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REVIEW

Pyogenic spondylodiscitis: An overview

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Summary Although uncommon, spontaneous and postoperative pyogenic spondylodiscitis entail major morbidity and may be associated with serious long-term sequelae. A review of the literature was done to advance our understanding of the diagnosis, treatment, and outcome of these infections.

The principles of conservative treatment are to establish an accurate microbiological diagnosis, treat with appropriate antibiotics, immobilize the spine, and closely monitor for spinal instability and neurological deterioration. The purpose of surgical treatment is to obtain multiple intraoperative cultures of bone and soft tissue, perform a thorough debridement of infected tissue and decompression of neural structures, and reconstruct the unstable spinal column with bone graft with or without concomitant instrumentation. Appropriate management requires aggressive medical treatment and, at times, surgical interventions. If recognized early and treated appropriately, a full recovery can often be expected. Therefore, clinicians should be aware of the clinical presentation of such infections to improve patient outcome.

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Introduction

Spinal infections are an uncommon but important clinical problem that often requires aggressive medical and surgical management. The spectrum of spinal infections includes discitis, osteomyelitis, epidural abscess, meningitis, subdural empyema and spinal cord abscess. Only discitis and osteomyelitis, usually occurring in conjunction with one another, will be discussed here and thus the term spondylodiscitis will be used in this review when referring to these types of infections.

In this article, we will discuss the diagnosis, treatment and outcome of spontaneous as well as postoperative bacterial pyogenic spondylodiscitis. Spondylodiscitis due to brucella, mycobacterial pathogens and fungi will not be covered.

Pathogenesis

Pathogenic organisms reach the spine hematogenously through arteries or veins (via Batson's plexus), or by direct inoculation from a diagnostic or a surgical procedure.

In children, isolated infection of the intervertebral disc occurs initially with subsequent involvement of the adjacent end-plates. The anastomoses present between the equatorial and the circumferential superficial metaphyseal arteries gradually shrink, eventually atrophying by the age of 15 years.

In adults, the subchondral spongy bone is supplied by nutrients end arteries where a small septic embolus may lodge in the setting of bacteremia and begin to proliferate leading to bone infarct and subsequent osteomyelitis. The spread of infection

to adjacent vertebral bodies occurs through bridging anastomotic vessels from one metaphysis to another [1]. After the infection settles into the subchondral space, it usually spreads contiguously into the disc, causing both an osteomyelitis and discitis. The infection can then spread across the disc into the adjacent contiguous end-plates [2]. Unlike spontaneous spondylodiscitis in the adult population which originates in the vertebral body and secondarily invades the disc space, postoperative or iatrogenic spondylodiscitis is caused by direct involvement of the disc space.

Pyogenic spontaneous spondylodiscitis

Pyogenic spontaneous spondylodiscitis is often the result of hematogenous spread from either the skin, respiratory tract, genitourinary tract, gastrointestinal tract or the oral cavity, giving rise to bacteremia [3–5]. In adults, the disc is avascular and the organisms invade the end-arterial arcades in the metaphyseal region adjacent to the disc. The infection then spreads by direct extension with rupture of the infective focus through the end-plate into the disc. It may extend from the vertebral body to the subligamentous paravertebral area, the epidural space, and contiguous vertebral bodies [6]. In around 5% of patients, a history of blunt trauma to the spinal column can be elicited. Approximately 37% of pyogenic spontaneous spondylodiscitis will not have an identifiable source [7].

The common organisms include *Staphylococcus aureus* and streptococcus species and in intravenous drug abusers Gram-negative bacilli are frequently isolated. *Mycobacterium tuberculosis*, fungal infections and parasitic infestations are

uncommon but are usually seen in immunocompromised patients [3].

Epidemiology

The incidence of acute hematogenous nontuberculous vertebral osteomyelitis is estimated to be 5–5.3 per million patients per year with a male predominance. A study from France reports an annual incidence of spondylodiscitis of 2.4 per 100,000 person-years that almost triples for ages older than 70 years [8]. However, some studies suggest that this incidence is rising, possibly due to an increase in the rate of nosocomial infections associated with vascular devices and other forms of instrumentation [9] and to an increasing prevalence of intravenous drug abuse [10]. Prompt and aggressive management of central line bacteremias can reduce the incidence of paraspinal and vertebral infections [11]. Males are more commonly affected than females in the ratio of 2:1 for unknown reasons. The average age at clinical presentation is in the fourth to fifth decades. The most common level of involvement is in the lumbar spine, followed by the thoracic, cervical and sacral levels [12]. Involvement of the cervical spine occurs in 6.5% of spinal infections, whereas thoracic involvement has been reported to occur in 35% of cases [13].

