Postoperative Spondylodiskitis: Etiology, Clinical Findings, Prognosis, and Comparison with Nonoperative Pyogenic Spondylodiskitis

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We studied 31 cases of postoperative pyogenic spondylodiskitis (POS), comparing them with 72 cases of nonpostoperative pyogenic spondylodiskitis (NPOS). POS represents 30.1% of cases of pyogenic spondylodiskitis. The onset of symptoms occurred an average (±SD) of 27.7 (± 25.3) days following surgery. Predisposing factors were less frequent in POS than NPOS cases ($P = .002$). Neurological complications and inflammatory signs in the spine were more frequent with POS than with NPOS ($P = .002$ and $P < .00001$). Coagulase-negative *Staphylococcus* and anaerobic bacteria were more frequent in POS than in NPOS ($P = .0001$ and $P = .05$). Percutaneous bone biopsies yielded the etiology in 66.7% of cases, open bone biopsies in 100%, blood cultures in 55.6%, and cultures of adjacent foci in 94.4%. Eleven patients (35.5%) were cured with antimicrobial treatment, but surgical treatment was necessary in 64.5%. No relapses or deaths were recorded. Seventeen patients (54.8%) had severe functional sequelae, which were associated with inflammatory signs in the spine ($P = .033$), higher levels of leukocytosis ($P = .05$), higher erythrocyte sedimentation rates ($P = .05$), and paravertebral abscesses ($P = .04$).

Spinal infections are relatively uncommon. Early diagnosis of spinal infection is difficult, and complications are frequent [1]. A rise in incidence has been reported over the past several years and is due mainly to an increase in the frequency of pyogenic nontuberculous cases [2], which number 1/250,000 persons per year.

Postoperative pyogenic spondylodiskitis (POS) is uncommon, but it is a severe, costly, and debilitating complication of vertebral surgery [3–9]. In the past several years, POS appears to have increased in frequency [7], in part because procedures performed on the spine have become more frequent and improvements in diagnostic capabilities have been made. POS presents insidiously. Symptoms such as pain or neurological deficits may be regarded as consequences of unsatisfactory performance and/or outcome of surgery or recurring disc herniation [10]. There are no hematologic, biochemical, or imaging findings that are unequivocally diagnostic of this process [11]. For these reasons, the diagnosis of POS is difficult and frequently delayed.

Staphylococcus aureus is the most common etiologic agent of pyogenic spondylodiskitis [1, 12, 13], followed distantly by aerobic gram-negative bacilli. *S. aureus* also is the most frequent etiologic agent in POS (at rates of up to 100% in some studies) [10, 14]. However, data from different studies suggest that the etiologic spectrum is wider [15,16]. Early diagnosis, identification of the causal agents, and specific antimicrobial treatment are essential for avoiding severe complications [2, 8, 17–19].

To our knowledge, no previous study has compared patients with POS vs. nonpostoperative pyogenic spondylodiskitis (NPOS). The purpose of the present study was to describe the clinical, etiologic, and evolutionary characteristics of POS, taking into account the differences with respect to NPOS.

Methods

Between January 1983 and December 1997, we diagnosed spondylodiskitis of confirmed etiology in 277 adult patients in two tertiary care centers in the south of Spain. These hospitals are 1,863-bed and 1,202-bed community and academic teaching hospitals, attending to a population of 1,250,000 and 1,000,000 inhabitants, respectively. They serve as regional reference centers.

Pyogenic spondylodiskitis was diagnosed for 103 patients, and 31 of them had POS. We carried out a retrospective study (1983–1989) and a prospective observational study (1990–1997) of these cases. Cases of tuberculous and brucellar spondylodiskitis were not included because these conditions present significant clinical, biological, and prognostic differences from pyogenic spondylodiskitis [1].

The diagnosis of pyogenic spondylodiskitis was established when all three of the following criteria were met: (1) clinical symptoms of back pain with inflammatory characteristic features (back pain unrelieved by rest) or fever (temperature of

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38°C), as well as pain or tenderness of the back on physical examination; (2) imaging findings compatible with vertebral osteomyelitis [20], evaluated independently by the clinician and the radiologist; and (3) microbiological evidence such as isolation of a microorganism from percutaneous or open bone biopsy specimens, cultured blood, and/or specimens from another adjacent infectious foci. Adjacent infectious foci included epidural abscesses or masses, abscesses in paravertebral or psoas or iliopsoas muscles, and surgical wound suppuration.

