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REVIEW ARTICLE

Diagnosis and treatment of spondylodiscitis in HIV-positive patients

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With an incidence between 1:100,000 and 1:250,000, spondylodiscitis is rare, but is increasingly reported due to longer life expectancy, risk factors, and comorbidities, with HIV+ patients being at greater risk. We reviewed the literature on the diagnostic tools, and on the benefits and drawbacks of different treatments of spondylodiscitis in HIV- positive patients. We discuss basic strategies and indications for surgery. Recently, the trend was toward early mobilization of patients after surgical treatment. Modern surgical and antibiotic treatment can prevent a recurrence in these patients. The decision to opt for conservative or surgical treatment should be made depending on the extent of infection and the responsible pathogen, without regard to HIV. However, these patients should be treated in a specialized hospital by an experienced interdisciplinary team of consultants.

Keywords : spondylodiscitis ; HIV ; immunodeficiency ; acquired immune deficiency syndrome ; CD-4-T-cell count.

INTRODUCTION

Although still relatively infrequent, spondylodiscitis is becoming more common due to an increasing susceptible population and better diagnostic tools (*8*). It may affect several anatomical structures and thus be described as spondylitis, discitis, spondylodiscitis, pyogenic facet arthropathy, epidural infections, meningitis, polyradiculopathy and myelitis (*36*). Frequently, at diagnosis, it cannot be determined exactly which anatomical structure was first infected since, as reported in literature, the first clinical manifestations arise between two to six months later (*29*). Early diagnosis and treatment improve the prognosis. Frequently, the patients suffer from unspecific back pain and are treated for degenerative disease of the spine.

Immunocompromised patients have a higher risk of developing spondylodiscitis (*33*). However, osteoarticular infections are relatively rare in HIV+ patients if intravenous drug abusers are excluded (*1*). Furthermore, in HIV+ patients, the lethality of musculoskeletal infections has been reported to be around 20% (*38*), and the incidence of spinal infections is significantly higher, even in non-intravenous drug users (*39*).

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We will discuss here the different diagnostic and treatment options for spondylodiscitis in HIV+ patients. Furthermore, we address the question whether HIV impedes the usual treatment strategies.

PATHOGENS AND DIAGNOSIS

Endogenous and exogenous infection pathways must be differentiated. Endogenous infection, which is the usual case, occurs where a pathogenic agent spreads haematogenously. In the vascularized subchondral bone, it finds its way into the bone marrow of the vertebral body, close to the endplates and near the longitudinal ligament. Exogenous infection may occur following spine surgery, infiltration, or invasive diagnostic procedures (*6*). In addition, it is important to discriminate accurately between non-tuberculosis (non-specific) and tuberculosis (specific) spondylodiscitis. *Staphylococcus aureus* is the predominant bacterial agent in 20-84% of non-tuberculosis cases (*22,30,40*).

Tuberculosis is the most common cause of spinal infection worldwide and accounts for 9% to 46% of cases in developed countries, with skeletal involvement in 1% to 3%. The spine is involved in about half of these cases (*8,34,37*). In HIV+ patients, the infection pathway is mostly endogenous (*35*). Among 2519 patients, only 1% with vertebral osteomyelitis were HIV+ (*10*). Weinstein *et al* have reported spinal tuberculosis in about 35% of HIV+ patients with spondylodiscitis (*39*).

Clinical symptoms, especially in the early stage, are uncommon. The patient should be examined for infected lesions, and assessed for his or her neurological status. Symptoms include pain on heel strike or axial compression and percussion. The patient often feels pain when bending forward and returning to upright position.

The first diagnostic step should be a laboratory examination, including leucocyte count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). In most reports, ESR is high in over 90% of cases, with mean values ranging from 43 mm/h to 87 mm/h (*2,8*), and also CRP in most cases. CRP is thought to be the best response marker (*8,13*) and leucocyte count the least useful.

In HIV+ patients, CD4 blood count is crucial in determining the clinical course of spondylodiscitis. A mild-to-moderate decrease (> 200 cells/μL) indicates discitis and/or osteomyelitis that responds to appropriate antibiotics. Patients with a larger decrease (50-200 cells/μL) are more prone to develop spinal tuberculosis, with a very low CD4 count (< 50 cells/μL) more epidural abscesses (*39*). A CD4 T-cell count less than 100 cells/μL increases the probability of mixed infections. In HIV+ patients with suspected spinal infection, CD4-T cell count, white blood cell count, ESR and CRP levels should be checked, in addition to blood cultures.