Clinical presentation

Sapico and Montgomerie reported that 50% of patients had symptoms lasting for greater than 3 months before the diagnosis is established [13]. In this same series, pain was present in 90% and fever in only 52% of patients; however, chills or fever spikes were rare. Pain, which is the predominant symptom, is generally localized to the spine, exacerbated by movement and may radiate to the abdomen, hip, leg, scrotum, groin or perineum [14]. Radicular symptoms are present in 50–93% of cases [15]. Paravertebral muscle tenderness and spasm, and, limitation of spine movement represent the predominant signs in patients with spondylodiscitis. Neurologic complications such as spinal cord or nerve root compression and meningitis occur in approximately 12% of patients [12]. The progression of spinal pain to radicular signs followed by weakness and paralysis suggests the formation of an epidural abscess [16] or a kyphotic collapse of the infected level. Eismont et al. found that sensory involvement is rare whereas motor and long-tract signs are more common because of mainly anterior cord compression [17].

Laboratory findings

The white blood cell count (WBC), often normal, may be elevated in 35% of patients but rarely exceeds 12,000 cells/mm³. An elevation of the erythrocyte sedimentation rate (ESR), although non-specific, is usually seen in almost all cases of spondylodiscitis. The ESR is usually above 40 mm/h on admission with a mean value of 85 mm/h (normal value 0–20 mm/h). With appropriate medical treatment, a progressive decline of the ESR is usually encountered [18]. Rath et al. have reported that the C-reactive protein (CRP), although non-specific, may be a more clinically useful index than the ESR, and should be used to follow the course of the disease [19].

Blood, urine and focal suppurative processes should be cultured. Blood cultures may be positive in approximately 50% of patients and are helpful in guiding the choice of antimicrobial therapy. An attempt should always be made to obtain direct cultures from the involved vertebral body and/or the disc space if an organism cannot be identified by less invasive culture techniques. Computed tomography (CT) or fluoroscopy directed percutaneous needle biopsy can be performed. Needle biopsy under CT guidance is reported to be safe and precise with a diagnostic accuracy rate ranging from 70% to 100% whereas open biopsies are diagnostic in more than 80% of patients [20]. In a review of spinal infections, Razak et al. showed an accuracy of 93.3% in open biopsy techniques [21]. However, the higher sensitivity of open biopsy is mitigated by higher associated morbidity [7]. Non-culture amplification-based DNA analysis is also highly sensitive and specific. It can complement standard microbiologic methods for identifying the cause of infectious spondylodiscitis and contribute to species-specific therapeutic orientation in patients with negative blood and disc aspirate cultures [22]. Whenever possible, antibiotics should be held until cultures have been obtained. In addition to bacterial cultures, cultures for fungi and mycobacteria should be obtained in cases where there is a higher suspicion for such infections based on a subacute presentation and a negative gram stain and bacterial culture.

Microbiology

A polymicrobial cause is unusual in pyogenic spontaneous spondylodiscitis and accounts for no more than 2.5% of the total number of cases. However, polymicrobial infections are much more common in sacral osteomyelitis (including anaerobic pathogens) related to a contiguous spread of infec-

tion from pressure ulceration. The main causative organism of pyogenic spontaneous spondylodiscitis is *S. aureus* [23]. Although its incidence remains quite low in spontaneous pyogenic spondylodiscitis, MRSA should be thought of in some settings, such as in patients from highly endemic countries like the United States, or those with prior colonization or infection with MRSA [24]. Staphylococci are followed in frequency by Gram-negative bacilli 4–30% of pyogenic cases) and streptococci/enterococci (5–30%). Gram-negative bacilli such as *Escherichia coli*, *Proteus* spp. and *Pseudomonas* spp. are often seen in association with immune deficiency, diabetes, intravenous drug use, and following procedures or infections involving the genitourinary and gastrointestinal tracts. Bacteria with low virulence such as *Staphylococcus epidermidis* and viridans streptococci may cause particularly indolent infections [25].

Anaerobic infections may account for 3% of axial skeleton infections. They are more common in diabetic patients and are caused mainly by *Bacteroides* spp., *Peptococcus* spp., and *Propionibacterium acnes* [8]. *P. acnes* was initially described in association with spinal fusion surgery but has been increasingly reported in native infections.

Unusual causative pathogens include *Salmonella typhi* and *paratyphi*, *Bartonella henselae*, *Clostridium perfringens*, *Coxiella burnetii*, *Capnocytophaga canimorsus*, *Echinococcus granulosus*, *Actinomyces israelii*, *Nocardia* spp., *Candida* spp., *Cryptococcus neoformans*, and *Scedosporium apiospermum*. Incriminated bacterial pathogens are summarized in Table 1.