Blood cultures were processed at 37°C for 2–5 days. Bone biopsy specimens and samples from adjacent foci were immediately processed and cultivated in aerobic media (Columbia agar, blood agar, MacConkey agar, and thioglycollate broth at 37°C) and anaerobic media (Schaedler agar, Columbia CDC agar, phenylethyl alcohol blood agar, and thioglycollate broth).

In addition to the above criteria, diagnosis of POS required that clinical symptoms appear in the 12-month period following spinal surgery. For classification of wound infection, the criteria of the Centers for Disease Control and Prevention were used [21]. All the patients were seen and followed up by one of the authors. Follow-up examinations were done over a period of at least 12 months, with special attention given to detection of relapses and assessment of functional sequelae.

We analyzed the following data: age; sex; duration of symptoms before consultation; patient-related risk factors or chronic underlying diseases (e.g., diabetes mellitus, intravenous drug use, previous bacteremia or focal infections, and immunodeficiency); clinical features (fever, chills/rigors, constitutional symptoms, inflammatory spinal pain, spinal pain, neurological symptoms and signs, muscular contracture, spinal deformity, and inflammatory signs in the spine [local erythema, swelling and/or heat, and/or surgical wound or spontaneous suppuration]); hematologic and biochemical features (blood count, erythrocyte sedimentation rate [ESR], and C-reactive protein [CRP] value); radiological features (from roentgenography, CT, and/or MRI); microbiological features; therapeutic records; and prognosis. We compared POS and NPOS with regard to all these data and studied the factors related to a poor outcome of POS.

Therapeutic failure was defined as persistent or worsening symptoms plus a high ESR or CRP level or worsening imaging findings, after a month of specific treatment. A relapse was defined as the reappearance of symptoms not attributable to other causes or new vertebral lesions accompanied by a raised ESR or CRP level (or both) after the end of treatment. Functional sequelae were considered to be severe when the disability prevented the patient from carrying out usual occupational or daily activities. Outcome was defined as poor if the patient had sequelae, relapsed, or died.

For the statistical analysis of data we used the BMDP Statistical Software Package (version 1.993; BMDP, Los Angeles). The Kruskal-Wallis nonparametric test or the Student's *t* test was used for comparison of means, and Fisher's exact test or Pearson's χ^2 test for comparison of proportions. Differences were considered significant at a P value of $<.05$. Results are expressed herein as mean ± SD.

Results

POS was diagnosed for 31 patients, accounting for 30.1% of the cases of pyogenic spondylodiskitis and 11.2% of all cases of spondylodiskitis during the period of study. Eighteen patients (58.1%) were male and 13 (41.9%) were female, and the mean age was 51.6 (±14) years (range, 17–77 years). The surgical procedures performed on the spine before occurrence of POS were diskectomy and/or laminectomy in 18 patients (58.1%); discectomy, laminectomy, and arthrodesis in 5 (16.1%); instrumentation for stabilization of the spine in 3 (9.7%); tumoral exeresis in 3 (9.7%); and nucleolysis in 2 (6.5%). The primary diagnoses were herniated disc in 24 patients (77.4%), spinal tumor in 3 (9.7%), spinal stenosis in 2 (6.5%), and scoliosis and vertebral fracture in 1 each.

Half the patients with POS developed clinical symptoms in the first 2 weeks following the surgical procedures; the onset of symptoms occurred at an average of 27.7 (±25.3) days following surgery. The most frequently involved vertebral level was the lumbar (87.1%), followed by the cervical (9.7%). There was only one case involving the thoracic level (3.2%).

The presence of predisposing factors other than surgery was less frequent in patients with POS than in those with NPOS ($P = .002$). Diabetes mellitus was registered for only six (19.3%) of the patients with POS vs. 19 (26.4%) of the patients with NPOS (P = NS). In addition, previous bacteremia and infections at other sites were less frequent in patients with POS than in those with NPOS (6.5% vs. 36.1% and 32.4% vs. 69.4%; $P = .004$ and $P = .002$, respectively).

Fever occurred in 21 (67.7%) of the patients with POS; of these, 19 (90.5%) had deep-incisional surgical site infection and/or paravertebral or epidural abscesses ($P = .02$). Fever was not more frequent in patients with positive blood cultures. Fifteen patients (48.4%) had inflammatory spinal signs. Seven patients (22.6%) had no fever and no inflammatory spinal signs. All patients with POS presented with back pain, and 29 (93.5%) had inflammatory spinal pain. Neurological symptoms were more frequent in patients with POS than in those with NPOS (87.1% vs. 55%; $P = .002$). In the same way, neurological signs were more frequent in cases of POS than in NPOS ($P = .007$), including Lasègue's sign (64.5% vs. 30.6%; $P = .002$), impaired or absent tendinous reflexes (58.1% vs. 17.8%; $P = .00008$), and objective sensory loss (54.8% vs. 25%; $P = .0045$). Severe neurological deficits were recorded in 12 cases (38.7%): 10 cases of paresis of one or two limbs and 2 cases of tetraparesis (6.5%). Table 1 shows the clinical differences between POS and NPOS.