At first plain radiographs should be made ; they have a sensitivity of 82%, a specificity of 57%, and an accuracy of 73% (*20*). However at this stage, they do not show any evidence of spondylodiscitis, but only minor changes, if any, such as endplate demineralization and/or irregularity (*19,24,32*). Later, the radiographs show how, as the infection progresses, it further destroys the vertebral body affecting the opposite end plate, eventually spreading through the anterior, lateral, and posterior surfaces. Although a paravertebral soft tissue mass with displacement of the surrounding structures may be seen, soft tissue contrast resolution is poor. If two neighbouring vertebral bodies are seen to be destroyed with a narrowed intervertebral disc, spondylodiscitis is the correct diagnosis (*14,26*) which can be best achieved using MRI .

In HIV+ patients, the physician must first distinguish between TB and pyogenic spondylitis in order to treat the specific type of infection. TB spondylitis manifests mainly as bone destruction with relative preservation of the disc, pyogenic spondylitis mainly as disc destruction (discitis) with mild-to-moderate peri-discal bone destruction. In TB, contrast enhancement is focal and heterogeneous, in the other case, relatively diffuse and homogenous. In addition, on the one side a paraspinal area of abnormal signal intensity is well-defined, on the other ill-defined with peri-discal rim enhancement. On the sagittal views, an intraosseous rim enhancement of the vertebra may occur (*3*).

In any case, injection of contrast medium is highly recommended during the procedure. In early stages of spondylodiscitis, even MRI, the gold stan-

dard, may show only minor subchondral changes to the endplate that may result in a misdiagnosis (e.g. Modic I, degenerative endplate change). If the clinical course raises suspicions, a second MRI after 2-3 weeks is imperative (*4*).

Antibiotic treatment is a pillar of therapy which requires accurate diagnosis with identification of the pathogen and its sensitivity to antibiotics. In HIV+ patients, considering the diversity of potential pathogens, this becomes vital. Furthermore, with antibiotic-resistant pathogens increasing, the causative agent must be precisely identified. Broad-spectrum therapy has been repeatedly shown to cause complications, such as *Clostridium difficile* - associated diarrhoea, and higher healthcare costs, and should be reserved for patients with severe sepsis, once blood cultures have been taken (*8,17*). The pathogen has been successfully identified in up to 85% of patients with spondylodiscitis (*18*), in our own collective of 20 HIV+ patients, in 75% of the cases (*35*). Failure is mainly due to previous systemic antibiotic treatment. If possible, antibiotic therapy should be initiated only after sample materials have been obtained.

Spinal infections are generally monomicrobial, frequently with a haematogenous source. Therefore, blood cultures are a simple and cost-effective method for identifying the pathogens. A positive culture can be expected in 40%-60% of clinically defined cases of pyogenic spondylodiscitis (*8,28*). The pathogen is often successfully identified not only in the acute phase of fever or in presence of sepsis, but also in clinically non-problematic cases of afebrile patients (*23*). Nevertheless, a high incidence of infective endocarditis (26%) has been reported during enterococcal and streptococcal spondylodiscitis. Routine echocardiography should be performed when these pathogens are suspected (*21*).