Radiographic evaluation

Routine radiographs of the spine

It may take as long as 3–6 weeks after the onset of symptoms for definitive bone destruction to become evident [26]. The first radiographic sign of infection is irregularity of the vertebral end-plate of the infected level. As the infection progresses, the erosion of the end-plate and adjacent bone becomes more prominent, leading to narrowing of the disc space, segmental collapse, loss of lordosis and structural deformity. After a variable period of time, bone regeneration occurs with visible reactive sclerosis as new woven bone replaces necrotic trabeculae (8–12 weeks) [27]. Widening of the paravertebral space due to expansion of the inflammatory process outside the disc causes displacement of the paravertebral line on routine frontal radiographs. Successful treatment will usually produce fusion across the disc space while total

Table 1 Bacterial etiology of pyogenic spontaneous spondylodiscitis.

	Percentages
Gram-positive aerobic cocci	
<i>Staphylococcus aureus</i>	57
<i>Streptococcus pyogenes</i>	4.1
Coagulase-negative staphylococci	3.4
Other streptococci	2.0
<i>Enterococcus</i> spp.	0.7
Gram-negative aerobic bacilli	
<i>Escherichia coli</i>	10.5
<i>Proteus</i> spp.	6.7
<i>Pseudomonas aeruginosa</i>	5.7
<i>Klebsiella pneumoniae</i>	1.8
<i>Enterobacter</i> spp.	1.8
<i>Salmonella</i> spp.	1.8
<i>Serratia marcescens</i>	0.5
Anaerobic bacteria	
<i>Propionibacterium</i> spp.	2.0
<i>Bacteroides fragilis</i>	0.5
<i>Peptostreptococcus</i> spp.	0.5

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vertebral collapse can occur when there is no therapeutic intervention.

Radionuclide bone imaging studies

Radionuclide bone scanning with technetium-99m pyrophosphate is highly sensitive, yielding positive findings within the first 1–2 days of infection. Neither in vitro labeled leucocyte scintigraphy nor 99mTc-anti-granulocyte antibody scintigraphy is especially useful, because of the frequency with which spinal infection presents as a non-specific photopenic area on these tests. Sequential bone/gallium imaging and 67Ga-SPECT are currently the radionuclide procedures of choice for spinal osteomyelitis. The drawbacks of these tests include low specificity, poor spatial resolution, and lengthy procedure time. Fluoro-2-deoxy-D-glucose (FDG) PET is a promising technique for diagnosing spinal infection and has several potential advantages over conventional radionuclide tests. The procedure can be completed in a single session and is characterized by high sensitivity and superior image resolution compared to single-photon-emitting tracers [28]. In addition, a recent study found that PET/CT is more effective than magnetic resonance imaging (MRI) in distinguishing between tuberculous and pyogenic spondylitis [29].

The specificity of bone scintigraphy is in the range of 75–80%. Single Positron Emission Computed Tomography (SPECT) has slightly better specificity [30]. Specificity can be increased by using indium-labeled leucocytes. All these modal-

ities are of very limited use with the availability of MRI.

Computed tomography (CT) scanning

CT scan is valuable in the assessment of septic involvement of bone and soft tissue [31]. It is a particularly useful tool for surgical planning and has further facilitated the performance and improved the yield of biopsies from the spine and adjacent phlegmons. In fact, diagnostic failure of needle biopsy is not uncommon and a second CT-guided biopsy or a surgical (open) biopsy is advisable. The zones of end-plate erosion are usually more obvious on CT than on routine radiographs, and are seen earlier in the course of the disease.

Magnetic resonance image (MRI) scanning

MRI has a higher sensitivity than the bone scan and became the gold standard in the evaluation of pyogenic spondylodiscitis. The findings on MRI are characteristic and occur early in the disease. MRI was found to have a sensitivity of 96%, a specificity of 92%, and an accuracy of 94% in the diagnosis of spondylodiscitis [32].

The postinflammatory phase is histologically characterized by the presence of vascularized fibrous tissue, fat bone marrow transformation, subchondral fibrosis and osteosclerosis. These changes are clearly demonstrated by MRI. Moreover, MRI can be used for monitoring of the therapeutic response during the course of spinal