Leukocytosis (leukocyte count of $>10.5 \times 10^9$/L) was present in 22 patients with POS (71%). Total WBC and neu-

Table 1. Clinical findings in and differences between cases of postoperative pyogenic spondylodiskitis (POS) and nonpostoperative pyogenic spondylodiskitis (NPOS).

Clinical finding	No. (%) of cases		*P* value
	POS (n = 31)	NPOS (n = 72)	
Fever	21 (67.7)	50 (69.4)	NS
Chills/rigors	17 (54.8)	41 (56.9)	NS
Constitutional symptoms	17 (54.8)	44 (61.1)	NS
Inflammatory spinal pain*	29 (93.5)	67 (93.1)	NS
Inflammatory signs in spine†	15 (48.4)	7 (9.7)	.00001
Muscular contracture	27 (87.1)	45 (62.5)	.02
Spinal deformity	6 (19.4)	3 (4.2)	.01
Neurological symptoms	27 (87.1)	39 (54.9)	.0004
Neurological signs	28 (90.3)	42 (58.3)	.0039

* Spinal pain unrelieved by rest.
† Local erythema, swelling and/or heat, and/or surgical wound or spontaneous suppuration.

trophil counts were higher in POS than NPOS cases (P = .0075 and P = .0092, respectively). There were no differences in leukocyte counts dependent on the presence or absence of positive blood cultures, abscesses, or epidural masses. A raised ESR (>15 mm/h) was recorded for all patients with POS, and a raised CRP level (>5 mg/L) was found in 100% of the 18 patients with POS for whom it was evaluated. There were no differences in ESR and CRP levels between the two groups. Table 2 shows the main laboratory findings and differences between POS and NPOS.

Thirty-five microorganisms were isolated in the 31 cases of POS. The most frequent etiologic agent was *S. aureus* (31.4%), followed by coagulase-negative *Staphylococcus* (28.6%). Nine (42.9%) of 21 staphylococci (4 *S. aureus* and 5 coagulase-negative *Staphylococcus*) were methicillin-resistant. Ten cases (32.3%) involved bacteria other than *Staphylococcus* species. Gram-negative bacilli were isolated in six cases (19.6%) and anaerobic bacteria in five (16.1%). Coagulase-negative *Staphylococcus* and anaerobic bacteria were more frequent in POS than in NPOS (28.6% vs. 5.1%, P = .005; and 17.4% vs. 5.1%, P = .05, respectively). Table 3 shows the etiologic agents isolated in both groups.

Table 2. Laboratory findings in patients with postoperative pyogenic spondylodiskitis (POS) and nonpostoperative pyogenic spondylodiskitis (NPOS).

Laboratory finding	Mean (±SD) value or other data		*P* value
	POS (n = 31)	NPOS (n = 72)	
Leukocytes (no. per mL)	13,931 (±6,663)	10,649 (±5,480)	.0075
Neutrophils (no. per mL)	11,218 (±6,741)	8,098 (±4,981)	.0092
Hemoglobin (g/dL)	11.29 (±3)	11.4 (±2.1)	NS
ESR (mm/h)	83.75 (±37.3)	83.7 (±35.3)	NS
C-reactive protein (mg/L)	72.4 (±66.8)	86.6 (±86.9)	NS
Positive blood culture* (%)	10/18 (55.6)	26/55 (47.2)	NS

NOTE. ESR = erythrocyte sedimentation rate.
* No. of positive results/total no. performed.

Table 3. Etiologic agents of postoperative pyogenic spondylodiskitis (POS) and nonpostoperative pyogenic spondylodiskitis (NPOS).