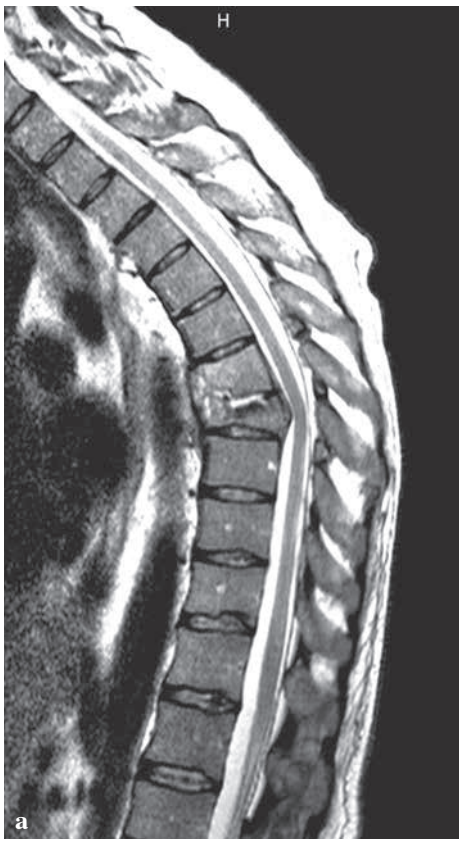
The pathogen can also be identified by percutaneous punch biopsy under anaesthesia, or CT-guided fine needle aspiration, possible in 40%-73% of the cases (*25,31*). Spinal biopsy leads to a direct change in management for 35% of patients, and is still worthwhile even if the patient has already been started on antibiotics. However, the pathogen can be identified more successfully prior to starting antibiotics (*25*). Otherwise, the treating physician should consider stopping this treatment for 2-3 days before the biopsy. Friedman *et a*. observed microbiological growth in 50% of cases after disc space biopsy in patients with spontaneous spondylodiscitis (*7*). Surgical sampling is the best technique to obtain biopsies to identify the pathogen, at best with two biopsies for histological and microbiological examination (*18*). In addition to culture, histologic examination of the specimen is helpful as it allows to discriminate between pyogenic and granulomatous origin of the disease. Regarding pathogen identification in HIV+ patients, the literature does not provide any specific recommendation.

TREATMENT

Conservative treatment

The existing literature offers no standardized guidelines as to the duration of intravenous antibiotic treatment. As a general rule, at least two to four weeks seem advisable to improve bioavailability ; less than four weeks may lead to higher treatment failure as reported in observational studies (*5,27*). In addition to antibiotic treatment, it is necessary to immobilize the affected region of the spine e.g. through reclining orthoses that distribute stress over the unaffected segments and their joints, thus providing relief to the affected ventral column. Wearing orthoses, patients can be fully mobilized. In elderly patients, well-known pathologies related to bed rest must be taken into account, e.g. decubitus ulceration, deep vein thrombosis, pulmonary embolism, and pneumonia. However, at least six weeks' bed rest is required in case of substantial defects of the anterior column or diseases affecting the lower lumbar or lumbosacral segments (*15*). In our study of HIV+ patients, only half of the conservatively treated patients wore a reclination brace, on average for 51 days. In addition, 4 operated patients also received such a brace. As their condition worsened under conservative treatment, 2 patients had to be operated (*35*).

Often the decision to operate is based on the patients' general condition, the stage of HIV disease, and life expectancy. Still, many surgeons fear post-operative complications, such as wound infections



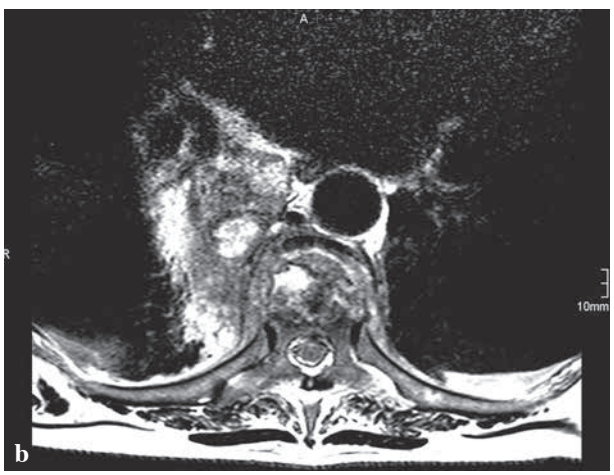


Fig. 1. — Spondylodiscitis at T7-T8 sagittal and axial T2-weighted MRI with contrast medium HIV FD 2001.

or delayed wound healing, even though, in this respect, the findings reported in the literature are inconsistent (*8*). Generally, if conservative treatment fails, surgery should be considered.

