematogenous bacterial or fungal infections (pyogenic or granulomatous), pediatric diskitis, epidural abscess, and postoperative infections. Successful diagnosis and treatment depend on an appropriate index of suspicion. The optimal management of patients with spinal infection requires understanding the circumstances that resulted in the infection, the organism involved, and the degree of bony and neurologic compromise. Early detection and medical treatment may obviate the need for surgical intervention. When surgical débridement is indicated, its prompt initiation appears to result in good clinical outcomes. In addition, maximizing the patient's nutritional status with hyperalimentation improves the outcomes of both medical and surgical treatment.

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