Etiologic agents	No. (%) of isolations	
	POS (n = 31)	NPOS (n = 72)
All agents, total no.	35	78
Gram-positive cocci	22 (62.6)	50 (64.1)
Staphylococcus aureus	11 (31.4)	36 (46.2)
Staphylococcus epidermidis	8 (22.9)	1 (1.3)
Other coagulase-negative *Staphylococcus*	2 (5.7)	3 (3.8)
Streptococcus agalactiae	1 (2.9)	. . .
Other	. . .	10 (12.8)
Gram-negative bacilli	7 (20)	23 (29.5)
Pseudomonas aeruginosa	4 (11.4)	6 (7.7)
Stenotrophomonas maltophilia	1 (2.9)	. . .
Escherichia coli	1 (2.9)	11 (14.1)
Enterobacter aerogenes	1 (1.9)	. . .
Other	. . .	6 (7.7)
Anaerobic bacteria	6 (17.4)	4 (5.1)
Peptostreptococcus species	1 (2.9)	1 (1.3)
Bacteroides fragilis	3 (8.6)	. . .
Prevotella melaninogenica	1 (2.9)	1 (1.3)
Propionibacterium avidum	1 (2.9)	. . .
Other	. . .	2 (2.6)
Corynebacterium species	. . .	1 (1.3)
Polymicrobial isolations	2 (6.5)	4 (5.1)

Twenty bone biopsies were performed in 18 cases of POS; 4 (66.7%) of the 6 percutaneous bone biopsies were positive and all 14 of the open bone biopsies were positive. The two patients with negative percutaneous biopsy cultures had positive open bone biopsy cultures. Blood cultures were performed for 18 patients with POS and 55 with NPOS and were positive for 10 (55.6%) and 26 (47.3%), respectively (P = NS). Cultures of specimens from adjacent infectious foci were done in 18 cases and were positive in 17 (94.4%), including cases of paravertebral abscess (9), surgical wound suppuration (7), psoas abscess (2), CSF infection (2), and epidural abscess (1). Eleven patients (35.5%) had the same etiologic agent isolated from two different samples (bone biopsy, adjacent foci, or blood).

All patients underwent imaging studies. Roentgenography of the spine was performed initially on all patients and showed abnormalities in 27 (87.1%). Vertebral CT was performed in 17 cases, showing signs of spondylodiskitis in 15 (88.2%). Spinal CT showed paravertebral abscesses in 9 cases (52.9%), psoas abscesses in 5 (29.4%), and epidural masses in 7 (41.2%). MRI was carried out in 23 cases and showed signs of spondylodiskitis in all 23. Moreover, MRI showed paravertebral abscesses in 12 cases (52.3%), psoas abscesses in 4 (17.4%), and epidural masses in 13 (56.5%). For two patients whose CT findings were normal, spondylodiskitis was suggested by MRI.

Twenty-eight (90.3%) of the patients with POS had involve-

ment of two or more vertebral bodies. Twenty-three (74.2%) presented with epidural masses or paravertebral or psoas abscesses (paravertebral abscesses in 13 [41.9%], epidural masses in 15 [48.4%], and psoas abscesses in 6 [19.4%]). There was no association between presence of masses or abscesses and identification of gram-positive cocci or gram-negative bacilli as the etiology of POS. There also were no differences between POS and NPOS with respect to the presence of masses or abscesses.

Diagnostic delay in NPOS cases was more prolonged than in POS cases (53.5 [±42.3] vs. 27.7 [±25.3] days; $P = 0.0011$). Diagnostic delay in POS cases was more prolonged for patients without fever (36.4 [±21.9] vs. 18.7 [±13.6] days; $P = .014$) or without inflammatory signs in the spine (31.6 [±21.5] vs. 17.3 [±11.3] days; $P = .036$). The diagnostic delay was less in cases of POS caused by *S. aureus* (20.4 [±19.3] days) than in those caused by coagulase-negative *Staphylococcus* (29.7 [±19.1] days) or gram-negative bacilli (50.3 [±50.4] days), although these differences were not significant.

Patients with POS were treated intravenously with specific antimicrobials for at least 3–4 weeks, followed by oral treatment for 4–12 additional weeks. Total duration of treatment depended on the criteria of physicians in charge of each patient. Twenty-six (83.9%) of the patients were treated for 45–90 days and five (16.1%) for >90 days. Duration of antimicrobial treatment was the same in cases of POS due to gram-positive cocci vs. gram-negative bacilli (73.1 [±28.6] vs. 75.4 [±30.2] days), in cases caused by methicillin-resistant vs. methicillin-susceptible staphylococci (67 [±24.5] vs. 78.4 [±31.86] days), and for patients with or without epidural masses and/or paravertebral abscesses (78.9 [±34] vs. 65.2 [±13.8] days).