Table I. — Indications for surgery of spondylodiscitis

Indications for surgery of spondylodiscitis
1. Neurological deficits
2. Sepsis
3. Significant bone involvement with instability
4. Impending or current deformities
5. Intraspinal space-occupying process (i.e. spinal abscess)
6. Unclear aetiology of the process and/or suspected malignant disease
7. Failure to respond to conservative therapy
8. Uncontrollable pain
9. Patient's lack of compliance

Surgical treatment

I. Surgery aims to relieve compression of the spinal cord, or to drain epidural or paravertebral abscesses to improve spinal stability (*16*). Alternatives include dorsal instrumentation with pedicle screw-based systems (Fig. 2), either minimally invasive if no spinal decompression is required, or using open technique in combination with spinal canal decompression (Fig. 2). Surgery indications for spondylodiscitis are listed in table I.

HIV+ patients must be treated for spondylodiscitis in the same manner as other patients, with the objective, under appropriate pain management, to eradicate the underlying infection, to restore and preserve the spinal structure and stability, and to correct any neurological deficits. At present, due to the very heterogeneous patterns of the disease, no general treatment guidelines are available for spondylodiscitis, especially in combination with HIV infection.

Outcome

Excluding HIV-infected patients, spondylodiscitis has a mortality rate of less than 5%, ranging from 0 to 11%. Early mortality is related to uncontrolled sepsis (*8*). Previous studies suggest that the clinical presentation of spinal tuberculosis is similar in HIV+ and HIV- patients, with good outcomes of the mycobacterial disease. In our HIV+ population, the mortality rate is higher, with 5% of inpatients and 20% of outpatients (*35*). Weinstein *et al* reported an

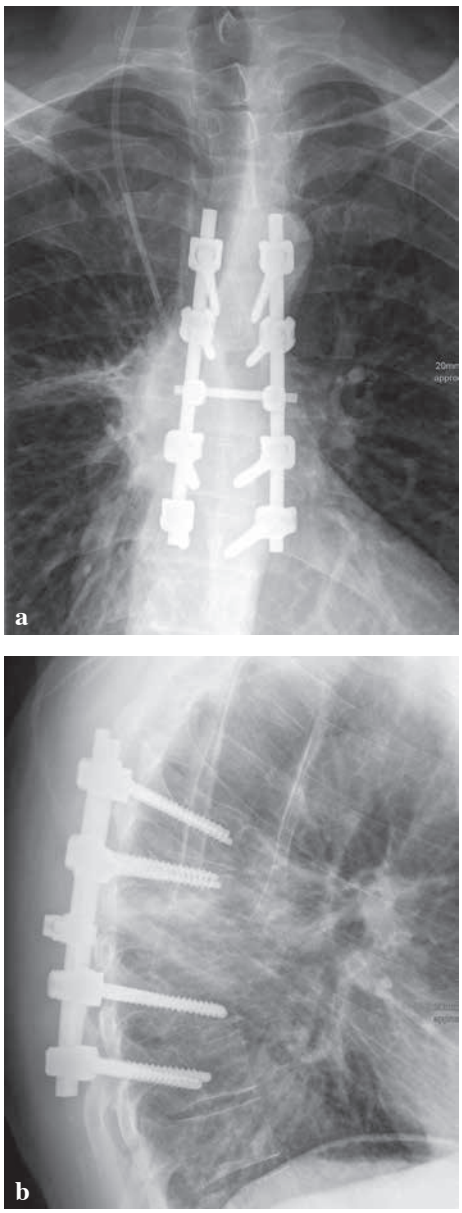


Fig. 2. — AP and lateral radiographs. after dorsal stabilization T6-T10 for spondylodiscitis at T7-T8 (Staph. aureus) HIV.

inpatient mortality rate of 17% (*39*). A cohort of 39 HIV-infected patients with spinal tuberculosis showed a 15% mortality rate within two years of surgery (*9*). Postoperative complications in HIV+ patients have been widely discussed as they have been reported with significantly higher frequency in such patients (*11*). In contrast, Horberg *et al* did not detect higher perioperative complication rates, except for pneumonia (*12*).