infection [33]. MRI appears to give the correct diagnosis or suggest pyogenic spondylodiscitis as a possible diagnosis in 55% and 36% of cases, respectively in patients who present with less than 2 weeks of symptoms. After 2 weeks, the percentage of correct and possible diagnosis of pyogenic spondylodiscitis is reported to be 76% and 20%, respectively. The earliest MR abnormalities are caused when edema and inflammatory cells infiltrate the vertebral body and disc space. The MR signal within the bone becomes diminished causing darkening of the marrow on T1-weighted images (Fig. 1a) and brightening of the marrow on T2-weighted sequences (Fig. 1b). The intervertebral disc will also become bright on T2-weighted images because of the increase in overall water content. T1-weighted scans performed following the intravenous infusion of gadolinium-diethylene-triamine pentaacetic acid (Gd-DPTA) contrast agents may show enhancement at the end-plate–disc interface fairly early in the course of the infection (1–2 weeks) (Fig. 2). Enhancement will spread away from the disc as the destructive process progresses. Fat signal-suppressed sequences correlate closely with T2-weighted images, showing increased signal intensity within the involved marrow [34]. However, caution should be used in interpreting follow-up MR images for the assessment of the therapeutic response as MRI scans ordered in patients responding well clinically often give conflicting results.

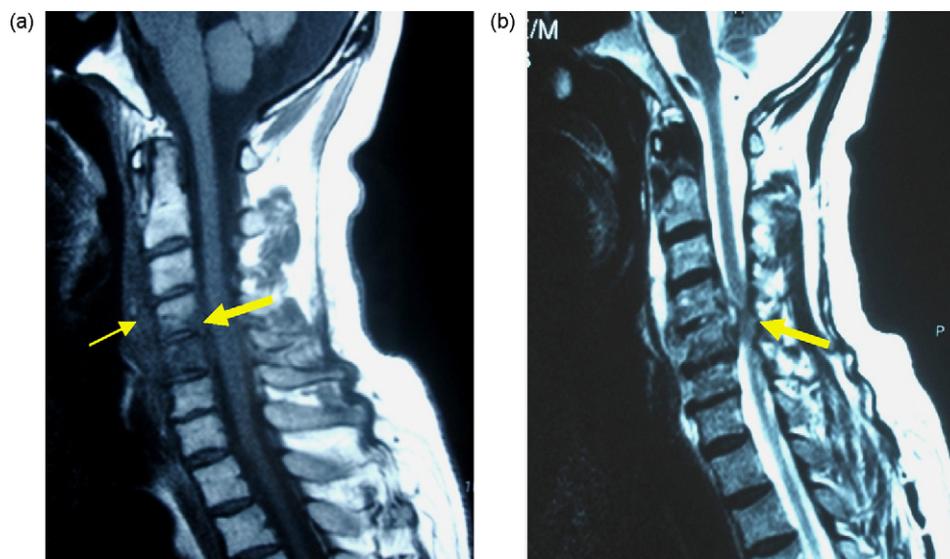


Figure 1 (a) T1-weighted sagittal image shows decreased signal intensity from C4, C5 and C6 vertebral bodies and presence of an epidural collection that extends from the mid-aspect of C4 to the inferior aspect of C5 (large arrow). In addition, there is evidence of a paravertebral fluid collection along the anterior aspect of the cervical spine extending from the inferior third of C2 to the level of C7–T1 disc space (small arrow); (b) T2-weighted sagittal image shows increased signal intensity from C4, C5 and C6 vertebral bodies. This is associated with increased signal from the C4–C5 intervertebral disc and evidence of an epidural collection at C4–C5 disc space level (arrow).



Figure 2 (a) T1-weighted sagittal image without gadolinium shows C3 and C4 vertebral bodies as well as their posterior corresponding elements with diffusely decreased signal intensity (arrow), with complete loss of the corresponding disc space associated with collapse of the lower end-plate of C3 and upper end-plate of C4, causing focal kyphosis of the cervical spine at that level, and evidence of spinal cord compression; (b) T1-weighted sagittal/axial images after gadolinium, respectively show significant enhancement in the anterior soft tissue (large arrow) as well as in the epidural space at the C3–C4 levels (small arrow).

Differential diagnosis

The differential diagnosis for adults presenting with back pain includes degenerative or metastatic spinal disease, disc herniation, vertebral compression fracture, and inflammatory spondyloarthropathies such as ankylosing spondylitis or reactive arthritis [25].

The differential diagnosis of pyogenic spondylodiscitis includes inflammatory, neoplastic, degenerative or granulomatous processes [16]. Inflammatory diseases such as pyelonephritis, appendicitis, abdominal abscesses and bowel infarction may have a similar clinical presentation to spondylodiscitis. Tumors of the spine, whether primary or metastatic, can occasionally simulate the radiological picture of infection. In general, however, spinal infections involve the disc whereas neoplasms involve the vertebrae and spare the disc. Degen-

erative diseases including disc herniation with disc space collapse, desiccation, bulge, end-plate erosion, or annular tear on MRI and osteoporosis with vertebral collapse also should be considered. Differentiating a pyogenic spondylodiscitis from a granulomatous etiology such as tuberculous infection can often be difficult, especially if cultures are negative. Other nontuberculous granulomatous infections involving the spine have been reported and must be considered in the differential diagnosis, including brucellosis, aspergillosis, candida tropicalis, blastomycosis and coccidioidomycosis [35].