Eleven patients with POS (35.5%) were cured with antimicrobial treatment alone. For 20 patients (64.5%), surgical treatment was needed because of the presence of large abscesses, cord or radicular compression with severe neurological deficits, highly destructive bone lesions with spinal instability, or medical therapeutic failure (one case). The time span from diagnosis to surgery was 10.8 (±9.3) days for patients with POS. For nine patients with POS, the surgical treatment was performed within the first week. Curettage and debridement were carried out in 16 cases, drainage of abscesses in 11, diskectomy and laminectomy in 2, and spinal fixation in 1. POS caused by gram-negative bacilli and/or anaerobic bacteria required surgery more frequently (88.9%) than POS caused by gram-positive cocci (57.1%). Only one case required a second surgical intervention, because of treatment failure. Surgery had no influence on the duration of antimicrobial treatment. There were no differences from NPOS in the need for surgical treatment.

No relapses and no deaths occurred among patients with POS. Seventeen patients with POS (54.8%) had one or more severe functional sequelae. Among these, 11 (35.5%) had severe pain, 4 (12.9%) had difficulty moving, 4 (12.9%) had monoparesis, and 3 (9.7%) had paraparesis. Twenty-seven patients with NPOS (60%) had severe sequelae (P = NS), five patients had relapses, and three died.

Hospital stay ranged between 30 and 180 days in POS cases. In comparison with that for patients without these features, the stay was significantly longer for patients with paravertebral abscesses or masses (89.39 [±48.16] vs. 62.16 [±33.9] days; $P = .05$), epidural masses (88.13 [±41.53] vs. 59.9 [±39.88] days; $P = .0135$), or severe sequelae (86.65 [±45.72] vs. 57.7 [±31.7] days, $P = .05$). In cases of POS, a poor outcome was associated with the presence of inflammatory signs in the spine ($P = .033$), higher levels of leukocytosis ($P = .05$), higher ESRs ($P = .05$), and paravertebral abscesses ($P = .04$).

Discussion

The incidence of POS varies from 0.21% to 3.6% in association with all surgical procedures [6–9], and the condition occurs with any type of intervention. The risk is lower in association with diskectomy and laminectomy than with bone graft, reconstruction with use of hardware, or spinal instrumentation, procedures that may carry a risk of infection as high as 36% [4, 14, 22]. Iatrogenically induced spondylodiskitis accounts for 20%–30% of all cases of pyogenic vertebral osteomyelitis [2, 18], figures similar to those recorded in this study.

The pathogenesis of POS is assumed to be direct inoculation during surgery as well as spread from other infectious foci [16, 22, 23]. Previous or concurrent infections (principally in the skin, urinary tract, and lungs) or immunodeficiencies increase the risk of POS [4, 14]. However, in this study, risk factors such as previous bacteremia and previous or concurrent infections were less frequent in cases of POS than NPOS. This suggests that POS is acquired mainly during surgical procedures and that in NPOS the principal pathogenic mechanism is hematogenous spread.

Injury to endplates, operative trauma to small vessels, hematomas in the intervertebral space, and necrotic tissue caused by surgery provide viable culture specimens and are risk factors for postoperative intervertebral disc space inflammation. In addition, there are reports that attribute the symptoms of POS to conditions such as postoperative avascular necrosis or chemical discitis, without consideration of a pyogenic etiology [5, 17, 24]. In the present study all the patients unquestionably had pyogenic spondylodiskitis, with positive cultures of the biopsied bone, blood, and/or other samples.

The mean age in this study was similar to that reported by other authors [3]. Although we found a predominance of males, it was not as high as the 75% proportion among the cases reported by others [3]. Predisposing factors for the development of POS include diabetes mellitus, treatment with steroids, and intercurrent infections [10, 22]. Piotrowski et al. [11] found that 26% of patients with POS had diabetes mellitus, whereas the incidence of diabetes among all surgical patients is 6.8%, similar to that in the European population. In the present study diabetes mellitus was present in 19.4% of POS cases and in 26.4% of NPOS cases. These data suggest that diabetes mel-

litus is a risk factor for pyogenic spondylodiskitis in general, not only for POS.

The clinical picture of POS is nonspecific. Postoperative symptoms appear from a few days to 10 weeks following surgery, after an initial period of pain relief [3–5,10]. In the present study the onset of 90% of the cases was in the first 50 days following surgery. Other studies have shown that the body temperature is elevated in only 33%–52% of patients [4, 5, 10, 11], although these studies included cases without bacteriologic confirmation. In our study 67.7% of patients were feverish, which may be explained by the fact that all cases included were of pyogenic spondylodiskitis. Severe back pain, muscle contractures, limited range of spinal motion, spasticity, sciatica, inability to bend, and a positive Lasègue's sign are the most common symptoms and signs. Several reports referred to the infrequency of motor and sensory signs [5, 7, 11]. On the contrary, in our experience 87.1% of cases had neurological symptoms.