To determine the clinical presentation and the outcome of spondylodiscitis in HIV+ patients according to treatment, we performed a national, multicenter, retrospective case series of HIV+ patients presenting with spondylodiscitis between 1991 und 2007. Twenty patients were included in the study with a mean age of 43.0 years. The gender ratio M:F was 2.3:1. On admission, 50% of the patients were in CDC stage C3. The CD4 T-cell count averaged 237.5/L. HIV had been diagnosed 8.5 years previously on average. Radiologically, paravertebral abscesses were seen in 80.0%, epidural abscesses in 33.3%, and psoas abscesses in 13.3% of the patients. The causative pathogen was identified in 75% of the cases (Table II). In 3 cases, mixed infections were present. Half of the patients underwent surgery, without wound infections or delay to healing. One patient died during inpatient admission. Eleven of the 19 patients completed an average follow-up of 13 months after discharge. During follow-up, 3 more patients died at an average of 45 months after discharge.

CONCLUSION

The incidence of spondylodiscitis is increasing due to factors such as the HIV epidemic, particularly in Sub-Saharan Africa, the large number of intravenous drug abusers, the currently widely used aspiration and catheter techniques, and the recurrence of tuberculosis in industrialized nations. Several weeks may elapse between the inception of symptoms and final diagnosis of spondylodiscitis. In principle, patients of all ages can contract spondylitis, but most likely 50 to 70 year-old patients.

In HIV+ patients, the peak age of disease lies much earlier : 10% are under 30 years of age when they first develop spondylitis. Worldwide, the estimated number of HIV+ patients is about 34 million, with an increasing tendency. In numerous regions of the world, 40% of new HIV infections are observed among the young population (15-24 years). Patients with any form of immunosuppression such as HIV have a significantly higher risk of developing spondylodiscitis.

Table II. — Demographic and HIV-related details

Gender		Age on admission	CDC	CD4 T-cell count on admission	CD4/CD8-ratio	HIV-RNA	Pathogen
m	w	years		[absolute/μl]		[copies/ml]	
				standard value: 435-1.600	Stand. value: 0.6-2.8		
	1	29	C 1	500	0.4	–	*Mycobacterium tuberculosis*
	1	41	C 3	330	0.9	–	Sterile
	1	21	–	–	–	–	*Mycobacterium tuberculosis*
1 1		41 54	– C 2	– –	– –	– –	*Staphylococcus aureus* *Staphylococcus aureus*
	1	48	–	–	–	–	Sterile
	1	33	B 2	50	0.4	1800	*Staphylococcus aureus, Pseudomonas aeruginosa*
1		67	C 3	100	0.4	< 50	Coagulase-negative Staphylococci
1		29	C 3	220	0.3	130	Sterile
1		28	C 3	400	0.5	< 50	*M. xenopi*
1		36	A 2	430	0.3	–	Sterile
1		40	A 2	–	–	–	*Staphylococcus aureus*
1		58	C 3	98	0.1	1.920	*Staphylococcus aureus*
1		49	A 2	310	0.2	< 50	*Mycobacterium tuberculosis*
1		42	C 3	82	0.2	< 50	*Staphylococcus aureus*, coagulase-negative Staphylococci
	1	39	A 3	157	0.3	–	*Staphylococcus aureus*
1		51	C 3	102	0.3	377	*Staphylococcus aureus*
1		33	C 3	129	0.1	< 50	*Mycobacterium bovis, Klebsiella pneumoniae*
1		60	C 3	355	0.4	–	Sterile
1		61	C 3	300	0.7	< 50	*Mycobacterium tuberculosis*

Spondylodiscitis can be treated to complete recovery if it is diagnosed and treated early, using the appropriate basic remedies : immobilisation of the affected spine segments, antibiotic therapy and depending on the extent of the disease, debridement, decompression, and stabilisation. Surgery is indicated in case of neurological deficits, sepsis, instability, impending or established deformities, intraspinal space-occupying lesions, suspicion of malignancy, and failed conservative therapy. Surgery can be indicated if the patient suffers from uncontrollable pain and does not comply with conservative therapy. In HIV+ patients, spondylodiscitis is associated with a low CD4 T-cell count, and a high mortality. A CD4 T-cell count below 100/L increases the probability of mixed infections, but there is no correlation between a low CD4 T-cell count and infection by MOTT (Mycobacteria Other Than Tuberculosis). As morbidity is not higher among HIV+ patients, the decision to use conservative or surgical treatment or to stabilize the affected spinal segments through surgery should be made without regard to this HIV context. However, these patients should be treated in a specialized institution by an experienced team of consultants.