Complications

The associated complications vary with the level of the spine involved and are related to the exten-

sion of the process to the surrounding tissues. Infections of the cervical spine can occasionally lead to a pharyngeal abscess whereas thoracic spine infection can be complicated by mediastinitis. Epidural abscess (Fig. 1b), subdural abscess, meningitis, loss of lordosis, segmental collapse with subsequent spinal instability and progressive neurological impairment may complicate spondylodiscitis involving any level (Fig. 2).

Epidural abscess is a serious complication, affecting 4–38% of cases of spontaneous spondylodiscitis. Chronic illness and diabetes increase the risk of epidural extension, which usually occurs in the anterior aspect of the canal, spreading from the posterior parts of the vertebral body and disc space. MRI with contrast can differentiate between epidural granulation tissue and an epidural abscess [36]. Epidural abscess carries a poorer prognosis than epidural granulation tissue. In one study, the rate of epidural extension complicating spondylodiscitis was 90% in the cervical spine, 33.3% in the thoracic spine and 23.6% in the lumbar spine, suggesting that spondylodiscitis affecting more cephalad regions of the spine carries a significantly increased risk of secondary epidural abscess. In addition, cephalad levels of involvement are more prone to the development of serious neurological deficit.

The long-term sequelae of vertebral osteomyelitis have recently been evaluated. There was a 33% rate of spinal dysfunction and only a 3% rate of severe dysfunction a median of 5.4 years after treatment. Neurological deficits, at least 8 weeks of delay in diagnosis, and chronic debilitating diseases were identified as predictors of worse outcome. Irreversible paralysis may still affect 4–22% of patients with SEA (spinal epidural abscesses). Long-term rehabilitation is often necessary [8]. The mortality rate has been reported in a range of 2–20% for vertebral osteomyelitis; it is around 5% for SEA.

Management and outcome

Spondylodiscitis is usually not recognized at an early stage, when the treatment is simple and effective, due to the non-specific nature of the symptoms at the onset of the disease. Early diagnosis is based on a high level of suspicion with emphasis on the following: (1) existing infectious focus; (2) presence of risk factors such as increased age, diabetes mellitus, rheumatoid arthritis, steroid use, ethanol abuse, immunosuppression, intravenous drug abuse (IVDA), infectious endocarditis, and history of recent surgical or invasive diagnostic spinal procedure [7,17,37,38]; (3) fever, localized spinal pain with paravertebral mus-

cle spasm, limitation of movement and evidence of neurological deficit; (4) laboratory studies that include ESR, WBC, blood cultures, purified protein derivative (PPD) test, CRP and direct cultures through fine needle or open biopsy; and (5) imaging studies.

The treatment of pyogenic spontaneous spondylodiscitis is either conservative or surgical. The goals of treatment should be to relieve pain, prevent or reverse neurologic deficits, eradicate infection, prevent relapse, and establish spinal stability.

Conservative treatment

The principles of conservative treatment include: (a) establishment of an accurate microbiological diagnosis; (b) treatment with appropriate antibiotics; (c) spinal immobilization; (d) careful monitoring for clinical and radiographic evidence of spinal instability and progression of infection or neurological deterioration.

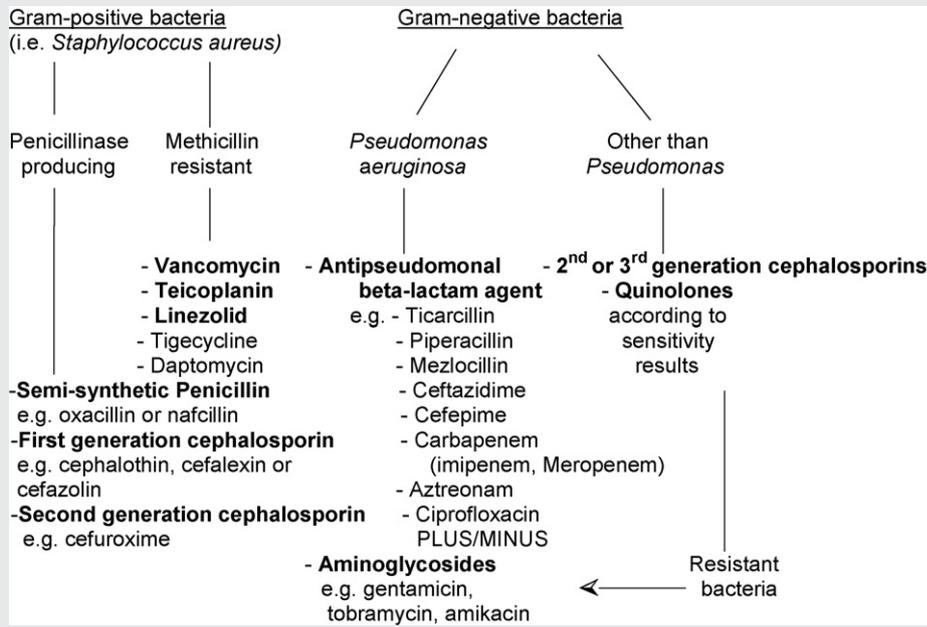
While awaiting laboratory and culture confirmation of infection, the spinal column should be immobilized. Specimens for microbiological studies should be taken from the portal of entry, and blood should be collected on three separate occasions for blood cultures, if possible during fever spikes or chills, after discontinuing any antipyretic agents. When these tests are negative, a percutaneous biopsy of the affected disc is in order. Blood cultures should be obtained routinely after the procedure [39].