POS is a special form of bacterial wound infection [11]. In this study 48.4% of patients presented with inflammatory signs in the spine. The presence of epidural or paravertebral masses is frequent (67% in this study), as has been reported previously [14].

The absence of fever and inflammatory signs in the spine, as occurred in 22.6% of our cases, does not exclude a diagnosis of POS. Reappearance of spinal pain after surgery may be attributed to recurring disc herniation, unsatisfactory performance and/or outcome of surgery, or psychoneurotic disorders [10]. Such alternative explanations, along with the absence of fever and/or inflammatory signs in the spine, may lead to a delay in diagnosis [25], as we showed in this study. Moreover, the ESR may be raised for 3 months following surgery [10], and initial roentgenographs of the spine can be normal or show nonspecific alterations.

Some studies of POS have not specified the etiology or have included cases without a bacteriologically proven diagnosis [3, 5, 7, 10, 11]. In these studies blood cultures have been positive for only zero to 33% of patients [3, 5, 10]. In our study, in which all cases were diagnosed bacteriologically, 55.6% of blood cultures were positive. These data indicate that blood cultures must always be performed, including for patients without fever, since 11% of them had positive blood cultures.

Aspiration or biopsy sampling of the disc space or of the vertebra was a sensitive method in our experience. Tronnier et al. found that 17% of cultures of specimens from the intervertebral disc space were positive [16], and Rawlings et al. found the rate to be 50% [10]. We obtained 18 positive culture results in 20 bone biopsies, and surgical bone biopsy was more useful than percutaneous bone biopsy (100% vs. 66.7% positivity). In addition, other specimens such as those from paravertebral or psoas abscesses or epidural masses were very useful for etiologic diagnosis. The cultures of these samples were positive in 94.1% of the 17 cases in which they were performed.

Pyogenic vertebral osteomyelitis is caused by a variety of organisms. *S. aureus* is the more frequent pathogen (40%–67% of cases), followed by gram-negative bacteria (30%) and *Streptococcus* species (7%–10%) [1, 3, 12, 14, 25, 26]. In POS the most frequent agents are gram-positive cocci, mainly *Staphylococcus* species (which are found in up to 100% of cases in some studies) [10]. Dall et al. [3], in a review of 46 cases of postoperative discitis, found that *Staphylococcus* species caused 42 (91%) (26, *S. aureus*; 16, *Staphylococcus epidermidis),* gram-negative bacilli caused 3 (6.5%), and *Propionibacterium* species caused 1.

Because of these findings, some authors argue that aspiration or biopsy sampling is not necessary, and therefore empirical treatment against staphylococci should be prescribed. However, in the present study, 40% of the agents involved in POS were organisms other than *Staphylococcus* species. In addition, 42.9% of the *Staphylococcus* species isolates were methicillin-resistant. In our own experience, the frequency of gram-negative bacilli in cases of POS has been similar to that in cases of NPOS (20% and 29.5%, respectively), and *Pseudomonas aeruginosa* has been the organism most frequently involved. Finally, anaerobes accounted for 17.4% of the etiologic agents of POS and 5.1% of those of NPOS, and 6.5% of the cases of POS were polymicrobial. These results strongly suggest the necessity of etiologic diagnosis of POS as well as NPOS, to enable prescription of appropriate treatment.

Hematologic parameters are of little value in the diagnosis of spondylodiskitis [1]. A mild to moderate rise in the WBC count is reported in only 30%–50% of POS cases [5, 10, 11, 14]. We found leukocytosis in 71% of cases, but there was no difference in leukocyte counts in relation to extravertebral extension. The ESR is raised in many patients with POS [5, 7, 10]. However, it is an unspecific parameter of inflammation; 54%–100% of patients with an uneventful postoperative course and without spondylodiskitis have a raised ESR [24]. Therefore, we did not consider a raised ESR to be a criterion for diagnosis of POS, unlike other authors [3, 7, 10].

CRP levels are a very useful parameter for supporting the clinical diagnosis and also for following the course of POS [10]. Although the CRP value is also unspecific, it is more conclusive than the ESR; an increase in the CRP value is very rare in uneventful postoperative courses [10].