REFERENCES

1. **Busch VJ, Regez RM, Heere B, Willems WJ.** Osteoarticular infections in HIV-infected patients : 23 cases among 1,515 HIV-infected patients. *Acta Orthop* 2007 ; 78 : 786-790.
2. **Carragee EJ, Kim D, van der Vlugt T, Vittum D.** The clinical use of erythrocyte sedimentation rate in pyogenic vertebral osteomyelitis. *Spine* 1997 ; 22 : 2089-2093.
3. **Chang MC, Wu HT, Lee CH, Liu CL, Chen TH.** Tuberculous spondylitis and pyogenic spondylitis : comparative magnetic resonance imaging features. *Spine* 2006 ; 31 : 782-788.
4. **Dunbar JA, Sandoe JA, Rao AS *et al.*** The MRI appearances of early vertebral osteomyelitis and discitis. *Clin Radiol* 2010 ; 65 : 974-981.
5. **Eismont FJ, Bohlman HH, Soni PL, Goldberg VM, Freehafer AA.** Pyogenic and fungal vertebral osteomyelitis with paralysis. *J Bone Joint Surg* 1983 ; 65-A : 19-29.
6. **Frangen TM, Kalicke T, Gottwald M *et al.*** [Surgical management of spondylodiscitis. An analysis of 78 cases.] (in German). *Unfallchirurg* 2006 ; 109 : 743-753.
7. **Friedman JA, Maher CO, Quast LM, McClelland RL, Ebersold MJ.** Spontaneous disc space infections in adults. *Surg Neurol* 2002 ; 57 : 81-86.
8. **Gouliouris T, Aliyu SH, Brown NM.** Spondylodiscitis : update on diagnosis and management. *J Antimicrob Chemother* 2010 ; 65 Suppl 3 : iii11-24.
9. **Govender S, Parbhoo AH, Kumar KP, Annamalai K.** Anterior spinal decompression in HIV-positive patients with tuberculosis. A prospective study. *J Bone Joint Surg* 2001 ; 83-B : 864-867.
10. **Grammatico L, Baron S, Rusch E *et al.*** Epidemiology of vertebral osteomyelitis (VO) in France : analysis of hospital-discharge data 2002-2003. *Epidemiol Infect* 2008 ; 136 : 653-660.
11. **Hoekman P, van de Perre P, Nelissen J *et al.*** Increased frequency of infection after open reduction of fractures in patients who are seropositive for human immunodeficiency virus. *J Bone Joint Surg* 1991 ; 73-A : 675-679.
12. **Horberg MA, Hurley LB, Klein DB *et al.*** Surgical outcomes in human immunodeficiency virus-infected patients in the era of highly active antiretroviral therapy. *Arch Surg* 2006 ; 141 : 1238-1245.
13. **Hsieh PC, Wienecke RJ, O'Shaughnessy BA, Koski TR, Ondra SL.** Surgical strategies for vertebral osteomyelitis and epidural abscess. *Neurosurg Focus* 2004 ; 17 : 1-6..
14. **Jevtic V.** Vertebral infection. *Eur Radiol* 2004 ; 14 Suppl 3 : E43-52.
15. **Klockner C, Valencia R, Weber U.** [Alignment of the sagittal profile after surgical therapy of nonspecific destructive spondylodiscitis : ventral or ventrodorsal method-a comparison of outcomes.] (in German). *Orthopade* 2001 ; 30 : 965-976.
16. **Lew DP, Waldvogel FA.** Osteomyelitis. *Lancet* 2004 ; 364 : 369-379.
17. **Lillie P, Thaker H, Moss P *et al.*** Healthcare associated discitis in the era of antimicrobial resistance. *J Clin Rheumatol* 2008 ; 14 : 234-237.
18. **Lucio E, Adesokan A, Hadjipavlou AG, Crow WN, Adegboyega PA.** Pyogenic spondylodiskitis : a radiologic/pathologic and culture correlation study. *Arch Pathol Lab Med* 2000 ; 124 : 712-716.
19. **Maiuri F, Iaconetta G, Gallicchio B, Manto A, Briganti F.** Spondylodiscitis. Clinical and magnetic resonance diagnosis. *Spine* 1997 ; 22 : 1741-1746.