Antimicrobial treatment should not be started until the organism is identified, except when clinical circumstances dictate otherwise, for instance in patients with neutropenia or severe sepsis [40]. The patient should be started on empiric broad antibiotic therapy based on the clinician's best assessment of the likely organism(s) [41], as well as the patient's risk factors (Table 2). Once an

Table 2 Likely offending organisms in pyogenic spontaneous spondylodiscitis, based on the patient's clinical history or physical findings.

Infection	Bacteria
Skin infection	<i>Staphylococcus aureus</i>
IVDA	<i>Pseudomonas aeruginosa</i>
Genitourinary tract infection	<i>Escherichia coli/Proteus</i> spp.
Respiratory tract infection	<i>Streptococcus pneumoniae</i>
Alcoholism	<i>Klebsiella pneumoniae</i>
Acute endocarditis	<i>Staphylococcus aureus</i>
Subacute endocarditis	<i>Streptococcus</i> spp.

Table 3 Antimicrobial treatment in pyogenic spontaneous spondylodiscitis.



organism has been identified, directed antibiotics should be administered intravenously (Table 3). Studies have reported that the incidence of treatment failure was higher when parenteral therapy was administered for less than 4 weeks [13,17].

Roblot et al. assessed the risk of vertebral osteomyelitis relapse and verified that this risk was not enhanced in patients who received 6 weeks of antibiotic therapy as compared with those who received a longer treatment (>6 weeks). Their results suggest that antibiotic therapy of vertebral osteomyelitis could be safely shortened to 6 weeks without enhancing the risk of relapse; however the follow-up of patients was only 6 months post-treatment [42]. The optimal total duration of antimicrobial therapy is unclear. In observational studies, treatment for less than 4 weeks [43], or 8 weeks [44] was associated with a marked increase in recurrence rates compared to treatment for longer than 12 weeks (>14%, 10%, and >15%, respectively, compared to 3.9%) [40]. Given that the diagnosis of pyogenic discitis and vertebral osteomyelitis is usually delayed, with a mean time from symptom onset of 6–7 weeks, Grados et al. suggested that antimicrobials should be given for at least 12 weeks, as recommended for chronic bone infections [40].

A useful measure of successful treatment is the CRP level. It is our practice to treat with parenteral antibiotics for 6 weeks (or longer) followed with oral antibiotics for an additional 4–8 weeks, according to the patient’s response. If stable, the

patients can be discharged home on IV antibiotics. When switching to oral therapy, high doses of antimicrobial agents should be used to ensure high levels in the bone. The choice of the antibiotic should depend on drugs with high bioavailability. Associated medical problems such as diabetes, renal failure and poor nutritional status require careful management to achieve a successful clinical outcome.

The role of bed rest and spinal immobilization are also very important, especially when there has been vertebral destruction. Cervical lesions should have collar or halo immobilization. Thoracic or lumbar infections should be treated with bed rest until symptoms of pain and spasm have diminished. The patient is then placed in a thoracolumbar sacral orthosis (TLSO) for thoracic infections and a lumbar sacral orthosis (LSO) for lumbosacral infections. Upper thoracic lesions should have a TLSO with a cervical extension. Spinal immobilization and activity restriction should be continued for 10–12 weeks or until radiographic evidence of healing is present.

Surgical treatment

The indications for surgical treatment are detailed in Table 4. The principles of surgical treatment include: (a) thorough debridement and removal of infected tissue; (b) decompression of neural elements; (c) restoration of spinal alignment; or (d) correction of spinal instability [31]. Surgical strategies for pyogenic vertebral discitis and osteomyelitis are presented based on the litera-

Table 4 Indications for surgical treatment in pyogenic spontaneous spondylodiscitis.