Roentgenographs may not demonstrate the early changes in POS [7, 10]; in our study, in 12.9% of cases of POS, roentgenographic findings were normal. Radionuclide bone scans are not useful, showing a nonspecific increase in tracer uptake, no different from that expected after uncomplicated disc surgery [10]. CT and MRI have higher sensitivity and specificity than other imaging methods [27, 28]. Although CT scanning is not helpful in the early diagnosis of postoperative spondylodiskitis in all cases [3], it was diagnostic in 88.2% of cases in the present study. However, it is very

useful for demonstrating paravertebral masses or abscesses [29]. MRI is more sensitive and was positive in 100% of cases in this study and useful in the diagnosis of extravertebral involvement. MRI may also be an unspecific diagnostic procedure [15]. Post-contrast enhancement, both in the disc and in the vertebral endplates, has diagnostic value as a sign of bacterial spondylodiskitis [30], although it is not always specific [31].

There is general agreement on the method of treating POS: immobilization plus prolonged antimicrobial therapy with bone-penetrating antibiotics and symptomatic treatment. Most authors favor some type of immobilization, ranging from a cast to bed rest [5], until the patient is comfortable. Generally, rigid immobilization is not necessary [3]. In this study, all patients received antimicrobial treatment for >6 weeks.

Surgical management must be individualized [22]. Some authors recommend that every case should be surgically treated, and others believe that this is not necessary unless conservative treatment fails or an abscess or epidural mass is present [3, 11]. Surgical debridement should consist of aggressive removal of all foreign materials and necrotic and purulent tissues. In the present study 11 cases (35.5%) were cured with antimicrobial treatment alone. The need for surgery was more frequent than in other studies (54.8% vs. 10%–25%) [11, 25, 30, 32]. This may be attributed to a higher incidence of neurological deficits, large abscesses, and highly destructive bone lesions.

With appropriate treatment, relapses of POS are uncommon, ranging from zero in the present study to 4% in others [10]. The duration of follow-up necessary to demonstrate no relapses is not well established [14], although it should be at least 12 months from the onset of treatment. The mortality associated with POS is negligible, ranging from zero in this study to 1.4% in others [9].

In the present study sequelae were very frequent compared to other studies. Only 45% of patients with POS returned to work and normal daily activities, a figure similar to that referenced previously [3]. In other studies of pyogenic spondylodiskitis [2, 3, 7, 8, 11, 17–19, 22, 23, 25, 30], the prognosis has depended on early diagnosis, identification of the causal agents, and initiation of a specific treatment. In the present study, poor outcome was associated with the presence of inflammatory signs in the spine, higher levels of leukocytosis, higher ESR, or the presence of paravertebral abscesses.

In summary, if POS is suspected on the basis of clinical data, then the ESR and CRP level should be determined and blood and wound sample cultures and MRI should be performed. Because there are many causes of POS (gram-positive, gram-negative, and anaerobic bacteria), if the previous cultures are negative, then a bone biopsy or aspirative paravertebral sampling should be attempted. A combination of bed rest, prolonged administration of antimicrobials, and surgery (when necessary) is the treatment of choice for POS, which is a serious process with severe functional sequelae.