20. **Modic MT, Feiglin DH, Piraino DW *et al.*** Vertebral osteomyelitis : assessment using MR. *Radiology* 1985 ; 157 : 157-166.
21. **Mulleman D, Philippe P, Senneville E *et al.*** Streptococcal and enterococcal spondylodiscitis (vertebral osteomyelitis). High incidence of infective endocarditis in 50 cases. *J Rheumatol* 2006 ; 33 : 91-97.
22. **Muller EJ, Russe OJ, Muhr G.** [Osteomyelitis of the spine] (in German). *Orthopade* 2004 ; 33 : 305-315.
23. **Nolla JM, Ariza J, Gomez-Vaquero C *et al.*** Spontaneous pyogenic vertebral osteomyelitis in nondrug users. *Semin Arthritis Rheum* 2002 ; 31 : 271-278.
24. **Price AC, Allen JH, Eggers FM, Shaff MI, James AE, Jr.** Intervertebral disk-space infection : CT changes. Work in progress. *Radiology* 1983 ; 149 : 725-729.
25. **Rankine JJ, Barron DA, Robinson P, Millner PA, Dickson RA.** Therapeutic impact of percutaneous spinal biopsy in spinal infection. *Postgrad Med J* 2004 ; 80 : 607-609.
26. **Sammak B, Abd El Bagi M, Al Shahed M *et al.*** Osteomyelitis : a review of currently used imaging techniques. *Eur Radiol* 1999 ; 9 : 894-900.
27. **Sapico FL, Montgomerie JZ.** Vertebral osteomyelitis. *Infect Dis Clin North Am* 1990 ; 4 : 539-550.
28. **Sapico FL.** Microbiology and antimicrobial therapy of spinal infections. *Orthop Clin North Am* 1996 ; 27 : 9-13.
29. **Schinina V, Rizzi EB, Rovighi L *et al.*** Infectious spondylodiscitis : magnetic resonance imaging in HIV-infected and HIV-uninfected patients. *Clinical Imaging* 2001 ; 25 : 362-367.
30. **Schinkel C, Gottwald M, Andress HJ.** Surgical treatment of spondylodiscitis. *Surg Infect* 2003 ; 4 : 387-391.
31. **Shaltot A, Michell PA, Betts JA, Darby AJ, Gishen P.** Jamshidi needle biopsy of bone lesions. *Clin Radiol* 1982 ; 33 : 193-196.
32. **Sharif HS, Aideyan OA, Clark DC *et al.*** Brucellar and tuberculous spondylitis : comparative imaging features. *Radiology* 1989 ; 171 : 419-425.
33. **Sobottke R, Seifert H, Fatkenheuer G *et al.*** [Current diagnosis and treatment of spondylodiscitis.] (in German). *Dtsch Arztebl Int* 2008 ; 105 : 181-187.
34. **Sobottke R, Zarghooni K, Seifert H *et al.*** Spondylodiscitis caused by Mycobacterium xenopi. *Arch Orthop Trauma Surg* 2008 ; 128 : 1047-1053.

35. Sobottke R, Zarghooni K, Krengel M *et al.* Treatment of spondylodiscitis in human immunodeficiency virus-infected patients : a comparison of conservative and operative therapy. *Spine* 2009 ; 34 : E452-458.

36. Tali ET. Spinal infections. *Eur J Radiology* 2004 ; 50 : 120-133.

37. Tuli SM. General principles of osteoarticular tuberculosis. *Clin Orthop Relat Res* 2002 : 398 : 11-19.

38. Vassilopoulos D, Chalasani P, Jurado RL, Workowski K, Agudelo CA. Musculoskeletal infections in patients with human immunodeficiency virus infection. *Medicine* 1997 ; 76 : 284-294.

39. Weinstein MA, Eismont FJ. Infections of the spine in patients with human immunodeficiency virus. *J Bone Joint Surg* 2005 ; 87-A : 604-609.

40. Woertgen C, Rothoerl RD, Englert C, Neumann C. Pyogenic spinal infections and outcome according to the 36-item short form health survey. *J Neurosurg Spine* 2006 ; 4 : 441-446.