1. Failure to respond to conservative therapy
2. Significant or progressive neurologic deficits
3. Large paraspinal abscess with local mass effect or septic embolization
4. Significant osseous disease with involvement of two adjacent vertebral bodies, or greater than 50% loss in a single vertebral body
5. Progressive deformity with or without incapacitating spinal pain

ture and according to the practice of the authors, with an emphasis placed on structural considerations, implant selection, and techniques for augmenting vascular tissue to the site of infection [45]. In general, only the anterior vertebral elements are involved in infection (Fig. 3). Some degree of stability is usually maintained by intact posterior elements, thus preventing significant subluxation. Therefore, decompression laminectomy alone may further destabilize the spine and result in an increased neurological deficit [17,38]. This procedure is only indicated in cases of dorsally situated epidural abscesses. In selected cases, a localized disc space infection can be treated by a posterolateral debridement. However, in general we, along with most authors, advocate anterior procedures for extensive debridement of the disc and vertebral bodies back to healthy bone, and subsequent autologous bone grafting with the use of either an anterolateral [46–48] or a posterolateral approach [17,38,48,49] for thoracic and

lumbar lesions that preserves the laminae, facets, and pedicles. The use of autologous bone graft after adequate debridement has been advocated in several series [50]; however, recent studies have supported the use of titanium mesh cages to bypass the morbidity associated with structural autografts and the slow rate of graft incorporation associated with structural allografts [51]. The most common sources of grafts include tricortical iliac crest, rib and fibula. Furthermore, a considerable amount of literature is available on the management protocols advocating prolonged bed rest, despite surgery, owing to a reluctance to use foreign implants for fear of perpetuating the infection. Only a small number of recent reports have recommended additional posterior spinal fixation after anterior decompression and fusion depending on the quality of bone, the number of segments involved, and the presence of pre-existing kyphotic deformity [52–54]. A trend toward fewer postoperative complications was reported for patients who had posterior stabilization or titanium cages [55]. Harrington rod systems were used, requiring an extended posterior exposure, but more recently Rath et al. have reported that the debridement, autologous interbody bone grafting and internal fixation can be successfully done using a posterolateral approach allowing early mobilization of the patient [19]. The use of methylmethacrylate for bridging osseous defects was associated with persistence and recurrence of infection and thus should be avoided [19]. Recent studies have demonstrated that primary arthrodesis and instrumentation can be performed in acute spinal infection [56,57].

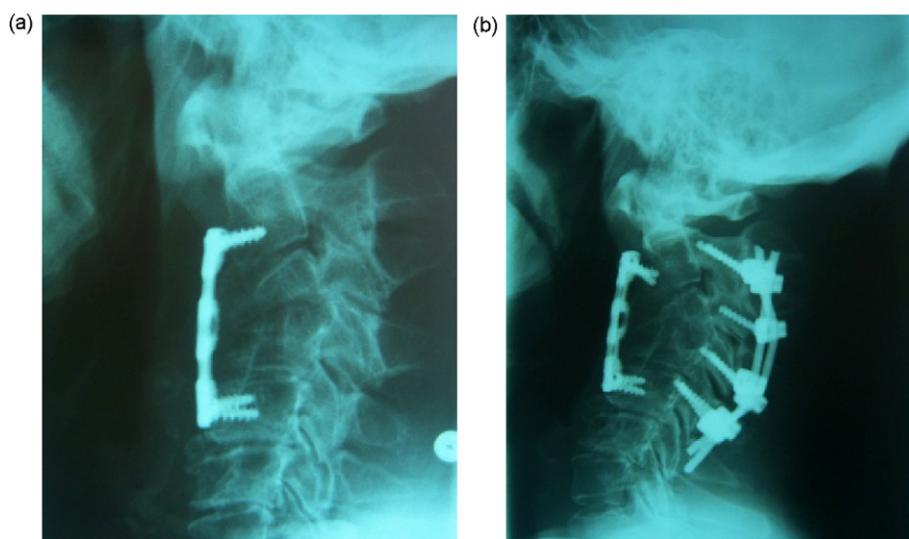


Figure 3 (a) Cervical spine X-ray (lateral view) performed after anterior cervical decompression and fusion with anterior Codman plate extending from C2 to C5; (b) cervical spine X-ray (lateral view) showing anterior plate failure. Posterior pedicle C2 and lateral mass screws and rods extending from C3 to C5 vertebral bodies.

Spinal instrumentation, used in the setting of subluxation or kyphotic deformity, is an extremely useful adjunct which can be successfully applied, provided a thorough debridement of infected tissue is performed with concomitant use of antibiotics.