References

1. Colmenero JD, Jiménez-Mejías ME, Sánchez-Lora FJ, et al. Pyogenic, tuberculous, and brucellar vertebral osteomyelitis: a descriptive and comparative study of 219 cases. Ann Rheum Dis **1997**;56: 709–15.
2. Bontoux D, Codello L, Debiais F, Lambert de Cursay G, Azais I, Alacaly M. Spondilociscites infectieuses. Analyse d'une série de 105 cas. Rev Rhum Mal Osteoartic **1992**;59:401–7.
3. Dall BE, Rowe DE, Odette WG, Batts DH. Postoperative discitis: diagnosis and management. Clin Orthop **1987**;224:138–46.
4. Levi ADO, Dickman CA, Sonntag VKH. Management of postoperative infection after spinal instrumentation. J Neurosurg **1997**;86: 975–80.
5. Puranen J, Mäkelä J, Lähde S. Postoperative intervertebral discitis. Acta Orthop Scand **1984**;55:461–5.
6. El-Gindi S, Aref S, Salama M. Infection of intervertebral discs after operation. J Bone Joint Surg Br **1976**;58:114–6.
7. Dauch WA. Infection of the intervertebral space following conventional and microsurgical operation on the herniated lumbar intervertebral disc: a controlled clinical trial. Acta Neurochir (Wein) **1986**;82:43–9.
8. Frank AM, Trappe AE. The role of magnetic resonance imaging (MRI) in the diagnosis of spondylodiscitis. Neurosurg Rev **1990**;13:279–83.
9. Deyo RA, Cherkin DC, Loeser JD, Bigos SJ, Ciol MA. Morbidity and mortality in association with operations on the lumbar spine. J Bone Joint Surg Am **1992**;74:536–43.
10. Rawlings CE, Wilkins RH, Gallis HA, Goldner JL, Francis R. Postoperative intervertebral disc space infection. Neurosurgery **1983**;13: 371–5.
11. Piotrowski WP, Krombholz MA, Mühl B. Spondylodiscitis after lumbar disc surgery. Neurosurg Rev **1994**;17:189–93.
12. Rath SA, Neff U, Schneider O, Richet HP. Neurosurgical management of thoracic and lumbar vertebral osteomyelitis and discitis in adults: a review of 43 consecutive surgically treated patients. Neurosurgery **1996**; 38:926–33.
13. Osenbach RK, Hitchon PW, Menezes AH. Diagnosis and management of pyogenic vertebral osteomyelitis in adults. Surg Neurol **1990**;33:266–75.
14. Dietze DD, Fessler RG, Jacon RP. Primary reconstruction for spinal infections. J Neurosurg **1997**;86:981–9.
15. Chevalier X, Lavabre C, Claudepierre P, Larget-Piet B. Iatrogenically induced vertebral osteomyelitis due to *Pseudomonas aeruginosa.* Clin Exp Rheumatol **1996**;14:191–4.
16. Tronnier V, Schneider R, Kunz U, Albert F, Oldenkott P. Postoperative spondylodiscitis: results of a prospective study about the aetiology of spondylodiscitis after operation for lumbar disc herniation. Acta Neurochir (Wein) **1992**;117:149–52.
17. Lyndholm TS, Pylkkänen P. Discitis following removal of intervertebral disc. Spine **1982**;7:618–22.
18. Meys E, Deprez X, Hautefeuille PH, Flipo RM, Duquesnoy B, Delcambre B. Place des spondylodiscites iatrogègenes parmi les spondylodiscites à germes banals: 136 cas observés entre 1980 et 1989. Rev Rhum Mal Osteoartic **1991**;58:839–46.
19. Torda AJ, Gottlieb T, Bradbury R. Pyogenic vertebral osteomyelitis: analysis of 20 cases and review. Clin Infect Dis **1995**;20:320–8.
20. Modic MT, Feiglin DH, Piraimo DW, et al. Vertebral osteomyelitis: assessment using MR. Radiology **1985**;157:157–66.
21. Horan TC, Gaynes RP, Martone WJ, Jarvis WR, Emori TG. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. Infect Control Hosp Epidemiol **1992**;13:606–8.

22. Stambough JL, Beringer D. Postoperative wound infections complicating adult spine surgery. J Spinal Disord **1992**;5:277–85.
23. Blankstein A, Rubinstein E, Ezra E, Lokiec F, Caspi I, Horoszowski H. Disc space infection and vertebral osteomyelitis as a complication of percutaneous lateral discectomy. Clin Orthop **1987**;225:234–7.
24. Bircher MD, Tasker T, Crawshaw C, Mulholland RC. Discitis following lumbar surgery. Spine **1988**;13:98–102.
25. Dendrinos GK, Polyzoides JA. Spondylodiscitis after percutaneous discectomy: a case diagnosed by MRI. Acta Orthop Scand **1992**;63:219–20.
26. Sapico FL, Montgomerie JZ. Pyogenic vertebral osteomyelitis: report of nine cases and review of the literature. Rev Infect Dis **1979**;1:754–6.
27. Sharif HS. Role of RM imaging in the management of spinal infections. Am J Roentegenol **1992**;158:1333–45.
28. Whelan MA, Schonfeld S, Post JD, et al. Computed tomography of nontuberculous spinal infection. J Comput Assist Tomogr **1985**;9:280–7.
29. Kopecky KK, Gilmor RL, Scott LA, Edwards MK. Pitfalls of computed tomography in diagnosis of discitis. Neuroradiology **1985**; 27:57–66.
30. Boden SD, Davis DO, Dina TS, Sunner JL, Wiesl SW. Postoperative discitis: distinguishing early MR imaging findings from normal postoperative disc space changes. Radiology **1992**;184:765–71.
31. Grand CM, Bank WO, Balériaux D, Matos C, Levivier M, Brotchi J. Gadolinium enhancement of vertebral endplates following lumbar disc surgery. Neuroradiology **1993**;35:503–5.
32. Pilgaard S. Discitis (closed space infection) following removal of lumbar intervertebral disc. J Bone Joint Surg Am **1969**;51:713–6.