Early surgical complications include wound infections, sepsis, pleural effusion, pulmonary embolism, cerebrospinal fluid fistula, ileus, ureteral damage, pneumonia, air leak, graft fracture and progressive neurological deficit. Multiple late complications have been reported and include graft resorption and fracture, nonunion, progressive kyphosis and refractory pain.

Postoperative and iatrogenic spondylodiscitis

Direct implantation of pathogen is the only way an adult, with avascular disc space, can acquire true discitis which is otherwise limited to the pediatric population.

The incidence of postoperative discitis after routine lumbar discectomy has been reported between 0.7% and 2.8% of operative cases [58]. When one adds a fusion to the procedure, the incidence rates rise from 0.9% to 6%. Spinal instrumentation adds further complicating factors, and infection rates average 7% with a range of 1.3–12% [59]. A recent study conducted at our center revealed the incidence of infection to be 2.7% [60]. Clearly, aseptic technique and appropriate antibiotic prophylaxis have dramatically reduced infection rates in the perioperative period [61]. Infection may also occur after lumbar puncture, myelogram, cervical laminectomy, lumbar sympathectomy, discography, chemonucleolysis and other procedures.

Several factors have been cited to increase the rate of infection following spinal surgeries, including increased age, poorly controlled diabetes mellitus, chronic malnutrition, steroid therapy, previously radiated area, pre-existing neoplasm, prolonged preoperative hospitalization, suboptimal sterile techniques, prolonged procedures, and increased operating room traffic. When compared to spontaneous spondylodiscitis, patients with postoperative spondylodiscitis were found in one study to be younger, with less frequent underlying diseases and a more prolonged interval between onset of symptoms and diagnosis [62].

The most common presentation of postoperative infection is an initial relief of symptoms with surgery followed by a return of back pain 2–6 weeks later, exacerbated by virtually any motion of the spine, and sometimes radiating to the hip,

leg, scrotum, groin, abdomen, or perineum. The cases are rarely acute and seldom present with a septic picture. Constitutional symptoms may include occasional fever, increased sweating, and chills. Almost all patients with postoperative discitis reported in the literature had paravertebral muscle spasm and limited range of motion of the spine [63]. Point tenderness is present in 33% of the cases [63]. The surgical site usually appears benign; only 10–12% of patients have signs of superficial wound infection and 0–8% have expressible pus [63]. Neurologic deficits are rare, and if present, a cauda equina from a recurrent disc or an epidural abscess should be suspected [64]. There is often lack of leukocytosis but the ESR is usually elevated. However, an elevated ESR can frequently be seen because of the underlying disease, but the trend of changes on serial ESR testing can be very helpful. Nonetheless, a persistent elevation of the ESR in a symptomatic patient may be indicative of indolent discitis. In postoperative spondylodiscitis, radionuclide tests are of limited use; the bone scan will often show increased uptake in the operative site due to the normal healing process. A gallium scan is likely to be of greater assistance and can be more indicative of the extent of infection. Plain radiographs are initially normal, and later (average 3 months) reveal decreased disc space height and blurring of the affected end-plates. MR imaging remains the test of choice and the findings are identical to those seen in spontaneous pyogenic spondylodiscitis. *S. epidermidis* is the most common pathogen in postoperative spondylodiscitis followed by *S. aureus*. Gram-negative organisms, including *E. coli*, *Pseudomonas aeruginosa* can also be incriminated [65].

Treatment of postoperative and iatrogenic spondylodiscitis should include analgesics, muscle relaxants (e.g. diazepam 10 mg PO TID), antibiotics, bed rest and immobilization in a brace or halo vest. We recommend that any suspected post-surgical infection be subjected to CT-guided needle aspirate and culture. If pyogenic infection is proven or strongly suspected, we recommend a minimum of 6 weeks of culture-specific intravenous antibiotics (or until ESR decreases significantly). Pending the identification of the organism, one should start with an anti-staphylococcal antibiotic (e.g. vancomycin with or without rifampin) and a broad spectrum anti-Gram-negative antibiotic, and then modify the regimen based on the sensitivity results. When the organism isolated is MRSA, and in view of the fact that low success rates have been correlated with vancomycin monotherapy, combination therapy or a newer anti-staphylococcal drug should be considered. Surgery should be reserved for cases of sepsis,

epidural abscess formation, and progressive neurological deficits. The surgical approach depends mainly on the extent of the problem. Patients discovered early in the course may be treated by re-exploration posteriorly. In more extensive cases or chronic cases, an anterior approach is recommended. A thorough debridement of necrotic tissue back to healthy bone and autologous grafting should be performed as in cases of hematogenous pyogenic spondylodiscitis.

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